GUY'S HOSPITAL REPORTS.

EDITED BY

C. HILTON FAGGE, M.D.

AND

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J. E. ADLARD, BARTHOLOMEW CLOSE.

CONTENTS.

The Editors regret that they have been compelled to defer the continuation of the series of Chromo-lithographs of the Ophthalmoscopic appearances of the Human Eye, commenced in the last volume under the superintendence of Mr. Bader. The arrangements made for the execution of the plates in Germany were absolutely frustrated by the war on the Continent, at a period so late that it was impossible to adopt any fresh plan. The series will be continued as early, and carried out as completely, as possible. In the meantime, the Editors trust that the Subscribers will accept, in lieu, in the present volume, a series of Coloured Illustrations received from Mr. Hinton, of various Morbid Conditions of the Membrana Tympani, as seen through the speculum.

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In Exchange.

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Royal London Ophthalmic Hospital Reports.

Archives of Medicine, Dr. Lionel Beale, 61, Grosvenor Street, West.

The London Hospital Reports.

LIST OF PLATES.

LIST OF WOODCUTS.

ON HYDROPHOBIA.

By J. COOPER FORSTER.

There is a general impression that hydrophobia occurs at the present time more frequently than formerly; we have therefore thought that the following cases of the disease might be advantageously reviewed.

The name hydrophobia we all know as a generally well-understood term applied to certain symptoms induced by the bite of a rabid animal.

It is unnecessary to notice the substitutes which have been proposed; for though the term hydrophobia has been frequently cavilled at, we know of none better to distinguish the disease; and the alarm it is said to inspire in the minds of those attendant upon the sufferer must be due rather to the nature of the affection than to its name.

There prevails at the present time an idea that a greater number of cases of hydrophobia have occurred during the last two or three years than in the preceding twenty; and judging from the records of our Hospital, this appears to be the truth. If we refer to the Registrar General's report, we find that since the year 1838, when twelve deaths occurred from hydrophobia, there have not been so many as nine cases occurring in any one year until 1865. The following tabular statement shows the number of deaths from the disease since a record has been kept of the deaths in the metropolis.

Deaths from Hydrophobia in London.

July 1st to Dec. 31st, 1837	7	Jan. 1st to Dec. 31st, 1852	0
Jan. 1st to Dec. 31st, 1838	12	1853	1
1839	4	1854	7
1840	1	1855	2
1841	3	1856	0
1842	4	1857	2
1843	2	1858	0
1844	3	1859	0
1845	2	1860	0
1846	1	1861	0
1847	0	1862	0
1848	1	1863	2
1849	0	1864	0
1850	1	1865	9
1851	1	Jan. 1st to June 30th, 1866	6

There may, therefore, be some little cause for anxiety in the public mind in the matter, but certainly not to any great extent. When we come to examine the matter dispassionately we see that even the first half of the present year (though there have been six deaths in London) was equalled in the latter half of the year 1837, when registration first commenced, and when there was a much smaller population.

Attempts have been made to show that bites from dogs are more frequent now than formerly; we can only give the numbers for two and a half years at Guy's Hospital; and this cannot be said to show much, except that dogs (and not dogs only) are accustomed to bite. In the year 1864, 51 cases of dog-bites were brought to the hospital; 6 were bad enough to be admitted. In the year 1865 there were bites from dogs 47; and from horses, men, women, donkeys, a cat, a rat, and a fox, 15; in all 62. In the first six months of the present year there have been 46 cases of bites from dogs and 9 from other sources, 13 of which occurred in March and 13 in June. That the numbers of bites from animals have increased, therefore, is apparent, but whether the creatures were rabid or not it is impossible to say. We do not appear to gather much from these reports.

In no one case yet have we seen the original bite and the patient subsequently affected with hydrophobia. The fact of our witnessing a larger number of bites during the last year or two than formerly is probably owing, as is a general increase in accident cases, to the removal of St. Thomas's Hospital.

The idea that animals are more disposed to become rabid during the hot weather of July has long been set aside by those who know anything of the matter. Of the cases we have reported, one bite is recorded as having taken place in June, one in July, one in September, one in October, and one in December, the hydrophobic symptoms following at various intervals.

Dr. Bright, in his valuable work entitled 'Medical Reports,' mentions six cases of hydrophobia as having been admitted into Guy's Hospital in the decade from 1820 to 1830. In two the year is not mentioned; of the four others one occurred in 1821, one in 1825, another in 1827, and the fourth in 1829. Two post-mortems were afterwards recorded by Mr. T. Wilkinson King, neither case having been admitted into the wards. These we propose to detail, with the subsequent case which occurred in 1837. From that period to the year 1856, a lapse of nineteen years, no case is to be found recorded. Two we saw in the year 1865. Another has presented itself in this year (1866) in our private practice.

We will first give the details of the seven cases that have occurred since the year 1829.

Case 1 occurred in the year 1831.—The lad, a private patient of Mr. Boddy's, at Walworth, was about ten years of age, and about three months ago was bitten by a dog, which animal appeared to be sulky and retired in its manners. The dog not only bit this lad, but also two other children, and the parents were so struck with the appearance of the beast, that although not belonging to them, they had it destroyed. The lad was attacked previous to the appearance of the hydrophobic symptoms with pain in one of his arms—it was thought the opposite to the limb bitten. He appeared generally unwell, and soon after, at intervals, showed a disposition to snap with his mouth and bite, to which he said he felt a strong inclination. It was remarkable that he talked of dogs, appeared to have a horror of them, and in his occasional fits of delirium called out "I won't be bitten by the dogs." He had a good deal of spasm in his paroxysms, and exhibited the usual horror of water, reluctance to swallow, and extreme irritability which characterise his formidable malady. It is not certain that he ever had an idea that he laboured under canine madness, but it is known that he had not for some time after the seizure. He had frequent priapism, and a constant inclination to apply his hands to the genitals. He died on the third day, and the expression of his countenance during his last distressing paroxysm was frightful, and was described as "Satanic." In an interval of tranquillity a short time before death he swallowed some fluid with tolerable facility.

P.M.—External appearances nothing remarkable; rather thin. The membranes of the brain offered nothing remarkable except that there seemed less fluid than usual beneath the arachnoid, and that the pia mater adhered with considerable firmness to the cortical substance of the brain, the external portion of which in several places more or less perfectly separated with the membrane. Excepting the state of the cortical substance favouring the separation, and a little congestion in the cineritious matter generally, the substance of the brain offered nothing morbid in colour or consistence. The spinal cord was examined throughout, but nothing particular could be detected in its white or cineritious substance. There were some slight adhesions between the two arachnoid surfaces, but they were evidently of long standing, if not of original formation. There was a considerable quantity of watery fluid in the sheath of the cord, yet possibly not more than is natural. This was noticed chiefly in reference to a remark of Majendie's, that the evacuation of the fluid in dogs gives rise to symptoms resembling those of hydrophobia.

Chest.—There was a slight old adhesion in the right pleura, but no other trace of inflammation, either old or recent. Both lungs exhibited interlobular emphysema along their anterior margins, and to a slight degree in some other parts. There was cadaveric infiltration posteriorly. The vivid redness described as conspicuous in some subjects who had died of hydrophobia did not exist in this case. The blood in all parts of the body, in the turgid veins of the pia mater, and in the right side of the heart, where it appeared at least as dark as in ordinary cases, became rapidly of a bright arterial hue on exposure. There was little or no unusual vascularity of the bronchial tubes. The heart offered nothing remarkable except that the blood in it was imperfectly coagulated. The œsophagus was of nearly, or quite, its natural colour, but its cuticle, about the middle and at its lower extremity, was easily detached, and appeared to have been already partially so.

Abdomen.—The mucous membrane of the stomach exhibited a faint blush, which, on close inspection, was seen to be occasioned by an infinite number of very minute red points, amongst which the follicles might be perceived as somewhat larger, but less numerous, opaque whitish spots. These appearances were most distinct about the middle of the stomach, near its smaller curvature. After a short exposure to the air the redness became much more intense.

The other viscera deserve no particular remarks.

Case 2.—The following case occurred in the latter end of October, 1831:—A stout man, a labourer, with red hair, about twenty-three years old, married, of moderate habits, was bitten slightly on the inner edge of the palm of the hand, five weeks ago, by a dog very familiar to him. The dog was subsequently killed in uncertainty, and little was done to the wound. He was attended by Messrs. Iliff and Boddy, in Lock's Fields. His spasms were frequent and violent, he scarcely drank, but once he voided some foul frothy fluid by a mixed effort of coughing and vomiting; his senses remained undisturbed. His first complaint was of his hand and arm. A constant symptom was repeated micturition with the spasms; no priapism; cajeput oil, in ten-drop doses upon sugar, was given frequently. Spinal frictions of mercury, cajeput, and belladonna. He died suddenly. The post-mortem was performed forty-eight hours after death.

The serous lining of the dura mater seemed somewhat injected, and was covered with a little very slightly sanguineous serum. The vessels of the pia mater were numerous and full, and the membrane, containing a very little serum, parted from the convolutions readily, leaving them natural in texture, but discoloured with blood effused at the time, and very easily spread out from each other. Rather an undue quantity of fluid existed in the ventricles and at the base. The pharynx and œsophagus were rather extensively ecchymosed. The mucous lining of the stomach was large and loose, it was bathed in a dirty fluid in which were suspended some globules of oil; the membrane was dark, partially injected, and somewhat thickened and softened. Nothing abnormal in any other viscus.

Case 3.—J. N. L—, æt. 35, a man with a dark complexion and rather below the ordinary stature, born in London, formerly addicted to drinking, but of late years more abstemious, was admitted under Dr. Addison's care into Billet Ward on the 1st October, 1837, at 10 a.m. The only history to be obtained from his wife was as follows:

She stated that for the last three weeks he had been in a low desponding way, so much so as occasionally to be confined to his bed for a day or two; he, however, continued to follow his usual occupation, which is that of a fishmonger, until last night. His wife heard to-day for the first time, from one of his companions, that about three months since he was bitten in the hand by a spaniel dog; the wound was trifling, did not bleed much, and produced but slight pain.

Yesterday morning he took his breakfast as usual, and left home to follow his occupation; he returned about ten in the evening, complained of feeling very unwell, and said that during the day he had been in a very agitated state; his wife took him to a surgeon, who gave him some medicine, which, as well as some tea, he took occasionally during the night; he was very restless, and slept only at intervals.

His present symptoms are the following:—Countenance anxious and dejected; great difficulty of breathing; this of a convulsive character, and made much more violent by any sudden motion, a breath of air, or sight of water; his pulse 80, with little power; skin natural, tongue parched; he complains of great thirst and slight pain in the head; pupils contracted; bowels not open since yesterday morning. He states that his own feelings are a peculiar agitation of his nerves, similar to the pricking of needles, and a sensation of suffocation, as if from drowning; he also complains of tightness across the larynx, which seems to produce the difficulty of breathing.

At 10.30 a.m. Dr. Addison saw him, and ordered him to be cupped over the epigastric region; not more than eight ounces of blood were obtained. The cupping excited him so much that it was thought advisable to desist. He also had an injection of Haust. Sennæ c. Ext. Coloc. co. ʒj, and two drops of croton oil to be taken directly, and repeated every half-hour until it operated; the enema quickly returned with a small quantity of feculent matter; he swallowed his medicine with great difficulty, wishing to have his eyes closed whilst taking it. A liniment composed of Ext. Belladonnæ ʒij, Ung. Hyd. ʒj, was ordered to be rubbed on his throat; the friction produced so much agitation that it was thought proper to apply it spread on lint.

At 3 p.m. Dr. Addison again visited him; symptoms on the increase; the croton oil had acted powerfully, stools watery and of a green colour; pulse much the same as in the morning; skin covered with cold sweat; pupils since the application of the belladonna dilated; the secretion of urine scanty; priapism. The purging was so violent that he was ordered ʒj of Vin. Opii immediately, and afterwards ♏xl of the Liq. Plumbi Diacet. every hour. The purging ceased shortly after he had taken the opium.

At 10 p.m. he was seen by Dr. Addison. He had taken the Liq. Plumbi Diacet. regularly every hour, and after the first three doses he seemed more tranquil; his pulse had become rather more accelerated, 110 in number; he complained of great thirst, and expressed a wish to have some porter; his wish being gratified, he took half a pint eagerly and asked for more, which he also drank. He still continued to take his medicine, which he was prevailed upon with great difficulty to do. He was constantly troubled with violent eructations, which became more violent after each dose of the medicine.

Dr. Addison, anxious to see the termination of the case, remained at the hospital, and paid him a last visit at half-past 2 a.m. He had been getting decidedly worse since 10 p.m. He appeared greatly distracted, imagining himself surrounded by various strange and hideous objects, and was with great difficulty kept in bed; fearing he might injure himself, he was ordered to have a strait-jacket put on; from about this period he became unconscious of everything around him; roared in a most awful manner; the muscles of his face were horribly contracted; a large quantity of frothy saliva poured from his mouth;

his eyes became livid, and he appeared writhing with excessive agony. In this lamentable state he remained until half-past 4, when he suddenly appeared quiet and exhausted, and died at twenty-five minutes before 5.

Post-mortem.—The lining of the dura mater was watery, injected, and discoloured by one or two wide, thin patches of dark adherent coagulum or fibrin. The arachnoid was opaque, much clouded and spotted. The brain and medulla healthy. The pharynx and rima glottidis were dark and thick, and in the lateral parts of the former were adherent patches of fibrin, whitish, thin, and firm. Below the level of the cricoid was an oval patch of superficial ulceration; a simple, very defined loss of epithelial lining, and a dark smooth surface with adjacent elevations as of follicles. At the corresponding point of the opposite side (both were posteriorly) a similar state of several follicles seemed to indicate the commencement of another ulceration. Nothing particular was observed elsewhere.

Case 4.—The following case is mentioned by Dr. Habershon in a paper on "Dysphagia" in the 'Guy's Hospital Reports:' —A young man, æt. 18, who was said to have been bitten by a dog five to seven years previously, was admitted into Guy's 15th May, 1854, under the care of Dr. Hughes. On the day of admission difficulty of swallowing came on and great excitement. He had had pain in his head three days previously. He was removed from his own home to one of the adjoining workhouses, and afterwards brought to the hospital about nine o'clock in the evening. He was a strong muscular man, and at first sight appeared to be affected with acute mania or delirium tremens; but there was a sudden starting, especially when a draught of cold air came in contact with his face, which more clearly indicated the character of the disease. This starting evidently resulted from spasmodic action of the muscles of the face and pharynx; his countenance had a wild and excited aspect; he thought that he was being murdered, that boiling water was dropping upon his face, and he said that he felt choked. The pulse at 9 o'clock was 90, at 11 it was 120; the tongue clear; the pupils widely dilated; the face bathed in sweat; the hands clammy; he would not attempt to drink, but dashed the cup away from him with a violent spasmodic action, but he ate a small portion of bread; he was frequently spitting out saliva. Restraint was required, for in his terror, which was fearful to witness, he rushed at the window and would have seriously injured himself.

About 12.30 a.m. an injection containing 10 grs. of cannabis indica was administered; the whole of the enema was at once returned.

At 1 o'clock a longer tube was passed, and the same quantity again injected; the paroxysms had then become very violent and frequent, and the pulse exceedingly small, varying and occasionally intermittent, 120 to 130 per minute.

At 2.15 he was still more violent, calling out as loudly as his strength would permit him. It was then determined to administer chloroform. Intense congestion of the eyes and face came on; the pupils became much smaller, the pulse a little more perceptible; the respiration, which had been catching and accompanied with gasping and sighing, became more regular. In four or five minutes after leaving off the chloroform the paroxysms began to return; the face, however, did not at once become sensible to impressions. Chloroform was administered three times during the hour, and on leaving it off the same return of paroxysm took place; the pulse became almost imperceptible, and the respiration more stertorous.

About 3 a.m., whilst under the influence of chloroform, 10 grs. of cannabis indica were placed in his mouth; it became mixed with saliva, but was all ejected. He died at 3.30 from exhaustion and apnœa.

Post-mortem.—On left knee a small white cicatrix, about one inch above the joint. Considerable venous congestion of the sinuses of the head; no disease of the brain or medulla oblongata; membranes of the cord healthy; upper part of the dorsal region of the cord rather softer than other parts. Mucous membrane of fauces much congested; constrictor muscles retracted to the utmost, so as to render every opening and canal patulous; œsophagus pale. Lungs emphysematous, parts much congested. Left ventricle of heart empty and firmly contracted; right containing a quantity of fluid blood. Every other part of the body apparently healthy.

CASE 5.—The following account is condensed from the report drawn up by Mr. Wootton Bushell, and published in the 'Med. Times and Gazette' of January 21st, 1865 :—

George R—, æt. 13, always healthy, was admitted into Guy's Hospital January 16th, 1865.

On December 18th, twenty-nine days ago, when returning home from school, he was bitten in the upper lip by a large dog; the dog was then under treatment for madness, but got loose in some way. The dog subsequently bit slightly a little girl in the hand, and was immediately afterwards killed. The boy went to a surgeon's directly, who within a quarter of an hour pared the edges of the wound and adapted them by pins as in the operation for harelip. The pins were removed in nine days, and very soon afterwards the wound was healed. He had severe rigors just before the pins were removed.

On January 12th he began to complain of headache, lassitude, and stiffness of the face, which commenced in the right side, where the scar was, and afterwards extended to the left. On the two following days he was not well, but on Sunday morning, the 15th, he first felt a difficulty in swallowing liquids, and had a spasm when trying to drink his tea, and stiffness of the neck came on. He swallowed a teaspoonful of gruel on Sunday with difficulty. On Sunday night he was very restless and did not sleep; at 2 a.m. he asked for a draught of water, but could not swallow, though thirsty.

He was admitted into the hospital at 12 o'clock on Monday morning. He had an anxious, frightened expression of countenance. The intellect was quite clear, and he answered questions intelligibly although unwillingly, because talking seemed to bring on spasm of the cervical muscles. He had a convulsive attack when first placed in bed, owing to the draught caused by throwing a blanket over him. The scar on the right side of the upper lip was like that after the operation for harelip. He said there had not been any pain, itching, or numbness, in the part, and there was no evident inflammation or swelling. He complained of pain in the neck and face. There was no spasm when in bed. The breathing was very peculiar, accompanied by sighing; respirations 14 to 16, laboured; pulse 92 to 98, irregular. When a glass of wine was brought he declared that he could not take any, but when pressed he raised the cup to his lips with a determined air, and succeeded in swallowing a little with much difficulty, but immediately spasms of the cervical and thoracic muscles came on, with a general convulsive fit. He then fell back into the bed exhausted and panting for breath. When asked some time after to take more he refused, and seemed almost to have a spasmodic attack at the thought of it. In a few hours, the other symptoms continuing, he had pains in both arms and legs. He had an enema of six grains of quinine.

At 7 p.m. for the first time he began to spit. The gas in the room was turned down, as the light troubled him. The starting spasms and inability to swallow continued to increase. The spasms were so severe that in one of them he threw himself out of bed. In the middle of the night, after several spasms, he became somewhat strange in his manner; he wanted bread and butter, but no fluid, the sight of which caused a spasm. The injection was now administered with difficulty.

At 3 a.m. on the 17th, the day after admission, the report says:—For the past forty-five minutes he has been in a state of constantly recurring spasms. At the onset of a severe spasm he springs up in bed, then puts his hands furiously to his throat, as if to tear something away; the head is thrown violently back, the mouth opened and the eyeballs protruded; then he makes several expiratory efforts, sometimes with a shrill screaming cry; the head is thrown violently from side to side, and the hands are tossed wildly about, beating his chest and striking anything that is near; the spasm generally ends by the expectoration of a viscid mucus, which recently has been tinged with blood; pulse 114; respirations very irregular, and so interrupted by the spasms that they cannot be counted.

4.15 a.m.—He is now in a state of almost constant agitation, throwing himself about in the most frantic manner. Skin so irritable that the least touch throws him into a spasm. Mind wandering.

6 a.m.—He has become so excessively violent that he is kept in bed with the utmost difficulty. Fights and struggles most violently, screaming and shouting, and spitting in all directions. Keeps coughing and hawking up a viscid bloody mucus.

About a quarter before 8 he answered questions intelligibly.

He swallowed a mouthful of wine pretty well with coughing and some spasm, and then asked for more, drinking altogether from two to three ounces; there was then very little general spasm, it being confined to the pharyngeal muscles. In about half an hour the pulse was imperceptible, he became insensible, and quickly died.

At the post-mortem, which was made by Dr. Wilks, there was nothing abnormal seen with the exception of redness of the tongue and fauces, and injection of the larynx and upper part of trachea. The medulla oblongata and spinal cord were prepared according to Mr. Lockhart Clarke's method by Mr. Durham, and carefully examined. The thin sections showed extreme congestion of the gray matter both of the anterior and posterior cornua of the spinal cord, especially in the cervical and dorsal regions. In different sections numerous minute patches of extravasated blood were found, and in every one the vessels were noticed to be most unusually distended and full of blood.

CASE 6.—*Hydrophobia, treated by the subcutaneous injection of atropine; death; autopsy.*

For the following report of a case of hydrophobia treated by the subcutaneous injection of atropine, and for the account of the autopsy, we are indebted to Mr. Eastes, the Surgical Registrar of the hospital, who took special notes at our request whilst acting as house-surgeon.

J. K—, æt. 48, was admitted into a private room adjoining Petersham Ward, under the care of Mr. Cooper Forster, on December 5th, 1865.

He had always been healthy and temperate, and never had a severe illness.

Seven weeks ago he was standing in a shop when a "King Charles" spaniel ran in; he attempted to take up and caress the dog, when it bit him on the right forefinger. He again attempted to stroke it, and was a second time bitten in the left hand. He then dashed the animal against the wall, so that it died the same evening of the injuries it had received.

The wounds on his hands, which were slight, were never painful; they soon healed.

The patient remained quite well till six days ago (November 29th), when he first complained of pricking and shooting pains about the arms and chest, and twitchings of the muscles of the arms.

On December 1st he became unable to drink water, nor has he since taken any. Has scarcely slept during the last four days; when he has dozed off he has soon woke in a fright, fancying he could see hobgoblins, &c. He, however, is quite aware that these are unreal, merely spectral illusions conjured up by his brain.

On admission (December 5th) is restless, and his eyes have a wild appearance. Talks sensibly upon any subject, but is not very communicative, and seems to court quietude. At intervals, not continuously, works his mouth and jaw about nervously, as if kneading something in his mouth. Often attempts to clear his throat, and then spits a white frothy liquid. Cannot take water, the sight or suggestion of it causing him to be violently convulsed about the neck and mouth; consequently has swallowed no limpid fluid for four whole days. Can take solid food or thick liquids, as arrowroot, gruel, &c. Respirations 23.

He took a mutton-chop and some rice pudding for supper, and ten grains of Dover's powder in the rice.

December 6th, 9 a.m.—Has been very restless all night, and out of bed several times. Two male attendants are at hand to restrain him. He talks ramblingly; says he sees dogs in the corner, and feels their paws on his arm. Eyes have a very wild look, and wander about the room; face wears a peculiarly anxious expression. Still spits a frothy liquid, apparently saliva, for it seems to collect in his mouth, and not to come from the chest. Takes solid nourishment pretty well, but experiences greater difficulty in swallowing thickened liquids. Could manage arrowroot pretty well yesterday, but now it almost chokes him with spasm about the throat, besides giving him a severe hiccough when he attempts to swallow it.

The diagnosis of the case is exciting much curiosity, some contending that the symptoms are those of mania, whilst the majority affirm that it is a case of true hydrophobia. Mr. Forster agrees with the latter, and orders 1-48 gr. of sulphate of atropine, dissolved in ♏x of water, to be injected beneath the skin by the House-Surgeon every two hours.

1 p.m.—Has just eaten the wing and breast of a chicken with difficulty, but can take no fluid.

9 p.m.—The atropine has been injected six times over the region of the great pectoral muscles—viz., at 10 a.m., 12 noon, 2, 4, 6, and 8 p.m., so that in all 1-8 gr. has been administered. The patient is now thoroughly under the influence of the drug. Pupils very dilated. He is much quieter than in the morning; altogether seems weaker; his voice also has become enfeebled. Has muttering delirium, but can be roused. Spits all over the room, not being sensible enough to use a vessel as at first. P. 100, full.

10 p.m.—An injection of the atropine is again given. He swallows two teaspoonfuls of tea with a little effort. Has passed about eight ounces of perfectly clear urine.

Midnight.—Pupils fully dilated; P. 130; tongue dry. Has slight spasm of muscles of face, and twitching movements of the arms and hands. Has swallowed a little beef tea and arrowroot.

The hypodermic injection of atropine to be discontinued for a while.

7th, 1 a.m.—Very restless, attempting still to get out of bed. Speech incoherent, and words very feebly uttered. Spits about the room in all directions. Is able at this hour to swallow a little rice boiled in milk, but vomits most of it almost directly.

2 a.m.—Some toast sopped in tea is given him; he shortly vomits it together with a greenish liquid on to the floor.

3 a.m.—Restless. Cannot take any fluid. Clutches at the bedclothes with his hands, and carries them to his mouth.

4.30 a.m.—Pupils a little less dilated, and slightly influenced by light. P. 122, weaker and smaller. R. 28. Spasm of the pharyngeal muscles almost continuous; in the intervals there is feeble muttering delirium. Whole of body bathed in perspiration. He is constantly spitting a very tenacious saliva. Subcutaneous injection of atropine (1-48 gr.) administered.

8.45 a.m.—Pupils well dilated; slightly acted upon by light.

P. 134. R. 34. Spasm (at intervals) of the muscles of the neck and of the arms. He continues to spit about, but the sputa are less viscid. Since 4.30 a.m. (to-day) he has vomited on three or four occasions some greenish fluid like that spoken of at 2 a.m. Atropine (1-48 gr.) again injected.

10.30 a.m.—Pupils dilated. P. 134, much weaker. R. 24, very irregular, and taken with difficulty. Spasm of muscles of face and neck, with jerking movement of arms. The spitting has ceased. He swallows some soft bread, but the greater portion of it is soon vomited.

10.50 a.m.—Spitting returned; sputa viscid. If a stranger enters the room, the patient watches until he has collected sufficient saliva in his mouth, and then spits at him.

11.45 a.m.—Has just vomited a quantity of very dark fluid. Spitting very much increased. Talks more than he has previously done, but with a very feeble voice. Tells persons on entering the room to be careful, yet is scarcely conscious.

12.15 p.m.—P. 142, very weak. Sputa darker, and the spitting very abundant.

2 p.m.—Atropine (1-36 gr.) injected. Patient to be kept very quiet.

3 p.m.—Much weaker. Pulse not to be counted at the wrist. R. 16—18, very irregular, consisting of one deep inspiration followed by three or four imperfect respiratory efforts. Spitting ceased. Feet and hands cold; skin generally clammy.

4.20 p.m.—Has somewhat lost the spasm in the neck, face, and hands. Has vomited a little dark-coloured fluid. Is quiet, insensible, and apparently moribund.

5.25 p.m.—Death.

Copy of Dr. Moxon's report of the examination of the body, conducted twenty-one and a half hours after death.

Rigor mortis strong.

General dusky purple tinge of surface of body, chiefly marked in dependent parts. Ghastly, half-sardonic face.

On either forefinger's terminal phalanx could be made out the trace of a scar, but nothing very definite.

Veins of dura mater and arachnoid full of blood (neck unopened). Membranes of brain thin. No diseased state of brain observable.

The section of the lower part of the medulla oblongata, or rather of the upper cervical portion of the cord, softish—decidedly softer than common.

Tongue brown and dryish. Glands at the base about the usual size. No tenacious mucus about the fauces, less, in fact, than usual. No injection of capillaries. Tonsils white, the right having in its lower third a small cavity containing a pea-sized quantity of purulent-looking fluid, with no injection about the cavity.

Larynx quite healthy.

Lungs, right and left, collapsed freely.

Intestines considerably distended.

Liver very supple and flabby, otherwise normal.

Kidneys healthy looking, congested with fluid blood.

The following case occurred lately:

Case 7.—A gentleman living in Manchester, on a visit to friends in London, called upon me on the 17th of May, 1866, and gave his history as follows:—He was not otherwise than healthy, æt. 34, a brewer; last June he was bitten by a dog in the leg; upon striking the animal to punish him, the creature seized his right hand. About half an hour previously the dog had bitten one of the men in the yard; this man is still quite well; the animal afterwards had convulsions and was shot. Immediately after the accident the hand was plunged into brandy, but not the leg; he was then taken to a surgeon, who first applied Tr. Ferri Sesquichlor. and subsequently Arg. Nitr. This treatment kept him an invalid for six weeks, and he subsequently stayed away from business another six weeks to recruit his strength. He has had twitchings in the hand and pain up the arm three or four times since he has been considered quite well, and the last few nights the pain has kept him awake. He now is suffering from pain of a most severe character, occurring in paroxysms along the right arm and obliging him to cry out. These paroxysms first came on early in the morning, and keep increasing in frequency. Yesterday he was so entirely free from pain that he was at "The Derby," and apparently enjoyed himself as well as any one. There is not any redness or tenderness in the cicatrices on the hand or leg, and he has no idea that they have anything to do with his pain. He appears in very good health. There is a marked difference between the temperatures of the two arms, the right, on which side the bite occurred, being much colder than the opposite. The pain in the arm cannot be localised in the distribution of any special nerve or nerves. He was ordered two large doses of quinine to be taken directly; and if no relief was obtained, to have morphia injected subcutaneously. Whilst walking home with his friend he stopped suddenly two or three times, and cried out with the pain in his arm. The arm was bent to a right angle when the pain came on, but could be moved as he liked when the pain subsided. Towards the evening the pain increased a good deal, and we saw him in consultation with Mr. Joyce, at Kensington, who had injected $\frac{1}{4}$ gr. of morphia previous to our arrival. He was placed in bed after having drunk a couple of glasses of hock without any difficulty under our observation; we then injected $\frac{1}{2}$ gr. more of morphia, as there was an intimation to him that the pain was about to recur. He slept well for four hours, that is, until 3 o'clock in the morning, when, upon attempting to swallow, he felt uncomfortable about the throat, and found he could not complete the act of deglutition; he, however, went to sleep again, and at 7 a.m. was seen by Mr. Joyce; he then had some cocoa brought to him, which he could not get down, though he ate some biscuit; he was then observed to attempt to clear his throat of mucus, with a peculiar noise, compared by those in the house to a bark of a dog. He was immediately, by desire, removed home to Manchester, accompanied by Mr. Joyce, who, previously to starting, injected 1 gr. of morphia subcutaneously. The patient bore the journey well, without pain, sleeping part of the way; during the transit he complained of thirst, and succeeded in getting down a little soda water, but with much difficulty.

At 5 p.m. on that day, the 18th, he was seen by his former medical attendant Mr. Turner, Mr. Williams, and Mr. Joyce. He does not appear to have complained any more of pain in his arm; there was not any intellectual disturbance; he was cheerful to his wife and those around. He occasionally hawked up some saliva into a handkerchief, which he was particularly anxious should be destroyed, fearing lest some one should be infected by the sputa. He complained only of an exhausted, faint feeling. The morphia hypodermic injections were continued. It appears he remained in the same state up to 4.30 a.m. of the 19th (the following day), when he had the first of a series of violent convulsions, accompanied by a barking cough and spitting; he was perfectly sensible during the intervals between the convulsions, which continued up to 8 a.m., requiring the united efforts of several men to keep him in bed. He suffered apparently most fearfully. At 8 o'clock he became insensible, and continued foaming and spitting until 9.25, when he died.

There was not any post-mortem.

The first interesting point in connection with cases of hydrophobia is the question; how long after the bite may hydrophobia occur?—a point rather fully investigated many years ago by the late Dr. Bardsley, of Manchester, in connection with a case of the disease published by him in the year 1805.

From one day to forty years is spoken of. But obviously either the poison acts slowly or rapidly, and one or other of these times must be incorrect. So far as we can speak in the matter, the poison is undoubtedly slow in its action. Preferring ourselves to take cases that have occurred in our hospital and under our own observation, leaving others to draw deductions from well-authenticated cases of their own, it appears to us that there is a considerable latitude in the period of incubation.

In thirteen cases, including the six cases published by Dr. Bright and the seven here given, the shortest time that elapsed was four weeks, and the longest—in one case only—five to seven years; the other eleven all took place at various periods within eleven months. Here we have the results of a poison apparently more tardy in its action than any other by which the human body is assailed. All other morbid poisons, such as that of syphilis, and even the more subtle and inappreciable, as that of pyæmia, appear to have some definite time of action. We know that syphilis never, under any circumstances, exceeds a month from the time of the contact to the appearance of symptoms; and we very much doubt if even so long a period as that can be allowed it. Again, the pyæmic poison ceases to be dangerous directly its source is removed. In hydrophobia, however, the poison may apparently lie dormant in the system at least a twelvemonth before showing its effects. If this be the case, it is scarcely possible that it can show itself in one day, and equally improbable that forty years should elapse.

On reviewing the cases above detailed, it naturally occurs to inquire whether there are any means by which we can predict the probable interval at which symptoms may manifest themselves—whether there are any circumstances which would lead us to expect an unusually rapid or an unusually late appearance of the disease. If from so small a number of cases any deductions may be drawn, it may be pointed out that when the disease appeared early, the bitten part was situated in the most vascular and sensitive region of the body, where the circulation is most active, and where we might naturally expect absorption to take place most rapidly. Besides the one recorded in this paper, two cases are mentioned by Dr. Bright, in which the patients were bitten on the lip; in these a period of four or five weeks only elapsed before symptoms of hydrophobia appeared. In those in which the hand was bitten, from five to seventeen weeks transpired, and in one case even as many as forty-seven. In the case related by Dr. Habershon the part of the body bitten is not mentioned; but in the report of the post-mortem allusion is made to a cicatrix upon the knee, a part where, if the poison was received, it might be fairly supposed that, owing to the presence of the clothes, the effects were less likely to be shown early than where no means of getting rid of at least a portion of the poison existed. In this case an interval of from five to seven years occurred; this is, however, an exceptionally long time. The clothes may be considered, we think, to have been a very great preservative in many cases against the poison for a time, though only for a time. It is possible that this may be the explanation of the tardy appearance of the disease in this case. In the case of Dr. Bardsley's, to which we have before alluded, the bite which appears to have been the cause of the hydrophobia occurred twelve years previously on the lower extremity, a part protected by clothing.

We may then, perhaps, draw some such conclusions as the following:

1. That when a bite has occurred on the face a rapid appearance of the disease may be looked for; that a few weeks, probably four or five, will elapse before evidence of it is shown, and therefore that if those few weeks pass over safely an immunity from the malady may be expected.

2. That when the bite has taken place on the hand a still longer time, from five weeks to a year at least, must be looked forward to with much anxiety. It may be that in the single case among the ten in which a year elapsed before hydrophobia occurred there was some special reason for the delay; to this we shall advert when speaking of the treatment.

3, and lastly. That when the clothes have been bitten through before the skin is injured some years may pass ere the disease occurs.

Some may be disposed to think that when hydrophobia appears some years after the occurrence of a bite it can scarcely be considered to be owing to the wound then inflicted. That the case No. 4 was an undoubted specimen of the disease, none of those who saw the patient could hesitate for a moment to pronounce, but whether such a disease as spontaneous hydrophobia can occur may be another question. This appears to be the only solution of the difficulty, but it is one which we cannot adopt. That cases do occur resembling hydrophobia in several particulars, except in the termination, there is no doubt; one was mentioned in 'The Times' newspaper not long ago, and vaunted as hydrophobia cured by salivation. No one who read that case could have the slightest hesitation in pronouncing it to have been hysteria. Several instances are on record of hysterical dysphagia, in which inability to swallow liquids was the marked symptom, and in the accounts of many, indeed of nearly all these cases, barking is adduced as a characteristic symptom, as if the disease must therefore be hydrophobia. In fact, however, though in well-marked cases of hydrophobia the former is a constant symptom, the latter is by no means invariably present. Dr. Bright mentions this hysterical dysphagia occurring in a woman forty years of age; the patient, of course, recovered.

In none of the cases that we have seen has any change occurred in the original wound previous to the appearance of the hydrophobic symptoms; some of the older writers, however, allude to a redness of the wounded spot. Indeed, in the article on "Hydrophobia" by Messrs. Gamgee, in Reynolds's 'System of Medicine,' under the symptoms of hydrophobia in man, it is said "the wound becomes intensely painful." "If the wound have cicatrized, the cicatrix becomes red and irritable; or if, as is generally the case when the injuries inflicted by the rabid animal have been severe, it has not healed, it assumes an unhealthy appearance."

Now let us inquire whether these remarks accord with the facts observed in our cases.

In the first case reported by Dr. Bright it is stated "the circumstance of the bite did not at the time come to the mind of either the patient or the surgeon;" surely it would have done so if any change had taken place in the wound.

In the second case "the scar of the bite was evidently inflamed."

In the third case there was "a tingling sensation in the palm of the hand."

In the other three cases of Dr. Bright's there is no allusion to the cicatrices, clearly showing no change had taken place.

In none of the cases we have detailed was there the slightest alteration in the wounds, and three of them we carefully examined. Though quite prepared to admit, therefore, *à priori*, that such a change as described by Messrs. Gamgee would be likely to occur, yet, since in only one out of the thirteen above referred to any alteration in the wounds was observed, we must hold it a very exceptional occurrence. On this point we can speak positively from our own experience.

That there has sometimes been great pain in the course of the nerves leading from the injured part there is no doubt. Indeed, this appears to be one of the most marked symptoms of the commencement of the malady, and one which first attracts attention.

In Case 7 the pain was more severe than in any other we ever witnessed, and in Case 6 it was exceedingly severe up the arm. In the former patient it occurred in the most paroxysmal manner; in the latter case (No. 6) the whole train of symptoms from the commencement was less marked, so much so that a question of diagnosis between hydrophobia and mania was raised once or twice in the early stage of the disease. Nevertheless, in this case, though no change took place in the wound, the same peculiar pain occurred, leading from the originally injured part.

When the bite has been in the face the same kind of pain has been observed about that part and the neck—we believe along the course of the nerves, and not along the track of the absorbents, as there is no enlargement of the glands or lymphatics at all indicative of any irritation.

The excessive pain is not, so far as we know, at all like that of any other disease, particularly when associated, after a longer or shorter interval, with the characteristic difficulty in swallowing. This latter symptom is not a dread of fluid, but an inability to perform the act of swallowing when the liquid is taken into the mouth.

In all the cases we have seen, the patient has taken the glass, or mug, or spoon, and earnestly wished to get the liquid down, but has found it practically impossible, apparently owing to an irregular contraction of the muscles of the pharynx.

We think these two symptoms taken together sufficiently characteristic of hydrophobia without any other. It has been said that there are no positive signs of this affection, but we consider those above mentioned quite sufficient. We know of no disease presenting at all the like symptoms.

Upon a careful examination of all the post-mortem appearances related, there seems to be no special organ which claims particular attention. But it may be observed that formerly even the condition of the pharynx, which is now generally admitted to be dilated, was not noticed. Whether this condition is cause or effect appears exceedingly difficult to decide, but we have seen an enlargement of that cavity quite out of all proportion to the œsophagus beyond, and causing an open condition of the Eustachian tubes: this was very peculiar, and especially to be noticed in Case 4.

The variety of treatment adopted shows at once the utter despair of achieving any good result felt by those who have had these patients under their charge, and we do not see how anything else can be expected unless we arrive at some more satisfactory solution of the pathology of the disease. As matters now stand, the treatment appears to be most empirical, though we doubt not that each surgeon or physician has had a reason for the plan he adopted. We can speak positively on this point in our own cases, which have been two.

In the first of Dr. Bright's cases bleeding was adopted, though the man was fifty-two years of age :—it was the fashion, or perhaps the necessity, to bleed patients at that time :—the poor fellow lived forty-six hours.

In the second case of Dr. Bright's nitric acid was applied to the wound by a medical man, and when the attack came on calomel, antimony, and opium were ordered, but it appears little good was done. The patient died in thirty hours.

In the third case of Dr. Bright's amputation of the arm above the elbow was had recourse to, and the patient lived forty-eight hours.

In the fourth hydrocyanic acid was given; the patient lived forty-eight hours.

In the fifth subacetate of lead was the remedy tried; the patient lived sixty-three hours.

In the sixth and last of Dr. Bright's cases an injection of half an ounce of rectified ether was administered, and repeated two or three times, with opium. The man lasted seven days.

In the first case mentioned in this paper it appears that nothing was done; at least, no particular remedy was tried, and the patient lived three days.

In the second case cajeput oil was used.

In the third case, Dr. Addison, under whose care the patient was, appears to have given free purging a good trial, with the result common to all the cases—death. This occurred in forty-eight hours.

In the fourth case, mentioned by Dr. Habershon, cannabis indica was used as an injection, and chloroform was afterwards administered.

In the fifth case, which occurred but four weeks after the bite, excision of the injured part had been performed immediately, and injections of quinine were tried; the boy lived seventy-two hours.

In the sixth and seventh cases two of the alkaloids were summoned to our assistance, with the like result in both cases; in the former case, however, the patient lived six days, in the latter only forty hours.

In the sixth case atropia was used, as we have seen. We were anxious to try the full effects of this drug, which Pereira has classed amongst the phrenics and anæsthetics, as being one of the most powerful of those medicines which have an especial influence over the cerebro-spinal system. Upon referring to the report it will be seen that a portion under the quarter of a grain was injected in twenty-eight hours, and there is no doubt that, after eleven hours of its use, the patient being fully under the influence of the drug, a marked quiescent condition appeared; the hydrophobic symptoms seemed to abate. Thus, he swallowed some tea with a little effort, and afterwards some beef-tea, although he had taken no fluids for five days before. The delirium from which he suffered before the atropia was given disappeared. The spitting about the room in which he indulged was more confirmed after the atropia than before.

After the injection of the 1-36th of a grain, at 2 p.m., December 7th, he rapidly sank, whether from the effects of the atropia or the hydrophobia, or whether from both combined, it would be difficult to say.

In the seventh and last case nitrate of silver and perchloride of iron had been applied pretty freely after the bite, so much so as to cause considerable suppuration in the hand, and to render the patient seriously ill for nearly six weeks. Hence, if the poison could be eliminated by these means, this was a fair opportunity for such a result. The delayed appearance of the symptoms may possibly have had some connection with the previous treatment.

Upon reviewing these cases, the results, and the treatment, can we indorse the remark in Cooper's 'Surgical Dictionary?'—that "happily, surgery possesses one tolerably certain means of preventing hydrophobia when it is practised in time and in a complete manner. Every reader will know that the excision of the bitten parts is the operation to which I allude."

Dr. Watson says, "The early and complete excision of the bitten part is the only measure in which we can put any confidence."

In the only case we have reported where excision was practised the disease appeared in four weeks. The bite occurred at a part where complete removal was easily practised without involving any serious complication, and yet it failed; and we believe it is much more likely to fail when practised in other parts of the body, where complete removal cannot be so easily performed. In reality, the only sure method of employing excision appears to us to be to amputate the member at a part between the bite and the body, and that immediately after the infliction of the injury. We should carry out the practice thoroughly, or not at all. We quite agree with Abernethy, and adopt the practice consistent with his opinion, that a patient bitten by a dog, whether mad or otherwise, should be treated on general principles applicable to all injuries. If he be about to have hydrophobia, nothing, so far as we know at present, can prevent it; and we believe that we shall not be able to achieve such a result, until we are better acquainted with the pathology of the disease. Excision or non-excision is alike in vain; sooner or later the disease appears.

The practice adopted at Guy's Hospital in the treatment of bites, whether by dogs, horses, men or women, donkeys, cats, rats, or foxes, varies according to the fancy of the dresser. During the last six months all have had nitrate of silver freely applied. Formerly nothing was done beyond treatment on general principles, except that in some months the house-surgeon or dresser applied strong nitric acid to the wounds. The nitrate of silver, however, as adopted by Youatt in his own person, has found the greatest favour.

THE TEETH,

AS

PASSIVE ORGANS OF SPEECH.

By S. JAMES A. SALTER, M.B., F.R.S.

The teeth play a very important part in the formation of articulate sounds: they constitute an essential element in the organs of speech. Without them the precise and clear pronunciation of a great many letters, particularly consonants, would be impossible, and the resources of the oral cavity, as an organ of speech, greatly circumscribed. Accordingly we find that when the teeth are lost certain imperfections in articulation are immediately entailed; and thus the knowledge of the particular way in which the teeth help to form articulate sounds is of much practical importance to the dentist, as it is only by this knowledge that the imperfections of articulation will enable him to tell what is amiss, how to correct it, and, indeed, whether the teeth are in fault or not.

With the formation of many articulate sounds, both vowels and consonants, the teeth have nothing to do, and, therefore, into the discussion of the mechanism by which these are produced it will not be necessary to enter. Nevertheless I think it well, before describing how the sounds are produced in which the teeth are immediately concerned, to say a few words on the mechanism of articulation generally, as it will render the more special part of the subject clearer and more intelligible.

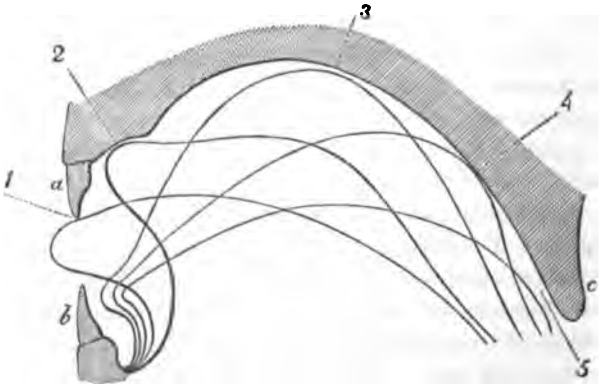
Articular sounds are essentially produced by the passage of air through the oral canal. Now, there are chiefly three parts of the oral canal that may severally impress certain characters on air passed through them, and these are the three points where closure, *perfect*, *imperfect*, and *modified*, may be produced at will—namely, the soft palate, the front teeth and gums just behind them, and the lips. Certain characters are also given to the sounds by the relation of the size of the oral cavity to the size and shape of these orifices.

Articulate sounds have been divided into *vocal* and *consonantal* sounds, or vowels and consonants, and to a certain extent the distinction is a good one and based on physiological truth; but as it is usually understood, and as we find it explained in grammars, it is quite incorrect. It is usually stated that vowel sounds are the true voice sounds, and that consonants are merely the method of commencing and terminating vowels; that vowels are open sounds, and consonants closed sounds; that vowels are capable of prolonged utterance, and that consonants are incapable of pronunciation without the aid of vowels. We see this implied in the very names themselves—vowels (*vocales*), voice-sounds; consonants (*con sono*), something necessarily sounded with something else.

Now, this error has arisen, as shown by Müller, from the supposition that voice sounds were necessary to articulate utterance; that articulation was, in fact, nothing more than the modification of laryngeal vibrations; whereas in truth almost all articulate sounds, both vowels and consonants, may be pronounced in a whisper without any vocalisation, as any one may satisfy himself in a moment; thus, V or S may be pronounced as well in a whisper as with the superaddition of the voice, and the sound prolonged with as much facility as the vowel sounds E or O. Indeed, in following my remarks on this subject and verifying for himself my description of the way in which the sounds of the different letters are produced, I should advise the reader to pronounce them in a whisper, and not to speak them "out," as by eliminating the laryngeal vibrations he will get the oral element of the sounds more unmixed and simple, and their analysis will be easier and more precise.

In what, then, does the essential difference between vowels and consonants consist? I think mainly in this—that while vowel sounds depend on the relation of the size of the oral cavity to the size of its orifices, consonantal sounds depend on some superadded condition of orifice quite independent of size. To take an example—in *far* and *fear* the difference is in the vowel sound, and the difference of condition is in the *size* of the oral cavity and aperture, both being more capacious in *far;* but in *oth* and *oss,* where the difference of sound is consonantal and the vowel sound the same, we find the size of the cavity and its relation to the orifice to be unchanged, but that the dental orifice is modified by the altered relations of the tongue to the teeth.

I am aware that this definition can only be stated approximatively; for as there is no real physiological distinction between some vowels and some consonants, so no definition can be laid down that would separate the whole of the one class from the whole of the other. This is illustrated in the accompanying diagram. It represents a vertical section,



Explanation of Woodcut.—Diagram intended to illustrate, by section, the relation of the tongue to the teeth and palate in the production of several sounds.

a. Superior incisor tooth.
b. Inferior incisor tooth.
c. Uvula.
1. Application of tongue to incisor tooth in producing the sound "*th.*"
2. Tongue to palate, sound "*d*"—closure complete; sound "*s*"—closure incomplete.
3. Sound "*ch*" (*German*), as in Liebchen.
4. Sound "*g*" hard and "*k.*"
5. Sound German and Scotch guttural, as "*Och!*"

in the middle line, of the upper jaw, the soft palate, and the tongue in various positions, and shows that the apposition of different parts of the upper surface of the tongue along the under surface of the soft or hard palate, or to the gum behind the teeth and upper incisors, produces the sounds of various letters, five of which are consonants and one a vowel. Thus, if the back of the tongue is applied to the soft palate the sound of K is produced; if the sound of the Scotch *Och* is pronounced it will be seen that the apposition takes place a little further forwards; for the pronunciation of the German *ch*, as in *Liebchen*, the apposition is still further in front; if, now, the upper surface of the front of the tongue is brought close against the front of the palate, whilst the tip is pressed against the back of the lower incisor teeth, so that a narrow horizontal chink is formed, on breathing through it the sound of E is produced; if the tip of the tongue is placed close against the gum behind the upper incisors we get S; lastly, if we carry it a little further forwards and place it against the edge of the incisors themselves, projecting the tongue a little beyond the teeth, we get *th*. Thus, the apposition of the tongue to the upper wall of the mouth at six successive points, from behind forwards, gives six definite articulate sounds, one of which is a vowel.

Thus, while the teeth have far more to do with the formation of consonants than vowels, the associated distinction between oral cavity and orifice, as connected the one with the formation of vowel sounds, the other with that of consonants, is lost.

The principal way in which the teeth assist in the production of articulate sounds is by acting as an arch, or horseshoe-shaped ridge, within which and against which the tongue may act as a valve, and by pressing against which it may produce modified and variously-placed partial or complete closure.

The outline of the tongue, when flaccid and in a state of rest, coincides with that of the alveolar arches both in size and shape; and as its border is on the same level as the line of meeting of the upper and lower teeth, the teeth and the edge of the tongue are always, when this natural condition of rest is not disturbed, in close juxtaposition. This is not strictly and absolutely true with regard to the anterior extremity of the tongue, which is slightly depressed, so that the tip of it rests, not against the line of meeting of the incisors of the two jaws, but against the back of those of the lower one. It is this approximate coincidence of shape and close propinquity that make the instantaneous application of any part of the edge of the tongue to the corresponding part of the dental arch of either jaw so easy, and this it is also that makes any considerable dental irregularity or deficiency destroy, by preventing this application, the power of producing certain articulate sounds. Hence (to state this proposition conversely) we may lay down the practical rule that all measures taken with a view to restore imperfect articulation due to the teeth must have for their object to restore this coincidence between the entire dental arch and the entire border of the tongue.

Although the tongue is so placed that its edges may be pressed with equal facility against the teeth of *either* jaw, it is against various parts of the dental arch of the *upper* jaw that it is, in actual fact, pressed in the formation of most of the dental consonants.

Whenever the border of the tongue is closely applied to the inside of any of the teeth, then closure is produced, and air cannot pass; whenever this closure is not produced, then air can pass, and then it is that the particular sound produced by this modification of aperture is generated. Thus, the seat of the sound is always, not where the tongue is in contact with the teeth, but where it is *not* in perfect contact with them. This is a point that it is of importance to bear in mind in drawing an inference as to the whereabouts of a deficiency in the teeth from the particular defect of speech that the patient may suffer from.

Upon the principles that I have laid down I have endeavoured to construct what may be called a physiological alphabet. The arrangement is based upon the situation of the closure by which the sound is produced, upon the completeness or incompleteness of the closure, and upon whether the breathing is soft or aspirate, and the table shows how completely symmetrical the whole subject is, and what a parallelism runs through it. It shows, too, to the formation of how large a number of letters the teeth contribute.

Of the consonants it will be seen that nine—B, D, G, P, TABLE.

Articulate sounds.	Vowels.			*a.*	terminally dental.
				e.	wholly dental.
				i.	terminally dental.
				o.	not dental.
				u.	inceptively dental.
	Consonants.	Closure complete, *mutes*.	Soft	*b.*	labial.
				d.	dental.
				g (*hard*).	palatal.
			Aspirate.	*p.*	labial.
				t.	dental.
				k.	palatal.
		Closure incomplete, *semi-vowels*.	Nasal	*m.*	labio-nasal.
				n.	denti-nasal.
				ng.	palato-nasal.
			Oral. Soft.	*v.*	denti-labial.
				z.	dental.
				r.	dental.
				l.	dental.
				j. English	dental.
				th. (in thou)	dental.
				zh. (= Fr. j.)	dental.
			Oral. Aspirate.	*f.*	denti-labial.
				s.	dental.
				rh.	dental.
				ll.	dental.
				ch. English	dental.
				th (in through)	dental.
				sh.	dental.
				ch. (*German*),	palatal.

T, K, M, N, NG—are subdivisible into threes in a very orderly and regular way. Three are labials, B, P, and M; three are dentals, D, T, and N; three are palatals, G hard, K, and NG (as in *thing*). Of the labials, one, B, has a soft breathing; the other, P, an aspirate breathing; and in the third, M, the sound is continued through the nose after the labial closure, and I therefore call this a "labio-nasal." In the same way the dental has a soft, an aspirate, and a denti-nasal; and the palatal a soft, an aspirate, and a palato-nasal; so that there are three with a soft breathing, three corresponding ones with an aspirate breathing, and three nasal, in which the sound is continued through the nose after the oral closure. In all these nine the oral closure, whether at lips, teeth, or palate, is complete, and those of them (the first six) in which the sound is not continued through the nose are therefore called *mutes*, because they produce complete arrest of sound.

In all the other consonants the oral closure is incomplete, and the characteristic sound of the letter is produced, not by the method of closure, as in the others, but by the particular shape and seat of the orifice or constriction produced by the partial closure. The sound in these is therefore continuous, and may be protracted any length of time, and the letters are hence called "semi-vowels," or continuous consonants. Putting aside the German *ch* (in English we do not possess it), they are fourteen in number, and may be coupled two and two, seven having the soft and seven the aspirate breathing, and each aspirate letter corresponding to one with a soft breathing. Thus V corresponds to F, Z to S, &c.; so that there are really only seven distinct consonantal sounds of this class and seven modifications of the parts engaged in their production, each of these sounds being doubled by the substitution of one breathing for the other. Two of the letters, V and F, are produced by certain relations of the teeth and lips, and I therefore call them "denti-labial;" the other twelve depend upon the relation of the teeth and tongue, and I therefore call them "dentals;" perhaps with more correctness they might be called "denti-linguals."

Let me now analyse more particularly and in order the way in which all these letters are formed. As my object is only to show the part which the teeth play in their formation, I shall not say anything about the labials and palatals.

In pronouncing D the tip of the tongue is placed firmly against the gum behind the upper incisors, and its edges against the upper alveolar arches and teeth, so as to produce complete closure, and these parts are then suddenly opened. In T exactly the same is done, only the breathing accompanying the opening is more forced. In N the method of oral closure is exactly the same, only after the closure is established the sound is continued through the nose. Thus T may be said to be D-aspirate, and N may be said to be D with nasal prolongation. This is how it is that when the nose is obstructed by cold or otherwise N becomes D; thus, "nonsense" pronounced "dodsedse" gives an idea of cold in the head.

Conversely, in cases of cleft palate, in which the posterior nares cannot be closed, and the sound therefore cannot be prevented from prolongation through the nose, D cannot be sounded, but is pronounced like N, so that a person with cleft palate, instead of saying "How do you do?" says, "How noo you noo?"

In the pronunciation of V and F the upper front teeth are brought against the lower lip and the air is driven through them —in V gently and in F forcibly. For this sound therefore the upper incisors are necessary. In old people who have completely lost their upper incisors, the gum answers nearly or quite as well; but the loss of a single incisor or of the two central incisors materially interferes with the pronunciation of these letters. It is probably in consequence of being unable to bring the thick and prominent lower lip against the upper teeth that Africans (negroes) are unable to say these letters.

In the pronunciation of consonants in which the sound depends upon air driven through a chink between the tongue and the teeth, the closure may be lateral and the aperture mesial, or *vice versâ;* thus in TH and S, the aperture is mesial; in L, the closure is mesial and the aperture is lateral.

In the pronunciation of TH the tip of the tongue is brought against the upper teeth, and the air is driven through the chink thus formed; in TH, as in "thou," with the soft breathing; in TH, as in "thick," with the aspirate.

If now the tip of the tongue is a little retracted and brought close against the gum behind the upper teeth, and a slight chink still permitted while the sides of the tongue are still firmly applied to the lateral teeth of the upper jaw (especially, as it seems to me, the bicuspids), and the air driven through this chink, the sound of S will be produced; if we substitute the soft breathing for the aspirate we shall have Z.

Let the tip of the tongue be still further retracted, so as to be brought against the edge of the gum where it makes an angle to pass up and form the roof of the palate, we shall get the sound of R; but in this case, from the way the tip of the tongue is twisted up, I think the lateral closure is not perfect, or at any rate that the chink is more laterally extended than in the pronunciation of S.

In sounding L, the closure is mesial and the aperture lateral. We make it by applying the tip of the tongue firmly against the gums behind the upper incisors, but exercising no lateral apposition—in fact, leaving a space open on each side; through these two lateral apertures the air is driven, and thus the sound is generated. Thus the difference between D and L is, that in the former there is lateral closure, in the latter lateral aperture, but the mesial closure is the same in both; and in pronouncing D and L in succession, as in "candle," we merely have to liberate the sides of the tongue from their contact with the upper alveolar arch and teeth.

SH is perhaps the most purely dental sound of any; for in its formation the air is simply driven through the closed or nearly closed teeth. In S the chink through which the air is driven is exceedingly slender and also laterally circumscribed. In SH we have a larger or coarser sibilation, the tongue not narrowing the chink, nor being concerned in the production of the sound at all. I think it is only through the teeth in front of the bicuspids that the air is driven in this sound (SH), and that it is prevented from lateral escape through the bicuspids and molars by the pressure of the cheeks, by means of the buccinator muscle, against the outsides of the teeth. Any one pronouncing the sound will feel that his cheeks are pressed against the outsides of his teeth, and that his buccinators are in a state of action; and this, no doubt, is how it is that the lips are protruded in uttering it; for, to fix the commissures of the mouth, that the buccinators may act from them with advantage, the orbicularis oris contracts, and when this is the case either the mouth must be closed, or, to prevent this, the lips must be protruded. In the lateral apertures between the teeth being closed, and the air prevented escaping through them by the pressure of the cheeks against their outside instead of the edges of the tongue against their inside, the formation of this sound differs from that of all others. That the lateral escape of air between the molars interferes with the firmness and integrity of the sound may be shown by introducing the finger between the cheeks and the teeth, and so holding the former out; the lateral escape of air at once alters the sound. The French J, which would be spelt in English ZH, is the soft breathing of which SH is the corresponding aspirate. The English J is really a compound sound, and begins with the sound of D; it is, in fact, DJ. Any one analysing in his own person how the J in "judge" is sounded, may satisfy himself of this, and that the movements of the parts concerned are those that would produce D and the French J in succession.

CH, as in "church," is the correlative aspirate of the English J, and might be spelt TSH.

Of the five English vowels, A, E, I, O, U, together with the additional vowel sounds in AH, AU, OO, only one can strictly be said to be dental. In the pronunciation of E it will be observed that the tip of the tongue is pressed against the back of the lower incisor teeth, so as to be flattened and expanded, and the air driven through a horizontal chink formed between the upper surface of the front of the tongue thus flattened out and the gum behind the upper incisors; and it would be extremely difficult to pronounce this vowel without thus pressing the apex of the tongue against the lower front teeth. But, if the pronunciation of the vowels A, I, and U be analysed, it will be observed that the sound of E enters into them all,—A and I terminating with the E sound, and U commencing with it. Thus E is a pure dental vowel; A and I terminally dental; and U inceptively dental. To these must be added Y, which so commonly has the sound of E, as in "yes" and "joy."

Now, the loss or injury of any of the passive organs of speech produces a corresponding defect in the articulation of the sufferer; and, if the teeth are the organs affected, then dental sounds are interrupted or interfered with. This is always very manifest, and by far most manifest, immediately after the loss of a tooth or teeth has taken place; for by degrees the loss is, to a certain extent, compensated by the adaptation of the soft parts, and the cultivated skill of the speaker. The injury inflicted on the speech is always more marked when the loss consists of a few teeth, and especially those towards the front of the mouth—there being thus produced a marked gap between teeth still standing. The loss, for instance, of the superior central incisors while the laterals remain, puts an end to the F's and V's of the speaker, and a blowing sound through the opening takes their places, or is superadded. When, however, several teeth are removed, and a considerable surface of gum is left free, the tongue or the lips, as the case may be, can be applied to it somewhat as they had been previously to the teeth; thus, if all the upper incisors and canines are gone, the upper gum rests on the lower lip, so as to imitate the sounds of F and V; but the want of the teeth with the little intervals between them deprive those sounds of the slight sibilant accompaniment which properly attends them, and which is produced by the passage of air between the teeth: the sound is dull and short; thus V has somewhat the sound of B, and F of P.

When lateral teeth (molars and bicuspids) are lost, the flattening out of the tongue, and the in-sinking of the cheek, remarkably fill up the gap and compensate for the loss; and thus, to a considerable extent, the consequent defect in articulation is remedied.

These compensating changes in the soft parts are not sudden, but progress by time. After a gap or a lateral vacancy has existed some months, the tongue, if the loss of teeth be on one side, loses its symmetry and bulges so into the hiatus as to fill it—at least so as to meet the cheek.

Every one who has supplied artificial teeth in such a case must have observed what is the consequence—how frequently the tongue and cheek are at first bitten by the substitutes for those natural teeth the removal of which has allowed the soft parts to enlarge and expand.

These changes in the soft parts are merely instanced as some of the compensatory conditions by which the effects on speech of the loss of teeth are remedied.

Without carrying this subject further, it may be stated in general terms that, though the loss of the teeth will injure or destroy those elements of sound which it is their office passively to assist in forming, the plasticity of the soft parts will greatly relieve or remedy such injury. Further, that an individual adapted by habit and long use to speech without teeth, is at first incommoded by their artificial restoration. But that those persons will ultimately have the best articulation who, having lost teeth, are supplied artificially by that which is the nearest approach to a perfect dentition.

ON THE

APPLICATION OF PHYSIOLOGICAL TESTS FOR CERTAIN ORGANIC POISONS,

AND

ESPECIALLY DIGITALINE.

By C. HILTON FAGGE, M.D.,

AND

THOMAS STEVENSON, M.D.

In the year 1856, about the time of the trial of Palmer for the murder of Cook by strychnine, Dr. Marshall Hall suggested[1] that the tetanic spasms produced in frogs by this poison might serve as a means of detecting its presence. This proposal, however, met with but little support from men of science in this country. It is known that other agents besides strychnia give rise to tetanic spasms when administered to frogs; and it was felt by many that, in so important a matter as a criminal trial, it would be unsafe to rely upon the physiological action of a substance supposed to contain poison, on creatures so remote from man in the animal scale. In the spring of 1864, however, a medico-legal inquiry took place in France, at which a large mass of physiological evidence was brought forward by MM. Tardieu and Roussin[2] for the prosecution. We refer to the trial of M. de la Pommerais for the murder of Madame de Pauw. In this case the materials for

[1] 'Lancet,' 1856, vol. i, p. 36.

[2] 'Annales d'Hygiène,' 1864, 2me série, t. xxii, p. 80.

analysis consisted of the stomach and intestines removed from the body of Madame de Pauw, and of the scrapings of the floor upon which she had vomited. Having ascertained by ordinary chemical methods that the viscera contained no *mineral poison*, MM. Tardieu and Roussin made no attempt to carry further a chemical analysis, nor to search for any organic poison. The symptoms noted during the life of Madame de Pauw suggested to these observers that digitaline was the agent which had caused her death, and they consequently determined to avoid any procedure which might alter or destroy this substance, supposing it to be present in the matters which came before them for analysis. They therefore simply treated the suspected matters with alcohol, and after filtering the solutions thus procured, evaporated them on a water-bath. In this way they obtained soft extracts, which were then administered to various animals, namely, to dogs, rabbits, and frogs.

We shall hereafter have occasion to refer to the details of these experiments; but it may be stated generally that the effects produced were of a very striking character, and indicated the presence of some substance acting principally on the heart. From these experiments, taken in conjunction with the symptoms observed during the life of Madame de Pauw, MM. Tardieu and Roussin concluded that there was a strong presumption that this lady had died from poisoning by *Digitaline.*

This case suggested to us the desirability of making a series of experiments, for the purpose of determining whether digitaline can, with certainty, be detected by its effects on the lower animals. It seemed to us that the question was one of great importance, from a medico-legal point of view; for this poison is one of those for which the chemical tests are most inconclusive.

We therefore made a number of experiments which extended from the autumn of 1864 to the spring of 1865. The results at which we arrived were laid before the Royal Society in May, 1865; and an abstract of them has been published in the Proceedings of that Society. In the present paper we propose to furnish the details of our investigations, and to give the grounds on which our conclusions are based. There are, also, one or two points upon which we were in doubt at the time of our communication to the Royal Society, but which our subsequent experiments have enabled us to clear up.

But, in the first place, we wish to insist particularly on the fact that physiological tests, to be reliable, must rest on a different basis from that which has been generally adopted. We have already alluded to the objection that the effects of poisons on the lower animals and on man are, in many cases, different. This difficulty has, indeed, been felt by M. Tardieu, for, in a recent article on the subject of poisoning,[1] we find him expressing himself as follows, with reference to the use of different kinds of animals, as the subjects of physiological experiment in medico-legal cases:

"Les grenouilles . . . surtout, sont précieuses comme moyens d'essai et de contrôle . . . . . . Mais les expérimentations sur les chiens sont indispensables pour conduire à des observations comparatives *qui peuvent seules permettre des rapprochements fondés avec les phénomènes de l'empoisonnement chez l'homme, et autoriser des conclusions positives.*"

But it might be answered that, even in dogs, the effects of a poison are not necessarily the same as in the human subject.

We therefore endeavoured, in our investigations, to avoid altogether this difficult question of "identity of action," upon which all physiological evidence in cases of poisoning had hitherto been based. "It appears to us," we said in our communication to the Royal Society, "that physiological tests may be made independent of any relation" between the action of poisons on man and on the lower vertebrata. "It is sufficient that the action of the substance supposed to contain the poison on the animal experimented on be identical with the known effects of that poison upon the same animal, and that these effects be capable of being produced by no other agent, or, at any rate, only by a limited number of such agents."

Since this was written, a case has occurred in Scotland, in which physiological evidence was largely employed by Prof. Penny and Dr. Adams, and in which the experiments were based on this very principle: we refer to the trial of Dr. Pritchard[2] for the murder by poison of his wife and of his

[1] "Etude médico-légale sur l'empoisonnement" ('Annales d'Hygiène,' 1864, t. xxii, p. 416).

[2] 'On the Detection of Aconite by its Physiological Action, being Notes of Experi-

mother-in-law, Mrs. Taylor. In the pocket of the latter was discovered a bottle, labelled "Battley's Sedative Solution." The liquid in this phial was, however, found to contain (we quote the words of Prof. Penny and Dr. Adams)—

"Antimony in a soluble form, in the proportion of one grain and a half of tartarized antimony to the fluid ounce. The discovery of an extraneous admixture directed special attention to this Battley's solution, which we shall hereafter call "Pritchard's Battley," and led to its chemical examination for the leading organic alkaloids, other than those existing in genuine Battley. After a fruitless search for the presence of strychnia, veratria, conia, and hyoscyamia, a small quantity of the liquid was evaporated in a watch glass, and the soft extract applied to the tip of the tongue, when there was felt quickly supervening upon the taste of *genuine* Battley's solution a peculiar benumbing and tingling sensation, increasing in intensity for a short time, and persisting for some hours. In numerous comparative trials with genuine Battley's solution, obtained from various sources, it became very clearly evident that there was a something characteristic in the taste of the Pritchard Battley, and suspicion turned to the probability of an admixture of aconite. Observations made with an evaporated soft extract of Fleming's tincture of aconite, with and without a mixture of genuine Battley, gave sensations so perfectly corresponding, that strong presumptive evidence was now obtained of the presence of aconite in the suspected fluid.

"By Stas's well-known process, and by other chemical means, more distinct indications of aconite were obtained; but the unreliable and altogether inclusive character of all known processes in chemistry for the detection and unequivocal identification of certain vegetable poisons, especially when contained in organic mixtures, and also the necessity of husbanding what remained of the suspected liquid, were sufficient reasons for determining that no time or material should be wasted in further chemical analyses, and a series of experiments upon animals was therefore determined on and planned.

"In view of the fact that different kinds of animals are differently affected by the same agent, we resolved to restrict our observations to one class, and several satisfactory considerations led us to select the rabbit. *It appeared to us of little moment that the phenomena manifested in our trials with the rabbit should differ from those exhibited by the human subject when under the influence of the same agent. For our purpose, it was sufficient if the toxic action was uniform and characteristic when employed on the same animal.*"

It is impossible not to be struck with the similarity between the language employed by Prof. Penny and Dr. Adams in the passage underlined by us, and that which we had ourselves used a short time before; and we think that every one who is unprejudiced must admit the importance of looking at the question of physiological evidence in cases of poisoning, from this point of view.

ments made in connection with the Trial of Dr. E. W. Pritchard,' by Prof. Penny, F.R.S.E., and Dr. Adams, Glasgow, 1865. For copies of this pamphlet, we are indebted to the kindness of the authors.

Moreover, if we adopt this principle, we are able to use, in the search for any particular poison, the animal on which that poison has the most marked action, irrespective of the position of the creature in the animal scale; whereas the passage which we have already quoted from M. Tardieu shows that, when the effects on the lower animals have to be compared with those observed in man, it is absolutely necessary to employ the higher vertebrata in these investigations.

Now, when *the operation of a poison on one kind of animal only* is the subject of inquiry, we think it undeniable that it is, *cæteris paribus*, far better to make use of batrachians, than of creatures occupying a higher place in the animal scale. For, if frogs are employed, it is possible to watch the action of the poison upon the different organs. The heart may be exposed, and, if the movements of this organ happen to be affected by the toxic agent, the different stages of its operation may be watched and noted at the time. Again, should the heart cease to beat, there is a considerable interval before the other vital functions cease, and we have opportunities of observing in what way the poison acts upon them. On the other hand, when dogs and rabbits are the subjects of experiment, it is only by inference that we can determine what organ is primarily affected; and when the functions of any one of the vital organs cease, those of all the others are almost instantly extinguished.

Further, frogs are not affected by fear, and are very little liable to be influenced by accidental circumstances, excepting, indeed, the temperature of the medium in which they are placed.

Another reason for preferring these animals is the rapidity with which the action of poisons manifests itself in them. Thus our experiments show that within an hour (or an hour and a half at the outside) all the characteristic effects of digitaline and the allied substances are produced. It is easy to carry on two or even three different observations at the same time.

Lastly, very small quantities of poison are sufficient to produce a characteristic action in frogs; and numerous experiments may therefore be made with an amount of poison which would prove altogether inert if given, in a single dose, to a rabbit or a dog.[1] Hence, in some instances, there is

[1] That this is the case is, indeed, shown by some of our own experiments.

practically no choice as to the kind of animal to be used for physiological testing.

We have no desire, however, to lay down a positive rule with reference to this point. It may be that future observations will show that certain poisons produce more marked effects on some other kind of animal than on the frog, and, in such case, it would be better to employ that other kind of animal. But we are strongly of opinion that, other things being equal, more constant and characteristic results are obtained by administering poisons to frogs than are observed when the higher animals are made the subject of such experiments. This, we think, is shown sufficiently by our own investigations.

The method of experimenting which we adopted was, as nearly as possible, the same in all our observations. After weighing the frog, and tying it down on a flat piece of cork, we exposed its heart. We then counted the pulsations of this organ, and noted any peculiarities in its action. If hæmorrhage followed the operation, the frog was set aside, and, unless the bleeding soon ceased, was not made use of for experiment.

It is necessary to bear in mind that the mere exposure of the heart has generally the effect of diminishing slowly the number of its beats. Thus, in one experiment, they fell, in half an hour, from 57 to 40 per minute; and, in another instance, from 48 to 41 within five minutes. Sometimes, however, the heart's action remains unaltered for a considerable time after exposure; and in one case, it rose in 34 minutes from 52 to 60—an effect which may, perhaps, have been due to an increase in the temperature of the room. When a frog struggles under these circumstances, the heart not unfrequently pauses in its action for an instant, remaining dilated; but this effect is always quite transitory.

Having then noted the frequency of the pulsations of the heart, we next proceeded to administer the poison with which we wished to experiment. This we usually injected beneath the skin of one or both thighs; but when the quantity was very large part of it was introduced into the flanks. The

Fluids vomited by human beings more than once yielded but a very small quantity of extract, which was all used in a single experiment upon a frog.

animal was then carefully watched; and the different effects were recorded as they were observed. When the full action of the poison on the heart had manifested itself, the frog was generally cut loose, in order to test the state of its muscular power.

Our experiments naturally arrange themselves into four groups.

Under the first head we have to describe the effects upon frogs of digitaline, and certain poisons allied to it in their action.

In the second place we propose to discuss the action of substances of a very different kind, namely, the alcoholic and acetic extracts of the contents of the human stomach, or of matters vomited by human beings. It has generally been assumed that such extracts cannot produce toxic effects, independently of the presence of any foreign substance; but we think that our observations prove conclusively that this is not the case, at least so far as frogs are concerned.

Thirdly, we have to consider the practical question whether the peculiar effects of digitaline upon frogs can be obtained from extracts of complex liquids to which it had been added before extraction, and from similar extracts derived from the stomach contents, and vomited matters of dogs poisoned by it.

Lastly, we shall see how far the results of our experiments confirm the evidence given at the trial of M. de la Pommerais, by which we were originally led to enter upon this inquiry.

I. *On the effects produced in frogs by digitaline, and by certain other poisons allied to it in their action.*

Before describing the physiological effects of digitaline, it is necessary to observe that there are two varieties of this substance, which differ in some of their properties. A memoir on this subject has recently been presented to the French Academy of Sciences by M. Lefort.[1] This observer speaks of the one form as "French," or "insoluble," of the other as "German," or "soluble" digitaline, and he thinks that the second of these varieties (which he states to be prepared, according to a secret process, by Merck of Darmstadt) is the better defined body of the two.

[1] 'Gaz. Médicale,' 1864, xix, p. 396.

In this country, however, the only kinds of digitaline which are to be procured are that made by Merck, and a substance of exactly similar properties, obtained from a firm at Stuttgart. Indeed, it does not appear that, even in France, the "French" or "insoluble" variety is an article of commerce. It is prepared by M. Homolle, the original discoverer of digitaline; and we are indebted to his kindness for a supply of this form of the substance. M. Homolle differs from M. Lefort, in regarding his own product as the purer of the two. In a note which we received from him, he informed us that he looks on all "soluble" digitaline as "*déja altérée.*"[1]

It does not appear that the two forms of this substance differ appreciably in their action on frogs. The nature of the symptoms caused by them is certainly the same, and in a few comparative experiments (Exp. 41, 42) made to decide this point, we have failed to detect any difference in the dose required to produce the characteristic effects of this poison.

It has been shown by Walz[2] that digitaline, as sold in commerce, is a mixture of several substances, chemically distinct, and soluble to a different degree in different menstrua. We therefore thought it possible that, after washing digitaline with a small quantity of ether (in which it is very sparingly soluble), the substance taken up by that reagent, and the undissolved residue, might be found to possess different degrees of activity. Our first two experiments (vide Exp. 20, 21), with the product obtained by the evaporation of the ether, seemed to

[1] The difference in the degree of solubility of the two substances in alcohol is very marked. There is also a decided difference in their odour, for the soluble form has a scent like that of hay or Paraguay tea, whereas no such scent is possessed by the product of M. Homolle. On the other hand, I cannot confirm the statement of M. Lefort, that hydrochloric acid gives a more marked green colour with the French than with the German digitaline. The tint seemed to me to be the same, whichever product was employed; but this reaction appears to me to be very unsatisfactory in its nature.

Quite recently M. Grandeau ('Gaz. Méd.,' 1864, xix, p. 383) has suggested another chemical test for digitaline. Concentrated sulphuric acid turns it of an olive-brown colour; and, when the mixture is exposed to the vapour of bromine, this tint changes to a mauve or violet. I have found this reaction to succeed tolerably well with both the French and the German varieties; but I am doubtful whether it is characteristic of digitaline.—T. S.

[2] Gmelin's 'Handbook of Chemistry' (Cavendish Society's Translation), vol. xvi, p. 331.

show that it impaired the voluntary muscular power more than the other substance, which is spoken of in the table as the "ethereal residue." But in two later experiments (vide Exp. 23, 24) in which we employed frogs of almost exactly the same weight, the effects of the two products were so precisely similar, that we did not hesitate to include these observations in the same table with those in which ordinary digitaline was given to frogs.

As we have already mentioned, digitaline is one of a small number of substances of which the effects on frogs appear to be almost identical. As the heart is the organ primarily affected by these agents, they may be termed "cardiac poisons;" a name which has already been applied to certain members of the group by MM. Dybkonski and Pelikan.[1] By M. Cl. Bernard,[2] some of these substances are included under the title of "muscular poisons;" but, as we shall see, this name is in the highest degree inappropriate to them.

The nature of the toxic effects produced by these various poisons was discovered at different times. It would appear that M. Vulpian,[3] in 1853, first observed the remarkable action of digitaline on the heart of frogs and some other batrachians. It had previously been asserted by Stannius that neither digitalis nor digitaline has much action upon frogs; but this result appears to have been due to his method of experimenting. "Frogs," he says, "which were made to remain for hours in an infusion of digitalis, after incisions had been made in their skin, presented only slight muscular weakness, which soon passed off. Solution of digitaline was also administered to them" (in what way he does not mention), "without effect."

The action of the other cardiac poisons on frogs appears to have been discovered in the following order. That of the upas antiar of Java (*Antiaris toxicaria*) was observed by Prof. A. Kölliker, in 1857;[4] and that of the *Tanghinia venenifera* of Madagascar, by MM. A. Kölliker[5] and Pelikan, in 1858. In the same year, corroval and vao, two arrow poisons

[1] 'Comp. Rend. de l'Acad. des Sciences,' 1861, Aug., p. 384. 'Mém. de la Soc. de Biologie,' 1861, p. 97. 'Ztschrft. f. d. Wissenschaft. Zoologie,' 1862, xi, p. 279.

[2] 'Med. Times and Gaz.,' 1860, vol. ii, p. 295.

[3] 'Comp. Rend. de la Soc. de Biologie,' sér. ii, tom. ii, 1855, p. 67.

[4] 'Verhandl. d. phys. med. Gesellschaft in Würzburg,' 1858, viii, p. 284.

[5] 'Verhandl. d. phys. med. Gesellschaft in Würzburg,' 1859, ix, p. 32.

brought from the Rio Darien in South America, were found by Dr. Hammond[1] and Dr. Weir Mitchell to cause similar effects. The action of the *Helleborus viridis* seems to have been observed independently by MM. Dybkonski and Pelikan,[2] and by W. Holm.[3] In the year 1864, Dr. Braidwood[4] described symptoms of the same nature as being produced by dajaksch, an arrow poison of Borneo.

To these substances we are able to add the *Scilla maritima*, which we have found to produce on frogs effects of exactly the same kind. Dr. Sharpey also has discovered the fact that the manganja, an arrow poison from the Zambesi expedition, obtained from Dr. Kirk, exerts a similar action. It is the pod of an apocynaceous plant, filled with oblong flattened seeds, each provided with a long feathered awn. The kernel and husks of the seed, as well as the pod, were found to be poisonous.

To the kindness of Dr. Sharpey we are indebted for specimens of the seeds of the manganja. With these we have made two experiments (vide Exp. 48) which fully confirm his observations. We have also ourselves investigated the action on frogs of antiar (vide Exp. 47, 49, 50), helleborus (vide Exp. 54—69) and digitaline. We have not been able to procure either the tanghinia or the arrow-poisons known as carroval, vao, and dajaksch.

We cannot but remark, not only on the wide geographical distribution of the "cardiac poisons," but also on the varied position which they occupy in the natural system of classification, representing, as they do, the Ranunculaceæ (helleborus), the Apocynaceæ (tanghinia and manganja), the Scrophulariaceæ (digitalis), the Artocarpaceæ (antiar), and the Liliaceæ (scilla).

When the proper quantity of one of these poisons is injected beneath the skin of a frog, a certain interval elapses before any effects are observed. During this time the cardiac beats sometimes become slightly accelerated, apparently in consequence of the pain experienced by the animal. More frequently, however, they beeome somewhat slower, as is the case in frogs

1 'American Journal of the Medical Sciences,' 1859, July, p. 13.

2 Loc. cit.

3 'Würzburg. Med. Ztschrft.,' 1861, B. ii, p. 448.

4 'Ed. Med. Journ.,' 1864, Aug.

whose hearts are simply exposed. The first change of importance in the heart's action usually begins quite suddenly. The rhythm is not generally much altered, but the beats of the ventricle become peristaltic, or different parts of its muscular wall contract and dilate at different times, and the blood, being driven from one part of its cavity to another, causes the relaxed portions of the ventricle to form pouches or, as we have often termed them, *protrusions*. Not unfrequently, again, the organ remains contracted longer than before, only dilating after each second or third beat of the auricles, and thus the number of the ventricular pulsations may be suddenly diminished to one half, or one third of what it had previously been. These effects appear to be produced indifferently by all the cardiac poisons, the action in each individual case depending rather upon the dose, and the rapidity with which the substance takes effect, than on the nature of the toxic agent itself. At any rate, every one of these poisons with which we have experimented has caused some of the effects we have been describing; and in the case of digitaline they are frequently all observed in a single experiment.

These remarkable changes in the heart's action are sooner or later followed by the total cessation of the dilatations of the ventricle, which then remains contracted, white, and perfectly empty. The auricles at the same time become distended with blood, but continue to beat for some minutes longer, after which they also stop, but in the dilated condition. It is stated by Drs. Hammond and Mitchell that, under the action of corroval, the capillary circulation was uniformly arrested one or two minutes before the ventricle ceased to beat. An experiment made by us shows that this is not the case with digitaline. The web of a frog to which this poison had been administered in the usual manner was watched under the microscope. When the heart's action became irregular, the flow of blood through the web began to flag; but, even after the complete stoppage of the ventricle, a sluggish circulation still continued for a time, the blood accumulating (as it appeared) in the larger veins.

At the moment when the ventricle ceases to beat, the muscular strength of the frog is quite unimpaired. The animal can leap as actively as before, and evidently retains also the power of sensation. After an interval, however, paralysis of the limbs is observed. It is not always easy to determine accurately the state of the voluntary muscular power. We have found that one of the best methods of testing the degree of paralysis is to place the frog gently on its back. If the animal can turn over, it will always do so within a short time; and its efforts, even if unsuccessful, show the extent to which its strength is diminished.

Now it appears probable that the gradual extinction of voluntary movements observed in these experiments is caused not by the action of the poison, but by the interruption of the circulation. For the same effect occurs, and within about the same time, when, without any poison being given, the circulation is stopped by ligature of the heart, or in any other way. Thus, the duration of voluntary power after simple ligature of the heart is stated by Kölliker to be from 30—60 minutes, and by Hammond and Mitchell to be from 25—35 minutes. From a few experiments of our own (vide Exp. 38, 39), we are led to conclude that, in this country, the longer period is the more correct.[1] Now, in our experiments with digitaline and squill, the muscular power was retained for from 30—67 minutes after the cessation of the beats of the ventricle.

The paralysis which follows the administration of antiar or hellebore is likewise ascribed, both by A. Kölliker and by W. Holm, to the interruption of the circulation. According to Kölliker, however, exhaustion of voluntary power occurs in the case of antiar in 30 or 40 minutes, and an interval of only 15 to 28 minutes after the cessation of the contractions of the ventricle is given by eight experiments of our own with antiar, the *H. viridis* and the *H. niger* (as sold in commerce). It is therefore possible that these poisons may, to some extent, directly impair the power of voluntary movement.

[1] The persistence of muscular power after ligature of the heart has also been shown by the very interesting experiments of Dr. A. J. Spence ('Edin. Med. Journ.,' 1866, July, p. 46). This observer found that a frog, the base of whose heart had been ligatured, and into whose stomach ¼ gr. of strychnia had been injected, could leap vigorously 35 minutes afterwards; whilst 50 minutes from the commencement of the experiment it could only move its limbs feebly. Another frog, similarly treated, was quite active half an hour after the application of the ligature. A third frog, the heart of which was ligatured, and into the abdominal cavity of which 2 grains of extract of nux vomica were then injected, was still able to leap after 44 minutes had elapsed.

We have already observed that, when the heart's action is made irregular, the capillary circulation becomes sluggish. It is, therefore, not surprising that (reckoning from the moment when the ventricle ceases to beat) we sometimes find paralysis occurring unusually early in cases in which the heart has been contracting irregularly and peristaltically for a considerable time before its final stoppage. This, however, is observed only when the quantity of poison is small.

It is generally stated that, soon after the extinction of voluntary power, it becomes impossible to excite reflex contractions in the limbs. Thus, Kölliker says that when antiar is the poison used, this occurs within from 50 to 60 minutes after the stoppage of the heart. We have found, however, that, under the influence of each of the cardiac poisons, the excitability of the spinal cord persists as long as that of the nerves. Whenever contractions of one lower limb have been obtained by galvanising the lumbar nerves of the same side, similar contractions have also followed stimulation of the nerves of the opposite side. The length of time during which we have been able to induce muscular contractions in this way has generally been about three or four hours after the cessation of the heart's pulsation. But in one experiment with digitaline, reflex and direct contractions were obtained even after five hours.

Soon after the extinction of the nervous irritability the muscles cease to be susceptible of the stimulus of galvanism, applied directly to themselves. After the lapse of four or five hours we seldom found any of the muscles contract under the influence of the galvanic current in frogs poisoned by digitaline. So, also, when squill, antiar, or manganja was the poison employed, the nervous and muscular irritability disappeared together. And this occurred still more rapidly than in the case of digitaline; for, in none of our experiments with the substances last mentioned were we able to obtain any muscular contractions after two or three hours—a result which accords perfectly with the statements of Kölliker with reference to antiar. The muscular irritability is, however, said to persist somewhat longer in frogs poisoned by the tanghinia.

Unlike the extinction of voluntary movements, these effects are undoubtedly caused directly by the poison. In frogs of which the heart was simply ligatured, contractions were distinctly produced by galvanising the muscular fibres themselves, after six hours in one experiment; after ten hours in another. In each of these instances, too, galvanic stimulation of the lumbar nerves induced contractions (both reflex and direct) after the lapse of four or five hours. That the cessation of the muscular and nervous irritability under the influence of the cardiac poisons is due immediately to the action of the poison may be proved also by applying a ligature to one limb, or tying its main vessels, so as to stop the circulation in it, before injecting the toxic agent. Dr. Sharpey informed us that he had in this way determined the action of the manganja, and we have done the same thing with digitaline. After five hours no contractions could be obtained in the right lower limb; while the muscles of the left limb, round which a tight ligature had been placed, retained their irritability at that time, and probably for a much longer period.

Besides the effect of the poison circulating in the blood upon the muscles of all parts of the body, we have always observed a marked local action upon the muscles of the region to which the toxic substance was immediately applied. When digitaline, squill, antiar, or manganja, was injected into one thigh, the irritability of the muscles of that thigh, and even of the calf, always disappeared more quickly than in the opposite limb.

There is a decided difference in the rapidity with which the heart's pulsations are stopped by the different "cardiac poisons." The following table shows the time required for the production of this effect in each case, according to the different authorities whom we have quoted.

Name of poison.	Time of stoppage of the ventricle after injection.	Observers.
Corroval	5½ minutes................	Hammond and Mitchell.
Antiar	5-10 „	Kölliker; Dybkonski and Pelikan.
Helleborus viridis	7-11 „	W. Holm.
Tanghinia venenifera ...	5-15 „	Kölliker and Pelikan.
Manganja	8-15 „	Dr. Sharpey.
Digitaline	10-20 „	Dybkonski and Pelikan.
Dajaksch	13-20 „	Braidwood.
Vao	15-20 „	Hammond and Mitchell.

It would therefore appear that antiar and *Helleborus viridis* are decidedly more active than digitaline. Our own observations fully confirm this; but it is remarkable that we have found the time required for the full action of the poison upon the heart much less in each case than that stated in this table. Thus, in our experiments, the *Helleborus viridis* and the antiar always stopped the contractions of the ventricle in 3 to $3\frac{1}{2}$ minutes; and manganja was not much less active, producing the same effect in $3\frac{1}{2}$ to 5 minutes. On the other hand, even under a dose of $\frac{1}{10}$ gr. of digitaline, the ventricle did not cease to beat in less than 7 to 14 minutes. The action of squill was, perhaps, somewhat slower still; when it was employed, the heart stopped in 14 to 20 minutes. We always injected the poison in solution; and this is probably one reason why effects were produced more rapidly in our experiments than in those of other observers; for the state in which any of these toxic agents is employed undoubtedly influences the rate at which its action becomes manifested. As we have mentioned, vao was found by Hammond and Mitchell to be much less active than corroval; but the alkaloids extracted from these two substances caused effects which were undistinguishable.

As the nature of the symptoms produced in frogs by the alcoholic extract of squill does not seem to have been previously described, it may be well to quote one of four experiments, in all of which effects identical with those of the other cardiac poisons were observed. On January 18th, 1865, we administered to a frog, by injection beneath the skin, some extract of squill, prepared by evaporation of the ordinary tincture (vide Exp. 52). The animal was carefully watched, but no effect was observed until sixteen minutes had elapsed. The frog then struggled, and the ventricle instantly stopped, remaining contracted and colourless. The auricles continued to beat regularly for at least two minutes, and became much gorged. Subsequently the base of the ventricle dilated somewhat, forming a well-marked crimson protrusion; but, within two minutes, it again became white and contracted, and remained in this state during 32 minutes. At that time, 50 minutes after the injection of the extract of squill, no impairment of the voluntary power could be detected. In other experiments (vide Exp. 46, 53) more marked irregularity of the ventricular beats was observed before the stoppage of the heart; but, in most instances, the interval between the appearance of the first effects and the cessation of the action of the ventricle was much shorter than might have been expected from the length of time which elapsed before any symptoms were produced.

In speaking of Helleborus, we have hitherto confined our remarks to the *H. viridis*. It is, indeed, doubtful whether other species of this genus exert a similar action. MM. Dybkonski and Pelikan state that the *H. niger*, though much less energetic than the *H. viridis*, does, after a time, produce paralysis of the heart. We found, however, that the effects of an extract of the fresh rhizome of the *H. niger*, prepared by ourselves during the spring of 1865, were different from those of the *H. viridis*. In two (vide Exp. 67, 69) of our three experiments with this extract the muscular power of the frog was destroyed long before the ventricle ceased to beat. The number of its pulsations was much diminished; the ventricle assumed a deep purple colour, and retained its dark hue even after it had stopped, though it was then contracted. Distinct peristalsis with protrusions was observed in two (Exp. 68, 69) of these experiments. On the other hand, the extract derived from the rhizome of *H. niger*, *as sold in commerce*, produced (vide Exp. 58—61) exactly the same effect as the extract of the *H. viridis*. We have employed two such extracts of the *H. niger*, the one made for us by Mr. Morson, the other prepared by evaporation of the tincture used at Guy's Hospital. Their action was the same in every respect. It is, indeed, possible that these results may have been caused by the accidental presence in the drug of portions of the rhizome of the *H. viridis*, an impurity which, according to Dr. Pereira,[1] is sometimes met with.

That toads are not affected by digitaline in the same way as frogs has been shown by Vulpian and Bernard, and an experiment of our own (vide Exp. 71) confirms the accuracy of their statements. Although some other poisons are likewise comparatively innocuous to toads, it appeared possible that these animals might be susceptible to the action of the hellebores. With the hope of establishing in this way a distinction between the

[1] 'Elements of Materia Medica,' 1857, vol. ii, pt. ii, p. 680.

two poisons, we administered some of the extract of the *H. niger* (as sold in commerce) to a toad (vide Exp. 70). No poisonous action manifested itself.

As it is possible, by means of ether, to extract digitaline from substances containing it, it appeared desirable to ascertain whether the active principle of the hellebores is also soluble in that liquid. We therefore washed some of the alcoholic extract of the *Helleborus niger*, prepared from the commercial root, with ether. The substance obtained in this way, after evaporation of the ether, produced the characteristic action on the frog's heart (vide Exp. 62, 63). The part of the extract insoluble in ether gave rise to very different symptoms (vide Exp. 64, 65). It caused irregularity of the *rhythm* of the heart, but no peristalsis or protrusions. The ventricle stopped *dilated* in 14—17 minutes, and the voluntary power was then already much impaired. It is therefore evident that, in addition to that which induces the remarkable "cardiac" effects, some other active principle exists in the extract of the *H. niger* of commerce. Whether this is due to the admixture of rhizomes from plants of different species, or to the presence of more than one principle in the rhizome of a single species, we must at present leave undecided. The fact that the action of the *H. niger* on frogs is very feeble, as compared with that of the *H. viridis*, is in complete accordance with the conclusions of Prof. Schroff,[1] drawn from experiments on rabbits.

According to our experiments, then, it is not possible, so far as the action of these substances on frogs is concerned, to distinguish between digitaline and the *Helleborus viridis*, or any other of the "cardiac poisons." There is, indeed, a difference in the length of time required for the development of the effects of these toxic agents; but, in practice, this could seldom be relied on. It might be supposed that augmenting the dose of digitaline administered to a frog would have the effect of increasing the rapidity of its action, and that, if very large quantities of it were given, the ventricle would cease to beat as soon as if antiar or the *Helleborus viridis* were the poison employed. Our experiments, however, seem to show that this is not the case. We have not ourselves administered more

[1] 'Prager Vierteljahrsschrift,' 1859, lxii, p. 49.

than 0·1 grain of digitaline to frogs. But M. Homolle[1] employed doses of 0·15—0·3 gr., and MM. Dybkonski[2] and Pelikan quantities still larger; and these observers found the effects to be less rapidly produced than was the case in our experiments. It would, indeed, appear that they always gave the poison in the solid form; and, if this were so, we should expect that its action would be somewhat delayed. However this may be, 7 or 8 minutes is the shortest time within which the ventricle has as yet finally stopped under the influence of digitaline, while, in our experiments the same effect was constantly produced by the *Helleborus viridis* in from 3 to 4 minutes. It is, however, obvious that this difference could rarely be of any value in a medico-legal inquiry. If the ventricle of a frog should stop in the contracted state within 3 or 4 minutes, we should probably be justified in saying that this effect was *not* due to digitaline; but no inference could be drawn from the fact that this result was produced only after 10, 15, or 20 minutes, for the action of any one of the cardiac poisons may, no doubt, be retarded when the toxic agent is mixed with other substances, instead of being simply dissolved or suspended in water.

We do not think, however, that in medico-legal practice there is likely to be any great difficulty in distinguishing between the different members of the group which we have denominated the cardiac poisons. Only three of these substances, in fact, are to be obtained in this country by ordinary persons; for carroval, vao, dajaksch, and manganja, are found only in the collections of toxicologists, and antiar and tanghinia are scarcely better known.

There remain, then, squill, helleborus, and digitaline. But squill, on account of the acrid property which belongs to it, is very unlikely to be used for criminal purposes, and it would appear[3] that the effects of large doses of this drug on man differ very decidedly from those of digitalis or digitaline. The fact that digitalis and squill are constantly employed together in therapeutics, under the idea that these substances differ in their action on the human subject, is of great interest, when taken

[1] 'Moniteur Scientifique-Quesneville,' June 15, 1864.

[2] Loc. cit.

[3] Pereira, 'Elements of Materia Medica,' vol. ii, pt. i, p. 205.

in connection with the identity of the effects produced by them in frogs.

It is probable that the purgative action of the helleborus would distinguish it from digitaline in a case of poisoning.

To recapitulate, we may sum up the effects of the cardiac poisons as consisting of three distinct elements—viz. (1) *a peculiar form of irregularity in the cardiac beats;* (2) *the stoppage of the ventricle in the white, contracted state;* and (3) *retention of the voluntary power at the moment when the pulsations of the heart first cease, and for at least* 15—20 *minutes afterwards.*

In no single experiment have we found this chain of effects to be produced by any substance excepting those which we have enumerated. We have ourselves administered to frogs the following agents, which were generally selected on account of there being more or less resemblance between some of their effects on man and those of some one of the cardiac poisons:—Aconitina (8 exps.), extract of *Veratrum viride* (3 exps.), veratria (3 exps.); nicotina (3 exps.), emetina (7 exps.), theine and infusion of green tea, resin of scammony, elaterium, watery extract of aloes (2 exps.); extracts of *Delphinium staphysagria* (3 exps.), *Ranunculus acris, Lobelia inflata,* colocynth, and barbadoes aloes; and, lastly, tartar emetic (2 exps.). All these substances (except, perhaps, the lobelia) were poisonous to frogs; but in no instance was their action similar to that of digitaline, helleborus, or squill.

We also made experiments on frogs with croton oil (2 exps.), and with the extracts of colchicum, and jalap, and the substance known as extract of gentianine; but these drugs appeared to be inert.

Of the substances above mentioned, two did, however, produce effects resembling, to some extent, those of the cardiac poisons. In doses of 0·1—0·2 grain (vide Exp. 208—212), *emetina* caused marked disturbance of the heart's action. Under the influence of this poison the ventricle of the frog beat peristaltically, and even displayed protrusions, which, however were not limited by borders so defined as are those caused by digitaline. Moreover, the muscular power, although it subsequently became affected, was not at first much impaired by this substance. But we think ourselves justified in distinguishing emetina from the cardiac poisons by the fact that in no one of our seven experiments did the ventricle stop in the contracted state.

Again, the extract of *Delphinium staphysagria* (vide Exp. 213, 214) caused, in one instance, most marked peristalsis with protrusions, and under its influence the ventricle became very slow in its action, and once even stopped in a condition intermediate between contraction and dilatation. But these effects were invariably accompanied by early and complete paralysis of voluntary power; and the ventricle had a dark colour, which is, perhaps, never seen as an effect of digitaline.

Besides administering digitaline to frogs, we thought it advisable to determine the action of the preparations of digitalis on these animals (vide Table VI). We found that a strong infusion of the dry leaves of this plant in water produced the characteristic effects. But, to our surprise, an extract derived from the tincture (obtained from the dispensary of the hospital) had a different action (vide Exp. 79, 80). Four other experiments with the same substance yielded similar results, but are not entered in the table. This extract caused great and often early impairment of voluntary power; and, under its influence, the condition of the heart varied, the ventricle failing to exhibit the tendency to stop contracted which is so marked an effect of digitaline itself. But by washing the extract with water, or, still better, with ether, and evaporating the solution so obtained, we got a substance which produced in frogs a characteristic action (vide Exp. 81, 82).

We were, at the time, much struck by this peculiarity in the effects of the alcoholic extract of digitalis, and we were inclined to associate it with the clinical fact, stated by many observers, that the action of the tincture of digitalis on man is not quite the same as that of the infusion. But our subsequent experiments have not confirmed our earlier ones. We have since ourselves carefully prepared an alcoholic extract of digitalis, which we have found to produce in frogs exactly the effects of a pure solution of digitaline (vide Exp. 75—78). We have little doubt that the difference observed in our previous experiments was due to want of care in the evaporation of the tincture, a process which was, in the first instance, carried out by others.

At the same time we may learn, from the mistake to which this gave rise, how liable organic extracts are to undergo changes by which their effects on batrachians are altered.

As the effects of the extract of staphysagria were not unlike those of the extract of digitalis with which we made our earlier experiments, it seemed possible that ether might remove from the former extract, as it did from the latter, a principle having the characteristic action of digitaline. We therefore treated the extract of staphysagria with ether, as we had treated the extract of digitalis. But the ethereal solution, when evaporated, was found to produce on frogs exactly the same effect as had been caused by the original extract (vide Exp. 215). There is, consequently, no reason to suppose that the action of staphysagria could be confounded with that of one of the "cardiac poisons."

With reference to the detection of digitaline by its physiological effects, it is evidently of the first importance that these effects should be *constantly* produced. On this point, however, we think that our experiments, forty-one in number (vide Tables I and II, Exp. 1—41), are conclusive. It is true that, when the dose of digitaline was small, its action was often limited to the production of peristalsis and irregularity of the ventricular beats; or, if the ventricle stopped for a time, it soon resumed its action. In the dose of ·005 grain even these effects were rarely observed, this quantity being (as it would appear) about the smallest which produces perceptible effects in the frog. But with doses of ·0078 grain and upwards the chain of symptoms above described was produced in all but two of our twenty-six observations. The exceptional cases were the following:—One of them (vide Exp. 2, 19) was among our very earliest experiments with digitaline. The frog was a large one; no effect was produced by ·005 grain. We therefore injected ·01 grain; and during 22 minutes, beyond which time we were unable to watch the animal, no effect was produced. The other instance (vide Exp. 23) is the one to which we have already referred, in which the muscular power was unusually affected by the product obtained by washing digitaline with ether. We do not, however, attach very great importance to these two experiments. When the quantity of digitaline was small the ventricle often subsequently resumed its action, and sometimes, even when large doses were employed, the organ, after a while, lost its white colour, turning of a livid or pinkish hue.

M. Hébert[1] is reported to have said, at the trial of M. de la Pommerais, that he had found digitaline innocuous to frogs; but as the dose which this observer used was less than ·005 grain, his results are in complete accordance with our own. As we have already stated, the effect of increasing the quantity of digitaline given to frogs is, within certain limits, to shorten the interval which elapses before the action of the poison manifests itself.

Although emetina and staphysagria cause somewhat similar effects, we are, nevertheless, disposed to attach considerable importance to the peculiar irregularity of the beats of the ventricle to which we have already several times alluded under the name of "*peristalsis with protrusions.*" It is, however, only when this appearance is well marked that we should rely upon it. The "peristalsis" produced by digitaline is the travelling of a rounded wave over the surface of the ventricle; each portion of the organ in turn relaxing from its state of rigid contraction. Another characteristic effect often observed is that the apex (or some other part of the ventricle) becomes white and contracted, and remains so even during the diastole. These "white patches" are often seen to enlarge, so that they finally take up nearly the whole surface of the ventricle, of which only one small portion then dilates, forming a crimson pouch or protrusion; or, perhaps, a zone of rigidly contracted tissue surrounds the ventricle, while the base and apex still dilate. Sometimes, again, the "protrusions" and "white patches" occupy different parts of the heart's surface at successive beats, and the appearance of the organ is then most extraordinary. Hammond[2] and Mitchell (describing these phenomena as caused by corroval) state that they are "among the most curious and striking physiological effects which have come under our consideration." As they rightly observe, similar appearances may be produced, by galvanizing

[1] 'Union Médicale,' xxii, p. 325.
[2] Op. cit., p. 45.

portions of the ventricle, in frogs to which no poison has been given. Wherever the current passes there is seen at the next beat of the ventricle a prominent red elevation. This effect, however, is only transitory. Very nearly the same condition is sometimes observed when the ventricle has been touched with the forceps during the operation of exposing the heart; and this was one reason why we preferred, in all our experiments, to have the ventricle fully under view before injecting the poison.

M. Vulpian,[1] who has very completely described these effects, and whose account of them coincides exactly with our own, states that, under certain circumstances, the action of digitaline is very different from that which has been generally observed by ourselves and others. He finds that when the frogs used are strong and healthy other signs of poisoning (such as general weakness and diminution of muscular irritability) precede the stoppage of the heart. The beats of the ventricle, he says, become slower and often irregular, no longer answering to those of the auricles. Sometimes it remains contracted while they make two or three feeble beats; sometimes it is relaxed, and becomes more and more gorged before it empties itself by one energetic contraction. At the end of an hour or an hour and a half, according to M. Vulpian, it is not uncommon to find the irritability of the muscles extinguished at all parts of the body, while the heart continues to beat slowly, remaining distended during the long pauses between its pulsations. The frogs with which he obtained effects of the kind which we regard as characteristic of digitaline had been kept fasting for more than six months, and their blood was thin and watery.

It is obvious that these statements are of the greatest importance. If correct, they would go very far to invalidate the applicability of the "frog-test" for digitaline. However, they are not corroborated by any of the facts which have come under our observation. Our own experiments range over a period of nine months, and we have more than once employed frogs which had been caught only the day before. Neither in April, 1865 (when the weather was extraordinarily warm), nor in June, 1865, did we find the effects caused by digitaline

[1] Op. cit.

differ from those observed during the preceding winter. It may, indeed, be urged that in April the animals were, perhaps, exhausted by spawning. But, if the statements of M. Vulpian are correct, it is remarkable that, so far as we can ascertain, nothing similar has been noticed in the case of the other cardiac poisons. Kölliker and Pelikan did, indeed, find that the irritability of the muscles and nerves, under the influence of tanghinia, lasted somewhat longer when the temperature was not above 43°—45° Fahr. than when the room was warmer. But Holm made his observations upon *Helleborus viridis* in July; and the effects caused by it at that season of the year were the same as in our experiments, which were made during the winter.

II. *On the action upon frogs of extracts derived from matters vomited by human beings or taken from the human stomach after death.*

As in cases of suspected poisoning the toxic agent has almost always to be sought in substances belonging to one of these two categories, it is of the greatest importance, in reference to the application of physiological tests, that the effects produced by such extracts, independently of the presence of any ordinary poison, should be thoroughly ascertained. Those who have advocated the use of this method of investigation have, however, very generally neglected this point; and, when it has been discussed, the idea of any fallacy arising from it has been set aside on theoretical grounds. Thus, in the report of their experiments in the case of M. de la Pommerais, MM. Tardieu and Roussin allude to this question in the following words:

"Auraient-ils" (the extracts A, B, and O, derived from the floor which received the vomitings, and from the stomach and intestines of the deceased) "pu donner la mort aux animaux par suite de la présence de matières animales putrides qu'ils auraient renfermées, et empruntées soit aux organes de la veuve de Pauw, soit aux vomissements répandus sur le parquet? Est-on fondé à comparer leur action éminemment toxique avec celle des chairs putrides et des viandes altérées? Serait-il possible, en un mot, d'établir une certaine analogie entre la piqûre d'une mouche ou d'un scalpel d'amphithéâtre, ou encore entre l'ingestion d'un boudin altéré et les phénomènes observés dans les expériences ci-dessus?

"La réponse est facile. Tout vient de la confusion que l'esprit pourrait faire entre *un virus, un ferment véritable* et *un poison*. Le propre du virus, du ferment, est d'agir à doses infiniment petites sur des masses infiniment grandes de matière; le poison, au contraire, ne produit d'action toxique sur l'économie qu'à dose fixe et régulière. Le premier est un être organisé, qui agit de proche en proche et propage lentement et successivement autour de lui une décomposition spéciale dont l'effet immédiat est de se multiplier lui-même à l'infini. Le second n'a rien d'organisé et ne se multiplie pas dans son action toxique sur l'organisme. Les agents antiseptiques, tels que l'alcool, détruisent les premiers et les rendent inactifs; les seconds ne sont en rien modifiés par leur contact avec cette substance et restent toxiques.

"Aucun corps organisé ni ferment putride ne se dissolvant dans l'alcool à 95 degrés, nous n'hésitons pas à dire que les extraits alcooliques A, B et O ne pouvaient renfermer aucun virus ou ferment putride capable de donner la mort par infection locale. L'expérience directe confirme entièrement ces observations: la viande la plus putride ne cède à l'eau ou à l'alcool aucun principe soluble capable de déterminer une intoxication quelconque, qu'on administre l'extrait de ces solutions intérieurement ou par voie endermique.

"Théoriquement comme expérimentalement, cette présence de ferments ou de matières toxiques solubles existant dans une solution alcoolique de viandes putrides, n'a pas le moindre fondement et ne représente qu'une fantaisie de l'imagination."[1]

MM. Tardieu and Roussin then proceed to state that this objection, even if it were well founded, could have no influence over their results; for not only did they employ alcohol of 95 degrees in the extraction, but the viscera were in a remarkably fresh condition, whilst the vomit scraped from the floor consisted of glairy mucus unmixed with any remains of flesh or other alterable substance.

Now, it so happens that in the case of the venom of toads a very similar argument had already been shown to be erroneous by M. Cl. Bernard.[2] We therefore thought it desirable to subject this question to the test of direct experiment.

For this purpose we made use of the contents of a human stomach taken from the post-mortem room of the hospital. These contents were divided into two equal parts, to one of which digitaline was added. Both portions, the one containing digitaline, the other free from this poison, were then dialysed, and the products of dialysis in each case were extracted with alcohol in precisely the same way.

The extracts thus obtained were administered to frogs,

[1] 'Ann. d'Hyg.,' 2ème sér., tom. xxii, p. 116.

[2] 'Med. Times and Gaz.,' 1860, vol. ii, p. 296.

the procedure adopted being similar to that used in experiments with the different cardiac poisons.

We then found that even the extract which contained no digitaline produced toxic effects of the most marked kind (vide Exp. 102—105).

We have repeated these observations several times, and generally with the same result. The vomited matters and post-mortem gastric contents which we used were of the most varied kind, and were taken without any selection. They consisted of a fluid, holding in suspension solid matters in greater or less quantity, including glairy mucus of various colours, bile, and undigested food.

Our object in extracting these mixed liquids being to obtain in a small bulk all the substances most soluble in alcohol, the process of extraction was conducted as follows:—The fluid was first dried on a water-bath. The dried mass was then pulverised, and was exhausted with methylated spirit; and, after filtration, the alcohol was driven off from the tincture by the application of a gentle heat. If, however, the liquid contained much fat and colouring matter, these substances were, as far as possible, removed by diluting the fluid with twice its bulk of water, and filtering it, before evaporation. The resulting extract was then digested with alcohol of about 85 per cent., and the whole was thrown upon a filter. The filter was not washed, but the filtrate was evaporated on the water-bath to the consistence of a thin syrup, or sometimes of a soft solid.

These extracts were introduced beneath the skin of the thighs, and also, in some cases, of the flanks of frogs, in exactly the same way as in our other experiments.

For experiments on the higher animals we employed a similar process, except that the re-extraction with alcohol was omitted. The extracts prepared in this way were also administered to frogs, and are spoken of in the table as "crude extracts."

As we have already said, these matters were very frequently poisonous to frogs; indeed, this was almost constantly the case. We have employed fourteen of these extracts, and have made with them twenty-nine experiments. In all but two instances (vide Exp. 95, 110) toxic effects were observed. We have included in the same table two experiments with a similar extract of a healthy dog's stomach, which extract also was poisonous (vide Exp. 97, 98).

Besides these, we have made three experiments (vide Exp. 116—118) with matters taken from the human stomach, but extracted, in the first instance, with acetic acid instead of alcohol. In each of these experiments effects of a marked kind were rapidly produced; and it is remarkable that one liquid, which yielded to acetic acid a poisonous extract (vide Exp. 117, 118) produced no toxic action (vide Exp. 95) when extracted with alcohol.

Under this head, too, we may include the experiments (vide Exp. 179, 180) in which we employed an extract of the liver of dogs poisoned with digitaline, and also those (vide Exp. 199, 200) in which we gave an extract of the empty stomach of one of these animals. For these substances, although poisonous, did not resemble digitaline in their action.

On the other hand, two experiments (vide Exp. 113, 114) with an extract of healthy human urine showed that this was inert.

The most frequent effect of the extracts of vomited matters or post-mortem gastric fluids was to impair simultaneously the action of the heart and the voluntary muscular power. The heart's beats generally became feeble. Not infrequently the ventricle ceased to beat, and was then distended with blood, and of a dark purple colour. Sometimes (vide Exp. 89, 90, 112, 116—118) these effects were very rapidly produced; but, as a rule, they showed themselves only after the lapse of a considerable time. In at least one instance (vide Exp. 92) the muscular power alone was affected by the poison, the cardiac action remaining unaltered. In other experiments, however, the reverse was the case (vide Exp. 94, 108), the heart's action being feeble, while the voluntary power was not at all or not so rapidly impaired.

In most cases (vide Exp. 88—90, 96, 98, 99, 100, 107, 109, 112) the effects observed were obviously very different from those which would have been produced by any one of the cardiac poisons. In a few experiments, however, the distinction was not so clear. The symptom which most frequently suggested a resemblance was the appearance of slight peristalsis or protrusions (vide Exp. 85, 97, 111). Even in these instances, however, the effects almost always differed from those caused by digitaline. The peristalsis was slight and transitory, the protrusions were ill-defined, and resembled those which may be induced by mechanical irritation of the heart rather than the sharply limited pouches which we regard as characteristic of the presence of one of the cardiac poisons. Moreover, the voluntary muscular power was not infrequently much impaired before even these effects made their appearance.

We did, however, meet with one extract (vide Exp. 83, 85, 91) by which slight peristalsis and occasional protrusions were produced, while the voluntary power remained unimpaired, either till the end of the experiment or, at any rate, till after these cardiac effects had shown themselves. Although the peristalsis and protrusions observed in these cases were not of a very decided character, it must be admitted that minute doses of digitaline might produce similar effects. But, even with this same extract, very different results were obtained in other experiments (vide Exp. 84, 87).

In only one (vide Exp. 109) out of the whole number of experiments with these extracts did we find the ventricle stop in the contracted state. In this case the heart had long been beating very slowly, and pausing in the dilated condition, and the voluntary power of the animal was extinct long before the final cessation of the ventricular pulsations. In two other instances (vide Exp. 101, 105) the ventricle became small and of a pinkish colour, but did not entirely cease to beat; and in another (vide Exp. 111), peristalsis showed itself at the end of an hour, and part of the ventricle at the same time became white and contracted. At that time the muscular power of the frog had long been slightly impaired.

In no single experiment, then, has any one of these extracts produced that conjunction of symptoms which is the almost invariable result of the action of a sufficient quantity of digitaline. And though we admit the absolute necessity of further experiments, our observations seem to point clearly to the existence of a broad line of distinction between the two classes of agents.

The table (Table VII) in which we have placed our experiments with these substances shows that, in several instances, a single extract produced, in different frogs, effects of different kinds. These variations we cannot altogether explain, but we are inclined to attribute them in part to alterations (ranging over 25°—30° Fahr.) in the temperature of the room, in part to differences in the weights of the frogs and in the doses of extract given to them, and to its having been administered sometimes in the liquid, sometimes in the solid state.

It becomes a question of importance to determine whether these extracts, which we have shown to be poisonous to frogs, are likewise poisonous to the higher animals.

Our own experiments on this point, however, have not yielded very satisfactory results. The extracts which we employed in these investigations were, as we have said, similar to those given to Batrachians, except that the second extraction with alcohol was omitted. A preliminary experiment was made with each extract, to prove that it was poisonous to frogs.

Having satisfied ourselves upon this point, we next proceeded to test the action of the extract on the higher Vertebrata. The substance, first mixed with a little water to render it more easy of absorption, was introduced into an incision in the skin of the back of the animal selected for experiment.

The results of these experiments were as follows:

I. A dog (vide Exp. 124), to which a large quantity of one of these extracts was administered, was not in any way affected by it.

II. Another extract was innocuous to a rabbit (vide Exp. 119).

III. A mouse, to which this same extract had been given (vide Exp. 120), was found dead in two hours. But this animal had not been watched; and as mice not infrequently die of fright, it would, perhaps, be unsafe to attach much importance to this result.

IV. Another mouse, which received the same extract (vide Exp. 121), was unaffected after two hours and three quarters, but was found dead at the end of twenty hours. However, a mouse, in whose back an incision of the same size was made for the sake of comparison, was nearly dead at the same time. (This experiment is not entered in the tables.)

V. To two other mice we gave another extract (vide Exp. 122, 123). One of these animals was in a very feeble state after the lapse of two hours, and in two and a half hours was dead; the other appeared at that time to be perfectly well, but was dead eighteen hours afterwards. It is at least doubtful whether the symptoms produced in the first of these two mice were not due to the viscid extract having become smeared over the surface of the skin. It seems quite possible that this might produce the same effects which are well known to be caused in some animals by covering the whole body with oil.

We feel very strongly that these experiments need repetition, and fully admit that it is, at present, an open question whether extracts of this kind are poisonous to other animals as well as to frogs. But the observations which we have described have greatly impressed upon us, from the very difficulties which we have met with, the advantage of using frogs rather than the higher animals as the subjects of such experiments. This is a point to which we have already once referred, and we shall again have occasion to speak of it when we come to discuss the nature of the physiological evidence brought forward at the trial of De la Pommerais.

The other experiments which belong to this branch of our subject were made for the purpose of determining whether the poisonous action of the animal extracts on frogs could be traced to any particular body contained in these extracts.

In the first place, when considerable quantities of these substances were used, part of the effects observed may, perhaps, have been caused by the alcohol present in the extracts. In large doses alcohol is well known to be very poisonous to frogs (vide Exp. 143—147). But we have estimated that the quantity generally present in the extracts which we employed did not amount to more than two or three grains. Now, on giving alcohol in these small doses to frogs (vide Exp. 139, 140) we found that it did not produce muscular paralysis till after forty or fifty minutes at least had elapsed. It is therefore clear that the action of the extracts on these animals could not have been due solely to the alcohol which these substances contained. Moreover, at least one of the extracts, in which the ordinary quantity of alcohol was present, was innocuous (vide

Exp. 95); whereas some of them which had been kept in the water bath for several hours produced toxic effects.

As the fluids from which these extracts were derived came from the human stomach, they must have been composed partly of food, partly of some of the secretions poured into the alimentary canal. All the so-called protein compounds, however, would be rendered insoluble by the joint action of heat and alcohol during the preparation of the extracts. Pepsine, again, is insoluble in alcohol, and direct experiment has shown (vide Exp. 128, 129), that, even had it been present, it could not have been the cause of the toxic action observed. Bile, though poisonous to frogs when given to them in large doses (vide Exp. 125, 126), is not contained in these extracts in sufficient quantity to account for their effects. We thought that the saccharine elements of food, when altered by the combined action of heat and dilute acids, might possibly be poisonous to frogs; but on administering caramel to one of these animals (vide Exp. 127), we found that it exerted no poisonous action within an hour and a half. As might be expected, some of these extracts contained free acid in small quantity. We were informed by Dr. Pavy that he had found dilute acids to have the power of stopping the frog's heart. We therefore made, with these substances, a few experiments by which this statement was fully confirmed. Indeed, we are not acquainted with any poison which stops the action of the frog's heart more rapidly than the dilute vegetable acids. When acetic, lactic, or butyric acid is injected in sufficient quantity beneath the skin of a frog, the heart often stops within two minutes, *remaining dilated.* The muscular power of the animal is also rapidly destroyed. In frogs poisoned by these acids we believe that we have observed the blood in the body generally to be acid, and not alkaline; and it is an interesting question whether the change in the reaction of the blood may not possibly be the immediate cause of the cessation of the heart's action. At any rate it is certain that the salts of the vegetable acids possess none of the poisonous properties which belong to the acids themselves (vide Exp. 135, 138). However this may be, it is clear that the acid contained in the extracts with which we are now concerned was not present in sufficiently large quantity to account for their toxic action; and this is further proved by the fact that, after neutralization with an alkali, these extracts still produced the same effects as before.

These negative results are all that we have been able to arrive at with reference to the cause of the poisonous action on frogs of extracts of vomited matters, or liquids taken from the human stomach after death.

III. *On the possibility of detecting Digitaline by its physiological effects when this poison is contained in the stomach or the vomited fluids of animals poisoned by it, or in other complex organic fluids.*

We have now to deal with the practical question, whether the effects which we have learned to regard as characteristic of digitaline (or, at any rate, of one of the cardiac poisons) can be obtained under the conditions which are met with in cases of poisoning. The first step which we took for the purpose of determining this point was to add some digitaline to liquids similar to those used for making the extracts described in the preceding section, and then to extract these liquids with alcohol. The quantity of digitaline which we mixed with each liquid was not large; it varied, in different cases, from 1·5 to ·75 grain. Three such extracts were made—one from urine, the others from fluids taken from the human stomach after death. With these extracts we made six experiments (Exp. 148—153). Another extract was made by acetic acid from gastric contents of the same kind, and with it four experiments (Exp. 154—157) were performed.

The results obtained with the alcoholic extracts were highly satisfactory. The effects were almost exactly those of a pure solution of digitaline, the only difference being that they were somewhat delayed, the final cessation of the ventricular beats occurring only after the lapse of from 32 to 60 minutes. The muscular power of the frogs usually remained unimpaired; in only one experiment (Exp. 153) was it much affected within 40 minutes.

On the other hand, the action of the acetic extract was less characteristic. In only two (Exp. 155, 156) of the four experiments made with this extract did we observe effects markedly similar to those of digitaline. As we mentioned in the preceding section, the extracts made with acetic acid from similar liquids containing no digitaline exerted a more poisonous action on frogs than the alcoholic extracts. It is therefore clear that the extracts used for these experiments should always be made with alcohol rather than with acetic acid.

When it is remembered that effects of an exactly opposite character were produced by extracts of vomited liquids, &c., to which no digitaline had been added, it may appear surprising that the extracts containing this poison should have given rise to symptoms so unmistakably like those caused by the cardiac poisons. As we have already stated, the effects were precisely those of a pure solution of digitaline, except that they were longer in making their appearance. There was an entire absence of symptoms referable to the constituents of the extract itself. The explanation of this apparent anomaly lies in the fact that in the experiments which we are now describing a much smaller quantity of extract was employed. The dose administered was, in fact, regulated according to the amount of digitaline calculated to be present in the extract, on the assumption that none of the poison had been lost in the process of extraction. Under the name of "*maximum quantity*" this calculated amount is entered in the table in which the details of these experiments are given. In the case of the alcoholic extracts it varied from ·125 to ·028 grain. The quantity of digitaline actually present was, no doubt, less than this.

In several instances we employed the process of dialysis before extracting the liquid in the ordinary way. It has been a matter of dispute whether digitaline (which is itself not a crystalline body) can be separated by this method from colloid substances, for though MM. Tardieu[1] and Roussin failed, both M. Grandeau[2] and M. Lefort[3] succeeded in doing this. Our own experiments with dialysis did not yield very satisfactory results. Thus, in one instance we added 1·5 grain of digitaline to a vomit, which was then dialysed. With the two extracts thus obtained we made eight experiments (Exp. 158—165); but neither the extract of the diffusate nor that of the interior liquid gave decided evidence

[1] 'Ann. d'Hyg.,' tome xxii, p. 105.
[2] 'Gaz. Méd.,' xix, p. 383.
[3] 'Jour. de Phar. et de Chim.,' Août, 1864.

of the presence of digitaline, the only effect observed being the production of peristalsis and protrusions in two (vide Exp. 159, 160) of the experiments made with the extract of the diffusate. Another extract made by this process also proved a failure (it has not been entered in the table). The remaining five extracts of fluids which had been dialysed produced effects more or less perfectly identical with those of digitaline. In one instance, however, the extract of the fluid which remained in the dialyser exerted a more characteristic action than that of the diffusate (Exp. 169, 171, compared with 166, 168).

It might be expected that, even if some of the digitaline were lost, the process of dialysis would yet have the advantage of separating that poison from the other poisonous substances contained in the liquid. The experiments to which we have just referred, however, show that this is not necessarily the case, although in other instances (vide Exp. 172, 173) it has appeared to be so.

When we compare the action of those extracts in the preparation of which dialysis had been used with that of the extracts made directly from the fluids themselves, we at once see that the symptoms produced by the second kind of extracts resemble much more closely those of pure solutions of digitaline. Hence, in the experiments which we are now about to describe we made no attempt to separate the poison by means of dialysis.

Having satisfied ourselves of the theoretical applicability of the physiological test for digitaline, and having also ascertained that the poison can be thus detected in animal liquids to which it had been intentionally added, we determined to carry our experiments a step further, by trying whether we could discover it in the viscera, stomach-contents, and vomited matters of poisoned dogs.

We therefore administered to each of three dogs five grains of digitaline. This dose was not unnecessarily large, for, according to the recent researches of M. Faure,[1] quantities of $1\frac{1}{2}$ to $2\frac{1}{2}$ grains of this substance do not constantly lead to

[1] 'Archives Générales,' 1864, Oct., p. 413.

severe symptoms in these animals. The poison was dissolved in a small quantity of alcohol, and water was added till the fluid measured five ounces, so that it then contained a grain of digitaline to the ounce. It was then injected into the stomach of the dog through a tube passed down the œsophagus.

The details of these experiments were purposely varied. We exposed the œsophagus of the first dog which we poisoned in this way, and put a ligature on the canal as soon as the liquid containing the digitaline had been introduced into the stomach. Consequently in this instance there was no vomiting. The dog was found to be dead after three and a half hours; and as it had received a meal not long before the injection of the poison, the stomach was found full of undigested food. A single extract was made, by means of alcohol, of the stomach and its contents. The process employed for this purpose resembled that described in the previous section; but after the first extraction precipitation with water was employed to remove fat and colouring matters, and a second extraction was found to be necessary.

The effects produced on frogs by this extract (Exp. 181—183) were not, in the first instance, entirely satisfactory. Their identity with the symptoms produced by digitaline seemed to be obscured by the toxic action of other constituents of the extract. With the object of removing some of these constituents, therefore, we added to the extract a few drops of alcohol, and afterwards a considerable quantity of water. We then filtered the liquid, so as to get rid of the fatty matters precipitated by the water, and again evaporated it until it was of a syrupy consistence. This extract was administered to frogs in the usual way (Exp. 184—191), and yielded unmistakable evidence of the presence of one of the cardiac poisons.

The liver of this dog was extracted in the same way as the stomach; but, as was to be expected, no effects resembling those of digitaline were obtained from this extract (Exp. 179, 180).

In the case of the second dog poisoned with digitaline, the solution was injected into the stomach in exactly the same way as before, and the œsophagus was tied. The animal died in three and three quarter hours. In this instance the matters removed from the stomach were roughly separated into a solid and a fluid portion before being extracted, so that two extracts were obtained. That of the solid portion of the stomach-contents yielded good results (Exp. 204, 205) when administered to frogs; but it was rather bulky, and the effects of the digitaline were, to some extent, obscured by the action of other substances contained in it. On the other hand, nothing could be more satisfactory than the action of the extract of the fluid portion of the gastric contents. Its whole weight was only 20 grains. In quantities of $3\frac{1}{3}$ grains it produced on frogs all the effects which we have described as most characteristic of the cardiac poisons (Exp. 201—203).

We think, therefore, that in investigations of this kind it is very advisable, when the matters vomited or taken from the stomach contain a large quantity of solid substances, to separate the fluid portion from the solid, before commencing the process of extraction.

Unlike the other two, the third dog to which we gave digitaline was allowed to vomit freely; consequently, after the animal's death (which took place within two and three quarter hours) the stomach was found empty. The extract of the stomach of this dog produced in frogs none of the effects of digitaline (Exp. 199, 200).

On the other hand, perfectly satisfactory results were obtained from the extracts of the vomited matters collected from the stone floor upon which they had been ejected. Before making an extract of the vomit, we separated its fluid portion from the solids contained in it. Hence we obtained two extracts. Of these, the one derived from the fluid part of the vomited matters produced on the frog's heart the effects characteristic of digitaline (Exp. 192—194); and, except when excessive doses were used, the muscular power of the animal was not impaired. The action of the digitaline contained in the solids was, in the first instance, obscured by that of the other substances present in the extract (Exp. 195). The method of precipitation by means of water, as already described, was therefore used to purify this extract, of which the effects then became unmistakably those of digitaline (Exp. 196—198).

IV.—*Remarks on the physiological evidence brought forward by MM. Tardieu and Roussin at the trial of M. de la Pommerais.*

We have already alluded to some of the features of this case; but we may repeat that, in experiments on animals, four different substances were employed.

1. An extract (named "*l'extrait O*") obtained by treating with alcohol the material scraped from the surface of, and the interstices between, the boards of the room occupied by Madame de Pauw, at the part of the floor which was soiled by the substances vomited by her.

2. An extract (named "*l'extrait P*") obtained in the same way from the boards underneath the bed, which were, of course, altogether free from vomited matters.

3. An extract ("*l'extrait A*") derived from the stomach and one half of the intestines of Madame de Pauw, by treating them with alcohol.

4. An extract ("*l'extrait B*") derived from the stomach and half the intestines, by the action of boiling-hot water.

Of these extracts, that known as the extract *O* was administered to a dog and to a rabbit.

In the first experiment five grammes of the extract were introduced into two small wounds in the thighs of a dog at five minutes past 1 p.m., the wounds being then closed by sutures. Before the operation the animal's heart was beating 110 in the minute. The animal afterwards moved about the room as usual. At the end of three quarters of an hour he lay down, and began licking the wounds. At 3.30 p.m. three attacks of vomiting came on; the dog threw up a glairy substance and a little bile, and then lay down again. He seemed anxious and much depressed. The heart was then beating only 94 times per minute; its pulsations were intermittent and very irregular; after being for some seconds hurried and tumultuous, they ceased abruptly, to be renewed a few instants later. The respiration was quicker than before the operation, and slightly intermittent. At 4.30 p.m. the heart's beats had fallen to 76; the animal again vomited. At 8 p.m. he was lying down, much depressed, and could with difficulty stand. The least movement seemed to give him pain, and excited vomiting or attempts at vomiting. The heart's beats were then 68, and presented the same kind of irregularity, with intermissions, as is above described; indeed, these were still more marked than before. At 8 the next morning the animal was almost cold; he seemed to have preserved his intelligence, for he still looked up and moved slightly when spoken to. The heart's beats had but little force, and their number was only 40 in the minute. Their irregularity and their "*intermittence précipitée*" were quite remarkable. When the hand was applied to the animal's body six or seven rapid beats were felt after an interval of some seconds, and then for a moment there was a complete pause in the heart's action. The respiration was hurried and intermittent.

These symptoms continued till 11 a.m., when the dog died almost without a struggle; it seemed to retain its intelligence to the last; it was certainly never in a state of coma.

The animal was opened some hours afterwards; the only point of importance was that the ventricles of the heart were remarkably contracted, while the auricles were dilated. All the cavities contained black blood, which was thick and partially coagulated.

In the second experiment two grammes of the extract *O* were given to a rabbit, by pouring it down the animal's throat. The symptoms observed in this instance were a considerable diminution in the frequency of the heart's beats, which became intermittent, with "*irrégularité et précipitation*." The respiration appeared painful, and slightly intermittent shortly before death. After two hours it was noted that the heart was making 41 pulsations per minute. After two and three quarter hours the rabbit died. It was opened the next day. The auricles were then dilated, the ventricles contracted.

MM. Tardieu and Roussin therefore concluded that the extract *O* included some poisonous substance derived from the vomited matters, and that this poisonous substance exerted a special action on the heart.

On the other hand, the extract *P* (derived from the part of the floor unstained by vomited matters) produced no effect when administered in the same way, in a dose of four grammes, to another rabbit.

As for the extracts *A* and *B* (of the stomach and intestines of Madame de Pauw), these substances were mixed together.

Five grammes of the mixture were then administered to a dog, by introducing the substance into a wound in the thigh. The heart's beats fell in an hour and a half from 102 to 86, and became irregular and intermittent. The animal also vomited twice; after five hours the heart's beats were 55, manifestly irregular and intermittent. Twelve hours later the dog was recovering; pulse 70. The symptoms gradually subsided, and the animal got well.

In another experiment with these mixed extracts, four grammes were given to a rabbit by mouth. The animal died in a few minutes, "*probablement,*" say MM. Tardieu and Roussin, "*par le fait d'une syncope, et avec une rapidité qui doit faire supposer qu'une complication accidentelle a pu hâter ici l'action du poison.*" We confess we should not be inclined to attribute to the poison any share in the death of this animal.

Before speaking of certain other observations, of which frogs were the subjects, we may make a few remarks on the four experiments in which poisonous effects were produced by the extracts *O, A,* and *B.*

The first question, of course, is whether the toxic action of these extracts was due to the presence of any ordinary poison, or was merely such as might be produced by any extract of matters vomited by human beings. We have already quoted the words in which MM. Tardieu and Roussin negative this last supposition. But our experiments on frogs with extracts of vomited matters show that it is not to be thus peremptorily set aside. At the same time, the observations which we have made with extracts of vomited matters on some of the higher animals (Exp. 119—124) do not in any way support the idea that such extracts can produce in dogs or rabbits the cardiac symptoms noticed in the experiments of MM. Tardieu and Roussin. We therefore think it most probable that these observers were right in believing that the extracts *O*, *A*, and *B*, contained some definite poisonous substance. This view is strengthened by the fact that the doses used were not very large, and the circumstance that these extracts were poisonous to rabbits when given by mouth perhaps points in the same direction.

The next question is, were MM. Tardieu and Roussin warranted in asserting that the effects observed were such as would be caused by digitaline, and in thinking it probable that this principle was contained in the extracts *O*, *A*, and *B*?

It appears to us to be, on the whole, most probable that they were right in both these points. The effects produced on dogs by the extracts *O*, *A*, and *B*, seem to have been very similar to those observed in our own experiments with digitaline, of which we have given the details in Table XVI. Thus, we found the number of the cardiac beats in dogs to be altered by this poison. The rapidity of the heart's action was constantly varying; in one case it was first 124, then 90, afterwards 126, 90, and, lastly, 120; all these changes being noticed within twenty-three minutes after the administration of the poison. It is, however, doubtful whether we may not lay too much stress on these results. M. Homolle,[1] speaking of one of his own experiments upon dogs, says that he did not note the pulsations of the heart, because previous observation had shown him that their frequency varied so extremely in the normal condition that no importance could be attached to any

[1] "Mémoire sur la Digitaline," Bourchardat's 'Archives,' No. 1, 1854, p. 119.

changes that might be observed. Again, M. Faure goes still further than this:—"*Jamais,*" he says, speaking of his experiments on dogs, "*nous n'avons pu saisir de changement notable dans l'état du cœur comme phénomène premier.*" And he quotes a remark made by Quevenne, that dogs are so impressionable that merely looking at them is sufficient to cause an instantaneous excitement of the heart's action.

M. Homolle and M. Faure, therefore, attach more importance to the vomiting than to the falling of the heart's beats, as a symptom of poisoning by digitaline in the dog.[1]

As we have already seen, vomiting was one of the principal symptoms in the experiments of MM. Tardieu and Roussin, of which we are now speaking.

In these observations death occurred at a period later than in our experiments. The dog poisoned with the extract *O* died only at the end of 22 hours, whereas, in one instance, we saw a dog die after $3\frac{3}{4}$ hours, and in two other cases the animal was found dead after $3\frac{1}{2}$ and $2\frac{3}{4}$ hours respectively. In three of the observations of M. Faure, again, 10 centigrammes of digitaline, injected beneath the skin, caused death in from 6 to 14 hours. In two other experiments, however, the same dose proved fatal at a later period, the exact time not being stated. In one of M. Homolle's[2] experiments a dog died in the course of the night, after 10 centigrammes of digitaline had been injected beneath the skin of the back at 10 a.m. It therefore does not appear that the length of the interval between the administration of the poison and the death of the dog in the experiment of MM. Tardieu and Roussin with the extract *O* is a valid reason for doubting that the effects were those of digitaline, especially as the dose of this principle was probably not very large.

When we come to estimate the quantity of digitaline which probably existed in the extract *O*, we do, however, meet with

[1] M. Faure, indeed, asserts that the vomiting caused by this agent differs from that produced by other emetic substances. The action of the stomach is not, he says, the principal thing, but is only the sequel of a series of convulsive movements, which commence in the limbs and abdomen, and are followed by violent expiratory efforts on the part of the ribs. However, in our only experiment upon a dog, in which free vomiting was permitted, we noted that the animal brought up the contents of its stomach easily and without great effort.

[2] 'Moniteur Scientifique-Quesneville,' 1864, Juin.

a serious difficulty. As we have seen, 5 grammes of this extract proved fatal to a dog. Now, according to M. Faure,[1] even doses of 10 or 15 centigrammes of digitaline do not necessarily produce very severe symptoms in dogs; and doses of 5 centigrammes produce no effect on these animals. The whole amount of the extract *O*, obtained from the scrapings of the floor, was 16·50 grammes. If, therefore, 5 grammes of this extract, by which the dog was killed, contained even 5 centigrammes of digitaline only, there must have been 16·5 centigrammes in the whole extract. But, in making this extract, only 12 of the 25 pieces of wood taken from the parquet of Mme. de Pauw were used, the remaining 13 pieces being set aside. It must therefore be assumed that, on the very lowest calculation, 33 centigrammes of digitaline (more than 5 grains) were actually present in the dried vomited matters on the floor of the room occupied by Mme. de Pauw. In addition to this, an amount of digitaline sufficient to poison (though not to kill) another dog is assumed by MM. Tardieu and Roussin to have been contained in a portion only of the extract (of which the quantity is not stated) obtained from the apparently empty stomach and intestines of Mme. de Pauw.

Considering how very bitter digitaline is, we confess that we hesitate to believe that so large a quantity of this substance as is implied by these considerations could have been given to Mme. de Pauw.

As for the experiments on rabbits, we have no data by which to subject them to a very close criticism. But both Homolle and Stannius[2] have shown that rabbits are less susceptible to the action of digitaline than dogs.

For the experiments on frogs the extract *O* was employed. Three observations were made at the same time: in one frog, the heart was simply exposed; in another, ·00924 grain of digitaline (dissolved in water) was injected beneath the skin of the abdomen; the third frog received about 50 centigrammes of the extract *O*. In the animal poisoned by digitaline the heart's action became "irregular" in 10 minutes,

[1] 'Archives Générales,' 1864, Oct., p. 413. See 'Year-Book' of the New Sydenham Society, 1865, p. 441.

[2] "Mémoire sur la Digitaline," &c., par E. Homolle et T. A. Quevenne, Bouchardat's 'Archives de Physiologie,' No. 1, 1854, pp. 179, 238.

fell to 15 in 20 minutes, and stopped in 28 minutes. In the frog to which the extract *O* was given the heart's beats became "irregular" in 10 minutes, had fallen to 20 in 20 minutes, and to 12 per minute in 28 minutes; the pulsations stopped altogether in 34 minutes. In each animal, when the heart had ceased to beat, the ventricle was contracted, the auricles gorged.

MM. Tardieu and Roussin state that they repeated these observations several times, with the same results. They always found that the extract *O* produced an irregularity of the heart's beats and a considerable diminution in their frequency. The irregularity, they say, was such, that towards the end of the experiment the heart never succeeded in emptying itself completely of blood. They then go on to say that they insist on these details, because of the striking analogy between the appearances observed by them and the "*déformations du cœur*" previously described by MM. Vulpian and Pelikan.

The account of these observations of the effects of the extract *O* on frogs is somewhat meager; but, so far as we can judge from the description given by MM. Tardieu and Roussin, these effects appear to have exactly coincided with those of the substances which we have spoken of under the name of cardiac poisons. We think that these experiments afford very strong evidence that digitaline was present in the matters vomited by Mme. de Pauw.

Since the above was written we have met with an interesting criticism by M. Alph. Devergie[1] on the evidence for the prosecution offered by MM. Tardieu and Roussin, on the trial of a French *officier de santé* for the supposed murder of his wife by poison. The exhumation of the body took place three months after interment, and the examination then made gave rise to much difference of opinion as to the state of the heart and brain, inasmuch as the woman, who, during her last illness had suffered from hemiplegia, was stated to have died from heart disease and cerebral hæmorrhage. With this question, however, we are not at present concerned.

By chemical processes, MM. Tardieu and Roussin extracted

[1] 'De l'Expérimentation Physiologique dans l'Expertise Médico-légale," par M. Alph. Devergie, 'Ann. d'Hyg.,' 1866, tome xxvi, pt. i, p. 168.

from the intestinal canal about 25 centigrammes (3·86 grains) of acetate of morphia, and from 35 to 40 centigrammes (5·4 to 6·17 grains) of an unknown substance, which had the following properties:—It was greenish-yellow, lustrous, scaly, deliquescent, having a very feeble scent and an intensely bitter taste; it was neutral to test-paper, burnt in the air after the manner of nitrogenous substances, but without any animal odour, and left an almost imponderable ash. It was soluble in water and in alcohol, less so in ether. Hydrochloric acid turned it of a slight green colour, and it was precipitated from its dilute solutions by tannic acid. These properties, which resemble those of digitaline, led MM. Tardieu and Roussin to surmise that digitaline was present in the substance under examination; and the fact that this poison, as well as morphia and veratria, had been purchased by the accused, favoured this view. They therefore resolved to try physiological tests in the hope of thus determining the question.

Some of the substance was given to frogs by injection beneath the skin of the belly; the animals died in 9 minutes. A dog, under the skin of whose thigh some of it was placed, became ill, but had quite recovered at the end of 24 hours. Some analogy was drawn between the symptoms exhibited by this dog and those of the deceased woman. Another dog, similarly experimented upon, died in 25 hours, after symptoms which were supposed to be analogous to those observed in this kind of animal when poisoned by digitaline. As we have already stated, however, dogs generally die much more quickly under poisonous doses of digitaline, and we have not met with an account of any one experiment in which it is positively stated that a fatal result occurred after so long an interval as 24 or 25 hours.

Three frogs were next made the subjects of experiment. The heart was exposed in each of these animals. The first was employed as a standard of comparison; to the second a solution of digitaline, of which the quantity is not stated, was administered by injection; and to the third a solution of the substance under examination. In the first frog the only change observed was that the cardiac beats were diminished by 5 or 6 in the minute; in the second they fell from 52 to 4 in 13 minutes; whilst the pulsations of the heart of the third fell from 58 to 36 in 8 minutes, and to 4 in 28 minutes. Another frog, to which a grain of the substance in solution was given, died in 12 minutes.

From these data MM. Tardieu and Roussin concluded—(1) that the woman had been poisoned; (2) that two poisonous substances had been extracted from the intestinal canal of the deceased, *each in sufficient quantity to cause death;* the first of these being acetate of morphia, the second a body which offered a great analogy to digitaline, and of which the deleterious effects were proved by experiments upon animals.

As morphia had been prescribed medicinally, in quantity corresponding with that found after death, and as the husband of the woman was kept some months in prison awaiting his trial, we suppose (though it is not stated positively in the report) that MM. Tardieu and Roussin were of opinion that she had been poisoned by digitaline. They say that the accused, though an "*officier de santé*," could have no legitimate use for the poisons he purchased, even for scientific purposes.

As we have not seen the original report of MM. Tardieu and Roussin, and have read only the remarks of M. Devergie upon it, we are not in a position to express a positive opinion as to the correctness of these conclusions. The experiments on frogs are dismissed in so few words that we are quite unable to say whether or not the effects observed in these animals resembled those of digitaline. But we find that the heart is not stated to have stopped under the influence of the substance experimented on, although this effect has always, in our observations, been produced by all except very small doses of digitaline. In any case in which we should obtain an extract in sufficient quantity to exert a poisonous action on dogs we certainly would not admit the presence of digitaline unless, when administered to frogs, the extract caused the heart to stop in the contracted state characteristic of this poison.

Conclusions.—The following are the chief points which result from our experiments:

We have found that digitaline, when injected beneath the skin of frogs in the proper quantity, produces three effects with almost absolute certainty. These effects are—(1) a peculiar form of irregularity in the heart's action; (2) stoppage of the ventricle in the white, contracted state; and (3) retention of the voluntary power for at least 15 to 20 minutes, and often for a much longer time, after the heart has ceased to beat. All these symptoms have unmistakably been produced by extracts of complex organic liquids to which digitaline had been added before extraction, as well as by extracts of fluids which had been vomited by dogs poisoned with this substance, or which were found in the stomachs of these animals after death.

Again, it does not appear that there is much difficulty in distinguishing the effects of digitaline from those of other agents which are poisonous to frogs. No substance which is to be obtained in this country, with the exception of squill, the *Helleborus viridis*, and perhaps the *H. niger*, is known to exert the same action. Of these, squill is most unlikely to be used as an instrument of murder or suicide; and the *Helleborus viridis* might, perhaps, be distinguished from digitaline by its purgative effects on man, and possibly, also, by the greater rapidity of its action upon frogs. Of course, however, we should not be justified in assuming the presence of digitaline, rather than the helleborus, in an extract of unknown composition which we found to produce the characteristic action on the frog's heart, merely because these effects occurred after a longer interval than when a pure extract of the helleborus has been administered.

With reference to the toxic action of extracts of vomited matters and gastric contents, it does not appear that their effects are ever very similar to those of digitaline. The fact that these extracts are poisonous to frogs, while they are not at present known to be injurious to the higher animals, might be urged as a reason for discarding frogs as the subject of physiological experiments in medico-legal inquiries. We have not, however, been led to this conclusion. We have already given the reasons which induced us originally to employ frogs for this purpose; and the experiments which we have made on dogs (see Table XVI) have convinced us that the symptoms of poisoning by digitaline in these animals are far less definite and far more difficult of accurate observation than those produced in frogs.

Unless some points of distinction should hereafter be discovered, the similarity between the effects of extracts of vomited fluids containing no toxic agent and those of *Veratrum viride*, staphysagria, and other vegetable poisons, will be a bar to the detection of these poisons by their physiological action.

At present, too, we regard it as very doubtful whether the action of aconitina on the frog is so characteristic as to be available for the discovery of this alkaloid.

On the other hand, we have not in any instance observed tetanic spasms to be produced by any substance which was not already known to excite such spasms in frogs. In our experiments, as in those of other investigators, veratria and theine have caused tetanic spasms when given to these animals. But no one of the other substances which we have already enumerated (vide p. 55), nor any one of the extracts of gastric contents or vomited matters, has been noticed to produce this effect.

It is true that, as the frogs were at first tied down, we had not very good opportunities of observing such spasms; but there is no reason to suppose that we ever overlooked their occurrence, and we therefore think that our experiments, so far as they go, give increased stability to the "frog-test" for strychnia.

Note.—In the tables which follow, the effects have, as far as possible, been arranged in vertical columns, according to the time at which they occurred. In some instances, however, great loss of space would have been involved by carrying out this plan strictly for effects which occurred at intermediate periods. In this case, therefore, the observations have been transferred to the next column, instead of being placed in the one to which they would most properly belong. When this has been done, the exact time has always been stated.

List of Abbreviations used in the Tables.

Aur. . . .	auricles, or auricular.
Cont. . . .	contracts, or contracted.
Contd. . . .	contracted.
Contn. . . .	contraction.
Contns. . . .	contractions.
Galvg. . . .	galvanising.
Min. . . .	minutes.
Ns. . . .	nerves.
P. . . .	pulsations of ventricle.
Musc. p. / Vol. p. / Vol. musc. p. .	voluntary motor power.
Ven. . . .	ventricle.

TABLE I.—*Experiments on Frogs*

No.	Date.	Weight of frog in grains.	Quantity of digitaline in grains.	Initial frequency of heart's beats.	In about 5 minutes.	In about 10 minutes.	In about 15 minutes.
1	Oct. 4, 1864	—	·005	44	P. 40	P. 40	P. 25. Peristalsis. Apex permanently pale
2	Oct. 4	(Large frog)	·005	21 (had fallen in 30 min. from 36)	P. 21	P. 22	—
3	Oct. 4	—	·005	34 (had fallen in 20 min. from 46)	P. 34	P. 34	Apex remaining pale, a little longer than the rest of ventricle
4	Oct. 5	270	·0051	41 (had fallen from 48 in 7 min.)	—	P. 37	—
5	Oct. 5	175	·0051	55 (had fallen from 60 in 5 min.)	—	P. 44. After 12 min. apex of ven. persistently pale; contracted portion gradually increased	Ventricle rigidly contracted. Aur. beating
6	Oct. 11	319	·006	70	P. 66	—	P. 68
7	April 4, 1865	236	·006 (German digitaline)	42	—	—	P. 28, regular
8	Oct. 24, 1864	540	·0078 (German)	52	—	After 12 min. protrusions near apex of heart. Heart stopped for an instant dilated; its action afterwards not quite uniform	P. 38

with Simple Solutions of Digitaline.

EFFECTS.				FURTHER EFFECTS.
In about 20 minutes.	In about 25 minutes.	In about 30 minutes.	In about 40 minutes.	
Ven. motion-less. contracted. Aur. beating	Ven. beating feebly at intervals; otherwise rigidly contracted	—	—	Animal began to revive after 45 min.; heart still mostly contracted. After 2 hours heart of a red colour, beating regularly 34 per min. Musc. p. of frog unimpaired.
—	—	No effect. ·01 gr. more injected; *vide* exp. 19, *infra*	—	—
P. 32. No obvious effect	—	·01 gr. more injected; *vide* exp. 18, *infra*	—	—
P. 32. One part of apex of ven. dilates before rest of organ	P. 30	No effect. ·0089 gr. more injected; *vide* exp. 15, *infra*	—	—
Ven. once dilated, instantly again contracted	—	—	P. 22, regular, feeble. Gradually increased in power	After 1½ hour ven. beating vigorously, crimson. Musc. p. tolerably good.
—	—	No effect. ·01 gr. more injected	—	15 min. after second injection ven. beat irregularly, afterwards protrusions, and ven. became contracted, scarcely dilating at all.
Protrusions after 18 min.	P. 28. Heart's action regular	—	Vent. (not observed since last report) white and contracted. Aur. gorged. Musc. p. a good deal impaired	Even after 2½ hours still feeble voluntary movements of limbs.
—	Heart's action peristaltic after a struggle. Apex contracted, not dilating thoroughly. Slight red protrusions	After 35 min. heart stopped, contracted, after a struggle; afterwards dilated once or twice imperfectly; protrusions	Animal leaps actively. Afterwards vent. white and contracted; soon began again to yield to contractions of aur.	After 1½ hour ven. slightly dilated, makes occasional beats. Frog could still crawl.

No.	Date.	Weight of frog in grains.	Quantity of digitaline in grains.	Initial frequency of heart's beats.	In about 5 minutes.	In about 10 minutes.	In about 15 minutes.
9	Oct. 24, 1864	467	·0078 (Merck's)	55	After 7 min. slight protrusions and irregularity of heart's action	Base of ven. alone dilates. A min. later whole ven. rigidly contracted.	Ven. still contracted. Musc. p. of limbs unimpaired
10	Oct. 15	—	·0089	41 (reduced from 45 in 9 min.)	After 6 min. ventricular contractions distinctly vermicular and irregular	P. 38. Heart's action steadier, soon afterwards again very irregular	After 17 min. ven. stopped, contracted
11	Oct. 5	409	·0089	40 (reduced from 57 in 30 minutes)	P. 36	Contractions vermicular 2 or 3 min., then accompanied by most marked and irregular protrusions. Most of ven. pale and contracted	Ven. motionless. Aur. beating. Musc. p. of limbs unimpaired
12	Oct. 26	298	·009 (Merck's)	56	P. 50	P. 48. Peristalsis. Apex white, contracted. After 11 min. ventricle stopped, white and contracted; afterwards again dilated	After marked protrusions of one part, the rest remaining white, ven. again became motionless after 14 minutes. Power of limbs undiminished
13	Oct. 24	680	·009 (Merck's)	56	After 7 min. ven. beating peristaltically, with protrusions	Protrusions marked. Every alternate dilatation of ven. imperfect	Apex of ventricle persistently pale
14	Oct. 24	518	·009 (German, but not Merck's)	49	During struggle middle of ven. white; apex and base each forming a protrusion at every diastole	After 8 min. ven. stopped, white and contracted; mus. p. of frog unimpaired	Heart again dilated, becoming crimson, but soon again stopped

EFFECTS.				FURTHER EFFECTS.
In about 20 minutes.	In about 25 minutes.	In about 30 minutes.	In about 40 minutes.	
—	—	Heart still motionless, now red. Frog more sluggish, but turns over when placed on his back	Frog now remains on his back when placed in that position. (Killed.)	—
The frog continues to struggle	After 23 min., ven. again dilated partially during a struggle	Ventricle again faintly dilating	Heart in same state	After 70 min. aur. contracting forcibly; ven. mostly contracted, dilating feebly at intervals. After 1½ hour, P. 18, pretty regular; two red spots remain even when rest of ven. pale. After 2½ hours, P. 20; heart otherwise in same state.
—	Ventricle persistently contracted	—	—	After 55 min. heart beating very feebly and slowly, but pretty regularly. Same report made 3½ hours later. Heart brick-dust red. Frog lively, could still leap.
Ven. still white, dilating with very feeble pink blush. Musc. p. unimpaired	—	—	Frog can crawl, but lies motionless on back for some time. Heart motionless, livid, pinkish-white	After an hour, heart dusky, livid, motionless, not contracted. Animal gives scarcely any sign of life.
White, contracted portion of ven. increases. Dilatations alternate. After a struggle, heart's action again regular	After struggle, almost whole ven. white and contracted; a small portion continuing to dilate for a short time. Musc. p. unimpaired	Heart beating pretty regularly. Frog rather torpid, can still crawl and leap	Heart scarcely beating, now red. Animal sluggish	—
Heart scarcely dilating at all	—	Animal rather torpid, still leaps well	—	At the end of 72 min. the heart, though pale, beating slightly. 30 min. later, animal could still recover himself when placed on his back.

No.	Date.	Weight of frog in grains.	Quantity of digitaline in grains.	Initial frequency of heart's beats.	In about 5 minutes.	In about 10 minutes.	In about 15 minutes.
15	Oct. 5	270	·0089 (second injection; *vide* exp. 4)	30 (originally 48)	Part of ven. near base persistently white; afterwards apex assumed same appearance; intermediate portion still dilating	Ven. persistently contracted; aur. distended, still pulsating	—
16	Oct. 10	356 (frog brought same day to hospital)	·01	48	P. 46. Heart soon afterwards irregular	Aur. distended, beating. Ven. contracted, pulsating slightly. A min. later ven. altogether motionless	—
17	Oct. 10	565	·01	52	P. 51. Heart soon afterwards peristaltic and irregular	—	P. 46. Heart stopped rather suddenly. Ven. afterwards made a few pulsations.
18	Oct. 4	—	·01 (second injection; *vide* exp. 3)	32 (originally 46)	After 6 min. contractions irregular and peristaltic, with protrusions	Aur. gorged, beating more frequently than ven.	Ven. motionless, contracted; aur. beating. Frog crawls actively
19	Oct. 4	—	·01 (second injection; *vide* exp. 2)	22 (originally 36)	—	—	P. 16. Animal let loose, torpid; crawls, but feebly
20	Feb. 23, 1865	866	·01 of the product obtained by washing dig. with small quantity of ether	48	P. 48	Peristalsis only while animal struggles	—
21	Feb. 23	476	·01 of ethereal residue of digitaline	55	—	P. 48, regular. After 13 min. rather marked protrusions	Slight tendency to protrusions at apex

Effects.				Further Effects.
In about 20 minutes.	In about 25 minutes.	In about 30 minutes.	In about 40 minutes.	
—	—	—	—	After 66 min. ven. still white, aur. black. Ven. has not dilated since last note. Frog feeble, can barely crawl.
—	—	Heart has remained white and motionless. Animal killed	—	—
—	Ven. now motionless, contracted, light purple; now making a few slow, incomplete pulsations	Killed	—	—
—	—	—	—	Next morning frog found dead. Ven. firmly contracted, empty; aur. contain blood.
Frog no longer watched	—	—	—	Next morning frog dead. Ven. partially, not completely, empty.
P. 38. Heart's action now peristaltic	—	Peristalsis now pretty marked	P. 36. Heart's action again regular. Musc. p. unimpaired	After 45 min. ven. beating very feebly, but regularly, pale liver-coloured. Musc. p. much more impaired. After an hour ven. nearly motionless, still livid. Aur. beating more fully. Frog nearly completely paralysed.
Ven. suddenly stopped, white and contracted. Aur. gorged, still beating	Musc. p. of animal unaffected. Heart afterwards pink	—	—	After 65 min. ven. still contracted, white, motionless. Vol. musc. p. unimpaired.

No.	Date.	Weight of frog in grains.	Quantity of digitaline in grains.	Initial frequency of heart's beats.	In about 5 minutes.	In about 10 minutes.	In about 15 minutes.
22	March 31, 1865	426	·0125 (Homolle's digitaline)	53	Effect began in 6 min. P. 46; only every alternate diastole perfect, the other barely perceptible. After 8 minutes protrusions; greater part of ven. white	After 9 min. ven. white, motionless; auricles gorged. Vol. p. perfect	—
23	Feb. 18, 1865	510	·0238, product of washing dig. with small quantity of ether	46	P. 36, regular. After 6 min. ven. motionless; 2 or 3 small protrusions appeared before it stopped	Heart still white, motionless. Vol. p. unimpaired	—
24	Feb. 18, 1865	511	·0238, ethereal residue	50	—	P. 45, regular	After 17 min. only a small point near base became red during diastole
25	Dec. 3, 1864	435	·0345, German digitaline	44	P. 49, regular	P. 46, regular	Heart suddenly irregular, every alternate dilatation imperfect. Distinct protrusions. Apex white. Heart soon again nearly natural
26	April 4, 1865	497	·0625, German digitaline	31	—	After 9 min. apex persistently white, base forming protrusions. After 11 min. left side of ven. alone dilates	At 13 min. heart beating regularly; P. 32. After 14 min. stopped, at first dilated; immediately afterwards white, contracted
27	Nov. 24, 1864	394	·066	44	P. 40. After 6 min. apex pale	Ventricle dark liver-coloured; base alone dilates. Afterwards beat more fully	After 13 min. ven. motionless, white, contracted. Musc. p. perfect

EFFECTS.				FURTHER EFFECTS.
In about 5[illegible] minutes.	In about 25 minutes.	In about 30 minutes.	In about 40 minutes.	
Heart in same state	—	—	Heart rather livid, pink. Vol. p. not perceptibly impaired	After 80 min. animal appeared dead.
Heart motion-less. Voluntary power nearly lost	—	—	—	—
After 18 min. ven. white, motionless, contracted. Aur. crimson, beating	Vol. p. unaffected	Ven. white, motionless. Musc. p. perfect	—	—
P. 14, quite regular. A min. later, ven. suddenly motionless, contracted. Aur. gorged, beating	Aur. beating. The ven. never again dilated. Musc. p. quite unimpaired	—	—	After 50 min. ven. in same state: when cut across found quite empty. Frog still crawls, and turns over when placed upon back.
Musc. p. unimpaired. Galvanization of aur. causes contractions; of ven. has no effect	—	—	—	After 50 min. vol. p. much impaired, but frog kicks violently. After 3 hours appeared dead. After 4 hours ven. still contracted; aur. full.
Ven. never again beat	—	—	—	After 50 min. frog crawls feebly, but lies helpless on back. Ventricle at one time pinker, now white. After 1½ hour frog showed few signs of life. Ven., when cut across, almost entirely empty.

No.	Date.	Weight of frog in grains.	Quantity of digitaline in grains.	Initial frequency of heart's beats.	In about 5 minutes.	In about 10 minutes.	In about 15 minutes.
28	Nov. 16, 1864	400	·1	38	P. 40. After 7 min. distinct protrusions after struggle	After 8 min. ven. suddenly white, dilating only in slight blush near base	After 11 min. ven. more pink, still contracted. Aur. beating
29	Dec. 3, 1864	320	·1	48	After 6 min. apex persistently white. After a few well-marked protrusions ven. in 7 min. stopped white, contracted	Afterwards ven. dilated feebly but regularly, 20 in the minute	After 12 min. slight wave of dilatation passes at intervals over surface of ven., which is pinkish
30	Dec. 24, 1864	380	Unknown quantity of ethereal washings of digitaline	50	After 6 min. heart peristaltic. P. 42	Marked protrusions. Ven. motionless, white, contracted, after 13 min. Aur. gorged, beating	Vol. p. unimpaired
31	Feb. 19, 1865	273	·006 ethereal residue of digitaline	40	—	—	—
32	Feb. 19, 1865	283	·006 ethereal washings of dig.	42	—	—	P. 39. Tendency to protrusions at apex, with every alternate dilatation
33	March 31, 1865	—	·0125, Homolle's digitaline	—	Protrusions after 5½ min. Capillary circulation then began to flag	After 8 min. ven. motionless. Capillary circulation nearly stopped; still sluggish flow into veins	—
	The capillary circulation of this frog was observed under the microscope.						
34	June 29, 1865	608	·02	71	P. 60, regular	—	After 12 min. apex pale; peristalsis. In 13 min. ven. stopped; pale, not white. Aur. beating. Musc. p. unimpaired
	This experiment made to show effects of digitaline in height of summer.						

EFFECTS.				
In about 20 minutes.	In about 25 minutes.	In about 30 minutes.	In about 40 minutes.	FURTHER EFFECTS.
—	Heart motionless. Vol. p. not impaired	—	—	After 1 hour ven. darker, still motionless. Frog torpid. After 1½ hour, frog leaps. Ven. again white. Pupils not dilated. Ven. cut across, quite empty.
Vol. p. not affected. Pupils not much dilated	—	—	—	After 50 min. heart still contracted, motionless, darker than previously. Animal almost dead.
—	—	—	Heart again beating 34 per min., irregular in rhythm	—
P. 35; alternate beats feeble	All beats of ven. again equal in force	After 38 min. p. 36; marked peristalsis; feebleness of every alternate beat; part of ven. persistently contracted	Ven. stopped, contracted, soon again beating. Musc. p. absolutely perfect	—
Every alternate dilatation feeble	—	P. 36. After 37 min. p. 27. Apex persistently pale. After 38 min. ven. motionless	Ven. beating regularly. Musc. p. quite unaffected	—
—	—	—	—	—
—	—	In 36 min. frog gasps; lies on its belly; still leaps, but sluggish	—	After an hour vol. p. distinctly impaired; after 83 min. only sign of life a feeble contraction of limbs when animal raised by them.

No.	Date.	Weight of frog in grains.	Quantity of digitaline in grains.	Initial frequency of heart's beats.	In about 5 minutes.	In about 10 minutes.	In about 15 minutes.
35	Oct. 5, 1864	432	·0089, German digitaline	—	Heart not exposed.	Experiment	
36	Oct. 5, 1864	280	·0178, German digitaline	—	ditto	ditto	ditto
37	Oct. 5, 1864	Very small frog	·027, German digitaline	—	ditto	ditto	ditto

TABLE II.—*Experiments showing the Duration of Voluntary Power and Heart, or Stoppage of its Action*

No.	Date.	Weight of frog in grains.	Nature and dose of agent employed.	Period of stoppage of heart's beats.	State of ½ an hour.
38	April 4, 1865	423	Heart simply ligatured	—	After 40 min. vol. p. much impaired; as much as in frog poisoned with dig. (exp. 26)
39	May 6, 1865	380	Heart simply ligatured	—	After 32 min. frog helpless on back, and could not turn over, though it tried

EFFECTS.				FURTHER EFFECTS.
In about 20 minutes.	In about 25 minutes.	In about 30 minutes.	In about 40 minutes.	
to test fatal dose of digitaline				After nearly 4 hours frog still tolerably vigorous, but heart found in state characteristic of digitaline; apex contracted; base dilating 14 per min.
ditto	ditto	ditto	ditto	After 52 min. frog feeble and ill. In 2 hours dead. Heart pinkish, moderately contracted, contracting still further when touched. Aur. moderately dilated.
ditto	ditto	ditto	Enfeebled and ill in 50 min.	In 2 hours feeble as before. In 2¾ hours scarcely able to move. Heart exposed, livid, neither dilated nor contracted; beating feebly at intervals, sometimes ceasing for a time.

of Nervous and Muscular Irritability in Frogs after Ligature of the by the different Cardiac Poisons.

muscular and nervous power, reckoned from time of cessation of action of heart.				FURTHER EFFECTS.
1 hour.	2 hours.	3 hours.	4 hours.	
Frog can still kick	Muscles becoming rigid	Animal quite dead, and decidedly rigid	—	—
—	In 1½ hour direct and reflex contns. obtained by galvg. lumbar ns. Muscles also contract perfectly on direct irritation	Condition as at last report	Condition as at last report	After 5 hours, on irritation of lumbar ns., no contractions. Muscles of thighs and shoulders contracted on direct application of galvanism. After 6 hours their state remained the same.

No.	Date.	Weight of frog in grains.	Nature and dose of agent employed.	Period of stoppage of heart's beats.	State of
					½ an hour.
40	May 2, 1865	393	Heart simply ligatured, and frog killed immediately afterwards. This and the next are companion experiments	—	Irritability of nerves and muscles perfect
41	May 2, 1865	549	·022 gr. German dig. (from Morson), injected into right thigh	Marked protrusions in 7 min.; ven. motionless and contrd. in 20 min. Frog soon afterwards killed by cutting off the head	Irritability of muscles and nerves to galvanism perfect
42	May 6, 1865	333	·008 gr. of Homolle's dig. into right thigh. This and the next are companion experiments	Peristalsis in 6 min. In 9 min. ven. stopped, contracted and white	After 44 min. frog lay flat on belly, and did not attempt to leap or crawl when touched
43	May 6, 1865	300	·008 gr. Merck's dig. into right thigh	Peristalsis in 8 min.; afterwards protrusions. Ven. motionless and contracted in 11 minutes	After 44 min. frog sits with head erect, but does not attempt to leap or crawl when touched
44	May 6, 1865	603	After firm ligature of left thigh, ·008 gr. dig. injected into right thigh. (Both limbs contracted equally on direct galvanization)	After 13 min. protrusions. In 16 min. ven. motionless, contracted, and pink. Frog crawls, dragging ligatured limb after it	After 32 min. lies helpless on back; but vol. p. not quite extinct

muscular and nervous power, reckoned from time of cessation of action of heart.				FURTHER EFFECTS.
1 hour.	2 hours.	3 hours.	4 hours.	
—	Irritability of nerves and muscles perfect after $1\frac{1}{2}$ hour	—	After 5 hours galvg. lumb. nerves caused contns. of lower limb of that side; also reflex contns. of opposite limb. Muscles perfectly irritable	At the end of 10 hours no contractions on galvg. the nerves. Distinct contns. on galvg. the muscles of either thigh.
—	After $1\frac{1}{2}$ hour no contns. of muscles of right thigh on galvg. them; otherwise state of muscles and nerves perfect	—	After 5 hours contractions of left leg, but not of right leg, by galvg. either right or left lumb. ns. Muscles of left thigh but slightly, those of right thigh not at all, susceptible of direct irritation. Muscles of both legs contract when galvanism is applied to them.	At the end of 10 hours no contns. by galvg. either the muscles or nerves.
Direct and reflex contns. on galvg. lumb. ns. Muscles of each thigh contract perfectly on direct stimulation	Direct and reflex contns. of both legs on galvg. lumb. ns. On directly galvg. muscles of right thigh no contns.; those of left thigh, feeble contractions; those of either calf, contractions	No contractions, direct or reflex, by galvg. lumb. nerves. Muscles of both thighs and of right calf insusceptible of direct stimulation; of left calf contract when galvanism applied to them	Recently exposed muscles of back alone contract on direct application of galvanism	—
Lies helpless on back; but vol. p. not completely extinct	Frog dead. Direct and reflex contractions of both thighs on galvg. lumb. ns. Muscles of left thigh and of right calf contract on direct galvanization. Of right thigh not susceptible	Muscles of left calf susceptible; of right calf and of both thighs no longer susceptible. Direct and reflex contractions of left limb by irritation of lumbar nerves	Shoulder muscles distinctly contract. No other evidence of sensibility to galvanic current	30 minutes later same observation repeated.
Dead in $1\frac{1}{4}$ hour. Galvanization of lumb. ns. causes direct and reflex contns. in right thigh, none in ligatured limb. Muscles of each limb contract on direct stimulation	Direct and reflex contns. of right limb on galvanizing lumb. ns. Muscles of left thigh and of calves susceptible, but of right thigh insusceptible, to galvanic current	Galvanizing lumb. ns. causes no contractions even in right limb; muscles of left thigh contract when galvanized; of left leg now contract more powerfully than those of right when galvanized	Muscles of left thigh and left calf contract when galvanized. Right leg has lost power of contracting	—

No.	Date.	Weight of frog in grains.	Nature and dose of agent employed.	Period of stoppage of heart's beats.	State of
					½ an hour.
45	May 6, 1865	530	·018 gr. Merck's dig. into right thigh	Marked peristalsis and protrusions in 7 min. Ven. stopped, contracted, in 20 min.	—
46	May 6, 1865	326	3 gr. of Morson's alcoholic ext. of squill, dissolved in water	Ven. stopped, contracted, in 20 min.; aur. still beat. Protrusions before stopping	Crawls, but feebly. Turns over when placed on back. In 40 min. paralysed and flaccid, but moves slightly on irritation.
47	May 6, 1865	380. Frog with right leg wanting	Solution of upas antiar into flank	Ven., which had not been watched, motionless and contrd. after 7 min. Auricles gorged and motionless	After 15 min. vol. p. impaired. Frog lay with chin on table, and could not turn over
48	May 6, 1865	350	Alcoholic ext. of 2 or 3 seeds of Manganja (Zambesi poison)	Heart's beats rose slightly, then fell from 50 to 44 in 4 minutes. Dilatations became feeble, and in 5 min. ven. motionless and contrd., aur. beating. Vol. p of limbs unaffected	Lies on back, and makes ineffectual attempts to turn over

muscular and nervous power, reckoned from time of cessation of action of heart.				FURTHER EFFECTS.
1 hour.	2 hours.	3 hours.	4 hours.	
After 50 min. vol. p. much diminished. After 67 min. lies helpless on back. After 80 min. direct and reflex contractions in both limbs on galvg. lumb. ns. Muscles of right thigh insensible to galvanism, locally applied	Galvanizing lumb. ns. causes direct and reflex contractions of left foot and slight contractions of left thigh. Muscles of left thigh do not contract on direct stimulation, even by a powerful current. Contns. of right calf are markedly less, on direct stimulation, than those of left calf	Direct and reflex contractions in both limbs on galvanizing lumbar nerves. Muscles of left calf alone react when muscles are galvanized	No contractions on galvg. lumb. ns. or muscles of hinder limbs	—
Sensation and vol. p. altogether lost. Left limb readily contracts on irritation of left or right lumb. ns. Right limb contracts less, toes alone moving slightly when either right or left lumb. nerves galvanized. Muscles of right thigh do not cont. on direct galvanization; of right leg but feebly; of left limb perfectly	No contractions of right limb on galvg. either nerves or muscles. Direct and reflex contns. of left limb on galvg. lumb. ns. No contractions on galvg. muscles of left thigh, and but feeble on galvg. muscles of left calf	All the muscles and the lumb. ns. insusceptible to galvanism	—	—
Irritation of either right or left lumb. nerves causes well-marked contraction of left foot. Galvanization of either muscles or nerves of thigh causes no contractions. Galvanization of calf-muscles causes contns.	Same results in every respect produced after 1½ hour	After 2 hours 40 min. no contns. on galvg. either the muscles or nerves	—	—
Galvanization of either right or left lumb. ns. causes contns. of left leg and thigh; those of the left thigh feeble. On direct galvanizn. of muscles of left thigh no effect; of calf, contns. No contractions of right thigh on galvanizn. of nerves or muscles	After 2½ hours no contns. obtained on galvg. nerves or muscles of either limb	—	—	In another exp. ven. stopped, white and contracted, in 3½ min., after protrusions, &c. Galvanism gave similar results.

TABLE III.—*Experiments with Upas Antiar and wi*

No.	Date.	Weight of frog in grains.	Nature of substance used, and weight in grains.	Initial rate of pulse.	Effects in about 5 minutes.	In about 10 minutes.
49	April 25, 1865	375	·1 ext. of antiar berry	48	Marked protrusions and white patches in 2 min. Ven. motionless, white, and contracted in 3 min.; aur. beating; vol. p. unimpaired	—
50	April 25, 1865	410	Ext. of husk of antiar	36	In 6 min., P. 40	In 9 min. P. 21. Ve dilates only at each a ternate systole of aur.
51	Jan. 17, 1865	291	Ext. of squill	24	P. 32, regular	—
52	Jan. 18, 1865	1065	Smaller quan. of ext. of squill	28	—	—
53	April 6, 1865	—	3 of Morson's ext. of squill	54	—	Diastole feeble, 2 (3 doubtful protrusion In 12 min. marked pr trusions. Parts of ve always pale

Scilla Maritima. (*See also Experiments* 46 *and* 47.)

In about 15 minutes.	In about 20 minutes.	In about 30 minutes.	FURTHER EFFECTS.
Aur. motionless; ven. contracted, livid	—	Frog struggled, and legs moved spasmodically, then lay helpless on back	After 40 min. galvanizing either nerves or muscles causes less marked contractions on side in which poison injected.
—	In 25 min. apex rather pale during diastole	After 40 min. heart beating very feebly. Diastole still affects base more than apex	After 1½ hour pulse very slow and feeble. After 4 hours ven. livid, less dilated, contracting at intervals of some seconds. Frog active as ever.
Frog has not been watched, but ven. now contracted, white, and motionless	Vol. p. not impaired	Crawls, but does not leap. Heart in same state. Killed	—
In 16 min., during struggle, ven. stopped suddenly, contracted; base afterwards dilated, forming a well-marked protrusion. Aur. much gorged, beating regularly	Ven. white and contracted. Aur. beating. Vol. p. not impaired	Ven. livid, pale, motionless	After 50 min. heart still motionless and contracted. Vol. p. unimpaired.
In 14 min. ven. white and contracted; aur. moderately gorged. Vol. p. perfect	—	—	After an hour vol. p. diminished, though the frog could crawl and kick. Ven. contracted, grayish-purple. After 1½ hour frog dead.

TABLE IV.—*Experiments with*

No.	Date.	Weight of frog in grains.	Nature and quantity in grains of poison.	Initial rate of pulse.	Effects in about 5 minutes.
					1. *Experiments with an Extract of the*
54	April 1, 1865	420	3·5, in a little water	48	In 3 min. ven. motionless, contracted, and pinkish; aur. beating. Vol. p. unaffected
55	April 1, 1865	318	·5	60	Slight crimson prominences, such as are observed on mechanical irritation of heart, on ven. immediately after injection. True protrusions, first at end of 1½ min. Only every alternate dilatation of ven. complete, organ tending to stop contracted. After 2 min. diastole only a slight blush at one side of ven. In 3 min. ven. stopped, contracted; aur. beating
56	April 3, 1865	336	·33	48	Effects after 3 min. the ven. tending to remain contracted, and immediately afterwards protrusions. In 3½ min. ven. motionless, white, and contracted; aur. beating. Vol. p. unimpaired
57	April 4, 1865	330	·25	—	In 3 min. most remarkable protrusions and peristalsis. In 4 min. ven. white and contracted; aur. gorged
					2. *Experiments with Extract of the Helleborus*
58	April 3, 1865	266	8—10 of Morson's ext.	49	After 7 min. most marked peristalsis and protrusions. P. 30
59	Dec. 22, 1864	520	Large quantity of ext. prepared from Guy's Hospital tincture	44	P. 36

different Preparations of Helleborus.

In about 10 minutes.	In about 15 minutes.	In about 20 minutes.	FURTHER EFFECTS.

fresh Rhizome of Helleborus Viridis.

In about 10 minutes.	In about 15 minutes.	In about 20 minutes.	FURTHER EFFECTS.
—	Heart motionless, contracted. Vol. p. nearly extinct	—	After 30 min. ven. motionless, contracted, livid
Vol. p. unaffected	Heart motionless and contracted, now red. Frog quiet, not able to leap, but when placed on back instantly turns over	—	—
After 8 min. aur. motionless. Ven. contracted, but not so white as before	—	After 27 min. ven. still contracted, but of a red colour. Musc. p. unaffected	After 31 min. frog lying on its side paralysed. Pupils dilated.
—	—	Vol. p. becoming impaired. Frog lies helpless on back	Vol. p. much impaired.

Niger (prepared from the Root as sold in Commerce).

In about 10 minutes.	In about 15 minutes.	In about 20 minutes.	FURTHER EFFECTS.
P. 20. Effects most marked. Ven. pauses dilated. In 11 min. ven. stopped, contracted, and had a peculiar bright colour. This peculiar colour of the heart was observed before the injection of the poison	Ven. motionless, pale gray, no longer red. Aur. gorged, beating. Frog crawls and leaps feebly	Vol. p. much impaired	—
P. 17, regular. In 13 min. contractions partial. Apex crimson, forming a well-marked protrusion. Distinct white patches	Ven. motionless, relaxed, red. Aur. beating. In 18 min. ven. as before; aur. gorged; pupils more dilated	After 25 min. heart as before. Frog turns over when placed on its back	In 37 min. the frog, when placed on its back, lay almost completely paralysed. Pupils rather dilated. Ven. still white and contracted. A little blood escaped from ven. on incision.

No.	Date.	Weight of frog in grains.	Nature and quantity in grains of poison.	Initial rate of pulse.	Effects in about 5 minutes.
60	Dec. 22, 1864	630	Same ext.	53	Heart's action rapid. After 5 min. most marked peristalsis and protrusion. After 6 min. P. 32. After 7 min. P. 26, pretty regular.
61	Dec. 23, 1864	344	Smaller quantity of same ext. to one thigh only	45	After 3 min. P. much slower. In 4 min. ven. stopped, dilated; 1 min. later it resumed its action, beating 14 per min.

3. *Experiments with Preparations derived from Extract of Helle-*

No.	Date.	Weight of frog in grains.	Nature and quantity in grains of poison.	Initial rate of pulse.	Effects in about 5 minutes.
62	Dec. 24, 1864	354	6 of that part of ext. soluble in ether (freed from resin)	39	Peristalsis and protrusions in 4 min. In 6 min. P. 20, very feeble; ven. mostly contracted and gray
63	Dec. 24, 1864	510	2·75, as in preceding experiment	38	Most marked protrusions and peristalsis in 7 min. In 8 min. heart scarcely beating, forming only a slight crimson protrusion at base and apex
64	Dec. 24, 1864	326	6·33 of part of ext. insoluble in ether, redissolved in alcohol	38	In 6 min. P. 30. Rhythm rather irregular. Tendency of heart to pause, dilated
65	Dec. 24, 1864	368	6·33, as in above exp.	48	After 6 min. rhythm of ven. irregular. No peristalsis or protrusions. After 8 min. P. 20
66	Dec. 24, 1864	406	Portion undissolved after digestion with ether and with alcohol	54	—

In about 10 minutes.	In about 15 minutes.	In about 20 minutes.	FURTHER EFFECTS.
After 8 min. ven. beating very feebly, with a tendency to remain contracted. In 10 min. ven. motionless, nearly white, and contracted. Aur. beating. Respiration continues. Vol. p. perfect	In 13 min. whole heart motionless. Aur. not gorged	Heart in same state. Frog lies helpless on back	After 33 min. musc. p. nearly extinct. After 80 min. ven. still white, contracted, motionless; when it was cut across, a drop of blood appeared on its cut surface.
In 7 min. P. 6. In 11 min. ven. motionless, contracted, pink rather than white. Aur. gorged, beating at intervals	Vol. p. extinct. Frog lies motionless on back. Heart as before. Pupils unaltered	Heart in same state. Respiration continues	Heart in same state; when cut across, ven. is found to be nearly empty. Frog moves limbs slightly when irritated.

borus Niger, made by evaporating the Commercial Tincture.

In 8 min. aur. gorged, beating; ven. motionless and white. In 10 min. frog could barely turn over, and neither leaps nor crawls	After 13 min. frog lay powerless on back. No respiration observable	—	—
In 9 min. ven. white, motionless, and contracted; aur. beating. Vol. p. unimpaired	In 13 min. ven. motionless, contracted, pale, but not white; aur. gorged	—	—
P. 17. No peristalsis or protrusions	Systole instantly followed by relaxation. Ven. pale, but tends to stop in dilated state; no protrusions or peristalsis. Vol. p. much impaired, as frog can only just turn over. In 17 min. ven. motionless, distended	P. 7. Ven. immediately relaxing after systole. Limbs very rigid	After 40 min. frog quite powerless. Ven. motionless and contracted, red. Aur. gorged.
In 12 min. ven. motionless, dilated. In 13 min. ven. again beating 11 per min. The contractions instantly followed by dilatations; ven. shows slight tendency to peristalsis and protrusions	In 14 min. ven. again stopped, dilated. Afterwards distinct protrusions	Heart stopped, dilated. Vol. p. almost lost	After 48 min. animal rigid and apparently dead. Heart as before.
—	P. 43, regular	—	After 1 hour P. 44. After 1½ hour no further effect observed.

4. *Experiments with an Extract of*

No.	Date.	Weight of frog in grains.	Nature and quantity in grains of poison.	Initial rate of pulse.	Effects in about 5 minutes.
67	April 3, 1865	272	9	50	In 4 min. P. 32, becoming gradually slower. In 6 min. P. 26, regular
68	April 1, 1865	620	7·5	58	P. 46
69	April 1, 1865	489	3·5	40	—

Table V.—*Experi-*

No.	Date.	Weight of toad in grains.	Nature of poison, and weight in grains.	Initial rate of pulse.
70	Dec. 24, 1864	320	Extract of Helleborus Niger, from Guy's Hospital Dispensary	39
71	Dec. 23, 1864	266	Digitaline, ·023 gr.; next ·1 gr.; a third time a large quantity	22

the fresh Rhizome of Helleborus Niger.

In about 15 minutes.	In about 25 minutes.	In about 35 minutes.	FURTHER EFFECTS.
P. 18, regular. Frog lies on back paralysed, but makes feeble attempts to turn over	—	—	In 43 min. heart contracted and motionless, dark in colour. Frog dead.
P. 37, regular	In 26 min. distinct peristalsis, with a protrusion at apex of ven., which is dark purple	In 35 min. heart scarcely beating. Vol. p. not obviously impaired	In 65 min. ven. motionless and contracted; when cut across, a little blood escaped. Aur. motionless, full of blood. Frog crawls feebly, dragging its posterior limbs.
—	In 26 min. P. 32. No other effect observable	In 36 min. P. 22, feeble, but regular	After 47 min. vol. p. lost. P. 18, with a tendency to protrusions and peristalsis. After 1½ hour frog dead and rigid; but aur. beat feebly, and a single peristaltic wave passes over ven. from base to apex every few seconds.

ments on Toads.

Effects produced after 5 minutes.	Effects produced after 20 minutes.	FURTHER EFFECTS.
—	P. 18, quite regular	After 50 min. P. 24, regular. After 2 hours vol. power not impaired.
Immediately before third injection P. 30. No effect produced by previous doses of dig. Large quantity finally given, dissolved in water, injected into abdomen. Some came into contact with heart	P. 10. Ven. remains long dilated	After 45 min. P. 8. Heart sometimes motionless and dilated for a time. After 80 min. P. 6. Vol. p. destroyed.

TABLE VI.—*Showing the Effects of various Prepa-*

No.	Date.	Weight of frog in grains.	Nature of preparation of digitalis employed, and dose in grains.	Initial rate of pulse.	Effects in about 5 min
72	Jan. 23, 1865	775	Infusion = $2\frac{1}{3}$ gr. leaves	20 (much hæmorrhage at first)	After 4 min. P. 10, regular
73	Jan. 23, 1865	640	Infusion = $2\frac{1}{3}$ gr. leaves	21	After 4 min. P. 25, regular; after 7 min. P. 27
74	Jan. 28, 1865	330	Ethereal extract of aqueous infusion = 6 gr. of the leaves	31	P. 36, regular
75	June 20, 1865	210 (emaciated frog)	2 gr. alcoholic extract, suspended in water	40, feeble	Dilatations of ven. affect apex only; no definite protrusions. Aur. beating. In 6 min. pulse regular, very slow
76	June 29, 1865	540	$2\frac{1}{4}$ gr. same extract in water	52	After 6 min. protrusions; after 7 min. ven. dilated only at long intervals. Vol. p. unimpaired
77	June 29, 1865	432	$2\frac{1}{4}$ gr. same extract in water	64	—

rations of the Leaves of Digitalis Purpurea on Frogs.

In about 10 min.	In about 15 min.	In about 25 min.	In about 35 min.	FURTHER EFFECTS.
P. 12. Frog struggles, and ventricular rhythm varies No protrusions	After 20 min. marked peristalsis and protrusions. P. very slow	In 30 min. ven. white and motionless. Frog struggles	After 34 min. frog, cut loose, crawls feebly; feebleness perhaps attributable partly to cold, partly to previous hæmorrhage	In 52 min. the frog had given no further signs of life.
—	After 13 min. P. 28, peristalsis and protrusions of most marked and characteristic kind	After 23 min. beats of ven. form slight waves at very long intervals. Ven. pale pink. In 28 min. ven. motionless, pale pink. Musc. p. unimpaired	In 34 min. ven. beating feebly at long intervals, and with slight protrusions. Animal still tolerably active	In 47 min. heart motionless, pale and livid; when ven. cut across, a drop of blood escaped. Musc. p. as before.
Marked protrusions; aur. gorged. In 12 min. no protrusions, but one beat of ven. to two of aur., and ven. chiefly white and contracted	Ven. white, contracted, and motionless. Aur. beating. The vol. p. of the animal is not impaired	Ven. still white and contracted, but dilates slightly at long intervals	—	After 44 min. ven. white and contracted. Frog can turn over readily, and sits up.
Ven. motionless. Aur. beating. Frog lies helpless on back, but struggles to turn over	—	—	—	—
—	In 13 min. ven. contracted, pale, not quite white, a slight blush passing along left edge at intervals; aur. gorged, beating regularly	—	—	In 50 min. lies powerless on back. In 1 hour 42 min. frog quite dead.
In 8 min. ven. motionless, contracted, and white, except a rare protrusion. Aur. beating. In 10 min. P. 22, contn. of ven. following each alternate beat of aur.	After 20 min. frog sluggish, gasps, and sinks on to its belly, though it can still leap. Vol. p. not impaired	—	—	After 44 min. vol. p. decidedly impaired. After an hour frog could scarcely move at all.

No.	Date.	Weight of frog in grains.	Nature of preparation of digitalis employed, and dose in grains.	Initial rate of pulse.	Effects in about 5
78	June 20, 1865	320	3 gr. same extract in water	46	In 3 min. P. Apex pale for s minutes; afterw beats irregular force
79	Feb. 11, 1865	335	A large quantity of the extract prepared by evaporation of the tincture	44 (hæmorrhage)	In 7 min. P. Rhythm irregu Pupils dilated
80	Feb. 11, 1865	310	ʒj tinct. of digitaline evaporated to small bulk	38 (hæmorrhage)	P. 28. Tende to protrusions
81	Feb. 7, 1865	947	Aqueous solution of alcoholic extract. Dose = 1 gr. of extract	23	P. 33
82	Feb. 11, 1865	423	That part of alcoholic extract soluble in ether. Dose = [illegible] gr. of extract	34	—

In about 10 min.	In about 15 min.	In about 25 min.	In about 35 min.	FURTHER EFFECTS.
In 9 min. the ven. motionless and contracted, not white. Aur. gorged, beating. Frog can still crawl feebly, and turn over	—	Animal now completely paralysed. Heart in same condition	—	—
P. 16. Pupils widely dilated	P. only 14; the ven. pausing in the dilated state. No protrusions or peristalsis	Peristalsis, as with digitaline, each alternate diastole alone complete. P. 9. In 30 min. P. 4 or 5. Ven. rather dilated, livid. There is still marked peristalsis	In 36 min. ven. motionless, contracted, nearly white. Frog can crawl, but feebly; cannot turn over	In 70 min. ven. pale at apex; the rest purple, motionless. Vol. p. extinct.
P. 16, regular	After 20 min. P. 13. Ven. wrinkled during systole, and rhythm irregular. No protrusions	Heart as before. Musc. p. much impaired	—	In 56 min. heart of a dark purple colour. Musc. p. extinct. Four other experiments with this substance yielded similar results.
P. 33. Pupils, which were contracted, dilating	Peristalsis and protrusions. P. 20; sometimes regular, then irregular	In 30 min. P. 15. Marked protrusions on white base. A little later, ven. white and contracted, scarcely beating	—	In 46 min. ven. motionless, making only an occasional feeble dilatation, with protrusions. Pupils widely dilated. In 2½ hours heart in same state. Motor power much impaired.
P. 32. In 13 min. apex white and separated by ridge from body of ven.	P. 31. In another min. characteristic protrusions. In 18 min. one beat of ven. to 3 of aur. After 20 min. dilatations rare and slight. Pupils not dilated	In 22 min. ven. almost motionless, white, contracted. Aur. gorged, beating. Frog rather sluggish. In 27 min. struggled violently	—	After 70 min. ven. again beat at rare intervals. Frog could turn over. Another experiment with this substance yielded similar results.

TABLE VII.—*Showing the Effects produced on Frogs by Alcoholic*
and of similar Extracts of

No.	Date.	Weight of frog in grains.	Nature of extract used, and quantity, in grains of dose.	Initial rate of pulse.	Effects in about 10 min.	In about 20 min.
83	Dec. 14, 1864	470	14 gr. of faintly acid extract of human post-mortem gastric contents	39	After 5 min. ven. slightly corrugated, one part of it not dilating freely. After 10 min. P. 18, more regular	After 15 min. P. 17, regular, but laboured. After 20 min. ven. pauses, dilated; its rhythm irregular. Occasional protrusions very like those produced by dig.
84	Dec. 14, 1864	525	14 gr. of same ext.	41	—	P. 32, regular
85	Dec. 22, 1864	401	14 gr. of same ext.	34	Ven. now beating peristaltically, 4 beats in the min.; immediately afterwards stopped dilated. Vol. p. unimpaired	Ventricular systole a mere wave. Ven. during the pause dilated and liver coloured. Frog sluggish, but can turn over
86	Dec. 13, 1864	755	14 gr. of same ext.	33	In 5 min. P. 35, full, regular	After 15 min. P. 35. Apex of ven. slightly pale; dilatations feeble. After 20 min. heart's action not quite uniform; no definite protrusions
87	Dec. 23, 1864	256	7 gr. of same ext.	41	In 5 min. P. 42	—
88	Dec. 13, 1864	397	14 gr. of a similar ext., without any of sediment	37	After 5 min. P. 37, regular, feeble	After 15 min. P. 28, regular, feeble

Extracts of Fluids taken from the Human Stomach after Death, Vomited Matters, or Urine.

In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
P. 28. Slight peristalsis and an occasional protrusion	Frog lies with chin on ground, is torpid, but can leap and turn over	Slight peristalsis observed	In 77 min. frog tolerably active and leaped. P. 36, heart beating regularly and fully	The animal was then killed.
After 25 min. P. regular, laboured	Ven. stopped, dilated. After 3 min. beats recommenced and were peristaltic. It again stopped, dilated, and subsequently again resumed its action, beating regularly 20 per min.	Frog sluggish; can crawl feebly, and turns over when placed on back. Otherwise as before	After 52 min. heart sometimes motionless and dilated, at others makes a few regular beats. Frog does not attempt to turn over when placed on back	Not observed after 52 min.
—	Frog lies powerless when placed on its back	Ventricle mostly gorged and dilated, but occasionally makes a slight peristaltic beat	After 1¼ hour slight quivering of limbs. Heart again made a few peristaltic beats. The ven. is still dilated	In 110 min. heart feebly beating at intervals, and of deep venous colour. Frog rigid, dead.
No further effect	—	P. 32, regular. Apex of ven. contracts later than base. Vol. power slightly, if at all, impaired	—	In 110 min. ven. beating at long intervals, but fully. Vol. p. as before. Half an hour later same report is made.
After 25 min. P. 34. Slight irregularity in action of ven.; it pauses, dilated, and there is slight peristalsis	P. 27, regular. No protrusions	—	In 1¼ hour P. 13, regular. Frog is stiff, quite dead	—
After 34 min. the ven. purple, beating slowly, so that its whole surface is pale at the same moment. P. 23	Frog is almost completely paralysed	—	—	In 105 min. heart livid, beating very feebly, but regularly

No.	Date.	Weight of frog in grains.	Nature of extract used, and quantity in grains of dose.	Initial rate of pulse.	Effects in about 10 min.	In about 20 min.
89	Dec. 14, 1864	545	21 gr. of same ext., with some of the sediment	40	Ven. dark, mostly dilated, beating at long intervals	In 15 min. vol. p. extinct
90	Dec. 14, 1864	220	14 gr. of same ext., with some of sediment	48	After 5 min. P. 46, regular, feeble. In 7 min. P. 16.; ven. remains distended during the pause. The frog appears dead	—
91	Dec. 23, 1864	384	12 gr. of same ext. as in Exp. 83, but purified by precipitation with water	54	After 6 min. peristalsis. 1 min. later ven. beating regularly 24 per min., tending to remain dilated. In 10 min. P. 40. Irregular rhythm of ven., peristalsis, and almost definite protrusions	In 15 min. P. 44, regular. In 20 min. vol. p. not impaired
92	Dec. 17, 1864	704	21 gr. of ext. of a post-mortem gastric fluid	31	—	In 15 min. P. 35, regular
93	Dec. 22, 1864	429	7 gr. of another ext. of a post-mortem gastric fluid	37	P. 42, regular, feeble	In 15 min. frog torpid; respiration much quickened. In 20 min. P. 42. Respiration rapid. Frog vainly tried to turn over from back
94	Dec. 22, 1864	456	3·5 gr. of another ext. of a post-mortem gastric fluid	33	P. 31	P. 28, regular rather feeble
95	Dec. 7, 1864	341	Unknown quantity of a similar ext.	52	—	—
96	Nov. 17, 1864	395	Unknown quantity of a similar ext.	42	After 5 min. P. 48	P. 45, quite regular

In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
—	—	—	Frog dead. Ven. purple and dilated, still beating at long intervals	—
No change. Killed.	—	—	—	—
Vol. p. as before. P. 48, with slight tendency to peri-stalsis	—	Heart beating regularly. Vol. p. scarcely impaired. Killed	—	—
In 25 min. P. 33, regular	P. 36, regular. Vol. p. not markedly impaired	Vol. p. decidedly impaired	P. regular. Killed.	Only this exp. made with this ext.
After 25 min. P. 42. Frog crawls	P. 46, regular, feeble	—	After 71 min. frog again vigorous; its heart's action regular	After 4 hours frog lively, its heart beating regularly. Only this exp. made with this ext.
—	P. 32. Frog sluggish; vol. p. not decidedly impaired	—	P. 34, regular, feeble	In 95 min. ven. stopped, dilated. Vol. p. good. 1½ hour later both the heart and vol. p. remained in same state. Only one exp. made with this ext.
—	—	—	No effect in 1½ hour	Same fluid, when extracted with acetic acid, poisonous to frogs (*vide* exps. 117, 118).
—	P. 38, regular; as no effect, dose repeated after 44 min.	P. 17, feeble and laboured. Vol. p. extinct	Heart in same state	All this ext. was used in this one exp.

No.	Date.	Weight of frog in grains.	Nature of extract used, and quantity in grains of dose.	Initial rate of pulse.	Effects in about 10 min.	In about 20
97	Dec. 23, 1864	698	15 gr. feebly acid extract of healthy dog's stomach	22	P. 24. Ven. corrugated. No protrusions	—
98	Dec. 23, 1864	342	14 gr. of same ext.	26	In 5 min. P. 22, feeble. In 7 min. very feeble	In 15 min. less feeble. min. P. 10. impaired
99	Dec. 23, 1864	294	7 gr. of feebly acid extract of a vomit	32	In 5 min. P. 33	In 15 min. regular
100	Dec. 23, 1864	254	7 gr. of same ext.	32	In 5 min. P. 25. In 10 min. P. 12, regular, laboured	In 15 min. full, regular. min. P. 9, reg
101	Nov. 17, 1864	—	Unknown quantity of extract of a vomit	63	—	P. 49, regul
102	Oct. 19, 1864	463	Dialysed vomit. Ext. of what did not pass out. Dose unknown	58	—	Heart actin gularly

In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
P. 19. Ventricle pauses after every 2 or 3 beats. (Room rapidly rising in temperature.)	—	—	P. 20, full. Rhythm irregular. After 1½ hour ven. tends to stop dilated, and beats with indistinct peristalsis and protrusions. Frog appears dead	In 150 min. ven. beating regularly; previously there were protrusions. Limbs contract slightly when mechanically irritated.
In 25 min. ven. liver-coloured, motionless, dilated. In 30 min. ven. again beating 9 per min.	—	—	P. 14, regular. Vol. p. much impaired	After 120 min. ven. still beating, but frog dead. This frog much smaller than the one used for the last exp.
In 25 min. P. 12, regular	P. 12, regular. Vol. p. impaired	—	P. 24. Vol. p. impaired. After 1½ hour ven. motionless, dilated. Frog rigid; irritating legs produces slight convulsive movements	After 130 min. ven. beating regularly at long intervals. Frog rigid and dead.
In 25 min. movements of frog very feeble. In 30 min. P. 6, not regular in rhythm. Ven. dark, aur. gorged	—	—	P. 10. Condition of frog same as at last report	After 2 hours ven. beating at long intervals. Frog now only makes a few quivering movements.
—	Heart as before. Vol. p. unimpaired	—	After 54 min. ven. pinkish, rather small, its action scarcely to be detected. After 1½ hour quite regular; frog struggles when pinched, and jumps pretty actively, but lies for a time passive when placed on back	After 2 hours heart beating feebly, regularly. Vol. p. extinct. This the only exp. made with this ext.
After 25 min. imperfect action of ven., which remains pinkish during diastole, and does not fill with blood. Vol. p. unimpaired; momentary rigidity of limbs	P. 78. Ven. not contracted, as with dig.; its diastole very slight. Vol. p. slightly impaired	—	P. 63. Heart in same state. Vol. p. decidedly impaired. In 1½ hour heart stopped, dilated, but afterwards resumed its action for a second or two, beating regularly	After 2 hours P. 54, heart's action nearly normal. Frog could crawl, but not leap.

No.	Date.	Weight of frog in grains.	Nature of extract used, and quantity in grains of dose.	Initial rate of pulse.	Effects in about 10 min.	In about 20 m
103	Oct. 19, 1864	465	Unknown dose of same ext.	68	—	—
104	Oct. 26, 1864	554	Unknown dose of same ext.	52	P. 56	Heart bea feebly, but regularly
105	Oct. 26, 1864	342	Unknown dose of same ext.	58	P. 60	—
106	Feb. 23, 1865	732	Unknown dose of crude ext., used also for mice	38	P. 43	P. 38, feeble
107	Jan. 26, 1865	312	Unknown dose of crude ext. of vomit, one of those used for rabbits and mice (all the alcohol not expelled)	42	In 5 min. P. 44, regular. In 10 min. P. 38. Vol. p. unimpaired	P. 36
108	Jan. 31, 1865	265	Unknown dose of same ext. (all the alcohol expelled)	29	In 5 min. P. 20. Pauses between beats of aur. and ven., the latter white during systole. In 10 min. P. 18, regular. Frog passive, but doubtful whether vol. p. is really impaired	P. 19, regu feeble

In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
P. 68, regular, feeble	Vol. p. slightly impaired	—	P. 64, regular. Frog leaps, but vol. p. impaired	After 100 min. no change.
Vol. p. perhaps slightly impaired. Action of ven. a mere wave passing over surface of heart. Ven. neither contracted nor dilated	P. 54, regular, very feeble. Vol. p. decidedly impaired	—	Vol. p. impaired. After 1½ hour heart beating regularly, but feebly	After 2 hours heart livid pink, beating very feebly. Frog sluggish, leaps when irritated. When cut across, ven. contained little blood.
P. 56	—	No effect. Dose repeated. Immediately diastole of ven. became deficient, and was instantly followed by contraction	Ven. small, pointed, pink, scarcely beating. Vol. p. unimpaired. After 81 min. vol. p. much impaired. Ven. still pink, beating very feebly	Ven. did not cease to beat.
P. 35, regular, but feeble	Motor power impaired	—	P. 30, very feeble. Ven. small, but not white. Mot. power much impaired. In 70 minutes P. 31, heart's action very feeble	—
P. 46, feeble. Vol. p. unimpaired	P. 50, very feeble. Sudden loss of vol. p. 5 min. after, P. 34	P. 22. Ven. reddish; its action barely perceptible	—	—
P. 19, scarcely perceptible. Vol. p. as before	—	P. 17, scarcely perceptible	In 1½ hour P. very feeble. Vol. p. almost extinct	—

No.	Date.	Weight of frog in grains.	Nature of extract used, and quantity in grains of dose.	Initial rate of pulse.	Effects in about 10 min.	In about 20 min.
109	Jan. 31, 1865	264	Same ext. in same state	29	In 5 min. P. 36. There was slight irregularity of rhythm, a minute or two after the injection, the ven. tending to pause, dilated. After 10 min. P. 26. Irregular rhythm, but no protrusions	In 15 min., ven. paused dilated; then P. 3, and whole heart gorged. No peristalsis or protrusions. After 20 min. P. 3, peristalsis. Systoles of ven. only at long intervals, whole heart much gorged during the intervals
110	Jan. 14, 1865	414	Unknown quantity of very viscid crude ext., used also upon a dog	40	—	—
111	Jan. 14, 1865	306	Same ext. diluted with weak alcohol	39	In 5 min. P. 34, feeble. In 10 min. P. 26	In 15 min. vol. p. slightly impaired. In 20 min. P. 26, feeble, regular
112	Jan. 14, 1865	352	Same ext. diluted with water	28	Temporary peristalsis after a struggle. 1 min. later P. 12, regular, very feeble; ven. tending to remain dilated. Frog distressed	In 18 min. P. 10. Heart red, large. In 21 min. ven. motionless, dilated. Motor vol. p. much impaired
113	Oct. 15, 1864	608	Unknown quantity of extract of urine	40	—	—
114	Oct. 15, 1864	383	Same ext.	50	After 5 min. P. 46	An occasional protrusion at apex (from injury?)
115	July 1, 1865	560	Ext. of vomit of patient affected with sporadic cholera	56	—	P. 48, regular

In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
One beat of ven. per min., with one protrusion at base on right border. Heart touched with forceps, after which it beat better. It then stopped, dilated, next beat 2 per min. Motor power nearly lost	P. 12, sharp, regular	—	Ven. contracted, white; aur. beating, not gorged. In $1\frac{1}{2}$ hour heart as before. Vol. p. almost extinct. Killed.	—
P. 40, no effect	P. feeble, regular. Vol. p. not impaired	—	—	No effect in 2 hours. This might be due to the viscidity of the ext.
—	In same state	—	Ventricle presents white constriction; aur. beating. Afterwards ven. again made a few imperfect beats. Frog lies quiet for the most part; can still struggle	Very like effects of dig., except late onset of heart symptoms and early impairment of vol. p.
After 25 min. ven. beating regularly, 16 per min.	P. 11, regular. Vol. p. almost lost	—	Ven. dusky red, semi-contracted, scarcely beating. Frog struggles	In 107 min. frog rigid, nearly dead; in 4 hours quite dead; ven. dilated.
—	—	—	No effect	—
—	—	No effect. Dose repeated	No effect	—
—	P. 48, regular	After 47 min. injected remainder of ext.	P. 48, regular. After 73 min. slight sluggishness	After 2 hours P. regular. Frog sluggish and feeble.

TABLE VIII.—*Showing the Effects produced on Frogs by two Acetic Extracts of Fluids taken from the Human Stomach after Death.*

No.	Date.	Nature and quantity of the extract administered.	Weight of frog in grains.	Initial rate of pulse.	Effect after 5 minutes.	Effect after 10 minutes.	Effect after 15 minutes.	FURTHER EFFECTS, AND REMARKS.
116	Dec. 7, 1864	Unknown quantity of ext. of fluid taken after death from a human stomach	336	49	P. 44, regular	—	In 15 min. peristalsis, and ven. very livid. After 17 min. ven. motionless, distended, and of a uniform colour. The frog completely paralysed and apparently dead	After 25 min. heart distended, motionless; limbs move slightly when mechanically irritated. Frog again breathes. Only this experiment made with this ext.
117	Dec. 8, 1864	Similar extract, having an acid reaction	420	60	P. 50. Ven. livid, distended; after 6 min. much distended, scarcely beating	Ven. passive, merely yielding to impulse of blood from auricles. P. 40	P. 46, very feeble. Vol. p. much impaired	After 50 min. P. 38, not quite so feeble. Slight movements of anterior limbs the only signs of vol. p. Same fluid, extracted with spirit, innocuous to frogs (*vide* exp. 95).
118	Dec. 8, 1864	Same quantity of above extract rendered alkaline by carbonate of sodium	600	52	Ven. stopped for a time, dilated, with the dark and the scarlet blood in its two sides, well defined and separate. It soon resumed its action	—	P. varies from 12 to 22; rhythm irregular. Vol. p. almost extinct	After 50 min. ven. dilated; with the exception of an occasional beat, motionless. Vol. p. extinct.

TABLE IX.—*Showing the Effects produced on the Higher Animals by Alcoholic Extracts of the Vomits of Human Beings.*

No.	Date.	Effect of the extract on frogs.	Quantity of the extract administered in grains.	Kind of animal employed.	EFFECT PRODUCED.
119	Feb. 6, 1865	Poisonous	160	Rabbit	No effect. The extract mixed with water introduced into a wound in the back.
120	Feb. 6, 1865	Poisonous	20 gr. same ext.	Mouse	After 2 hours animal found dead. It was still warm.
121	Jan. 31, 1865	Poisonous	20 gr. same ext.	Mouse	No effect after $2\frac{3}{4}$ hours. Found dead 20 hours after administration of the extract.
122	Mar. 31, 1865	Poisonous	Unknown dose of similar extract	Mouse	In 2 hours very ill. When the limbs are pinched they and the whole body are thrown into clonic contractions. In $2\frac{1}{2}$ hours mouse dead. Respiration feeble. On exposure the heart was beating feebly. The skin of the animal was soaked with the viscid extract. Was its death due to this cause?
123	Mar. 31, 1865	Poisonous	Unknown dose of same extract	Mouse	No obvious effect 2 hours afterwards; but the animal was found dead after 20 hours.
124	Jan. 18, 1865	Poisonous	100 gr. of similar extract	Dog	No effect.

TABLE X.—*Showing the Effects on Frogs of various Substances which might have been present in the Poisonous Extracts.*

No.	Date.	Weight of frog in grains.	Nature of substance employed, and dose in grains.	Initial rate of pulse.	Effects in about 5 minutes.	In about 20 minutes.	In about 40 minutes.	FURTHER EFFECTS.
125	Dec. 13, 1864	437	7 gr. human bile	30	P. 36	—	—	In 50 min. heart's action very feeble. Frog still active.
126	Dec. 13, 1864	910	3·5 gr. same bile	34	In 10 min. P. 32, regular	P. rather feeble. In 25 min. feeble action and imperfect diastole of ven. Not like the effects of dig.	In 40 min. motor power unimpaired	After 50 min. imperfect diastole of ven., but it is not contracted, and is pink. After 2 hours frog in same state.
127	Dec. 19, 1864	398	7 gr. caramel	21	—	—	In 40 min. temporary protrusions during a struggle	In 50 min. P. 20, regular. After 1½ hour vol. p. unimpaired; after 2 hours slightly impaired and P. very feeble.
128	April 27, 1865	392	Unknown dose of solid pepsine	62	—	—	P. 56, regular	No effect in 1 hour.
129	April 27, 1865	356	Infusion of pig's stomach (pepsine)	80	P. 72	—	—	No effect in 1 hour.

130	Dec. 8, 1864	257	·5 gr. lactic acid in 10 parts water	41	—	After 25 min. no effect. Dose repeated (see next exp.)	—	Same frog to which butyrate of sodium previously given (exp. 138).
131	Dec. 8, 1864	257	2 gr. undiluted lactic acid	—	Ven. stopped, dilated, in 3 min. Blood faintly acid	—	—	Same frog as in exp. 138 and 130.
132	—	570	1 gr. lactic acid in 10 parts water. Then 2 gr. undiluted acid	48	P. 48. In 10 min. P. 48, pehaps slightly feeble	In 15 min. blood apparently neutral. Dose repeated	Ven. pale, not contracted, beating feebly	—
133	Dec. 18, 1864	330	5 gr. lactic acid in 3 parts water	58	Heart's action at once slower, and in 1¼ min. stopped dilated. Vol. p. impaired	Heart as before. Frog dead	—	—
134	Dec. 7, 1864	361	5 gr. glacial acetic acid in 3 parts water	46	In 1¼ min. ven. motionless; whole heart purple and distended. Motor power unimpaired till 10 min., and then most in hinder limbs	Vol. p. almost lost. Much blood escaped on section of ven.	—	—
135	Dec. 13. 1864	560	5 gr. acetate of sodium, in water	26	P. 35, regular	—	—	No effect.
136	Dec. 3, 1864	413	·875 gr. butyric acid with 10 parts water	—	In 1 min. ven. scarcely beating, distended, purple. After 2 min. frog sluggish, but could turn over from back. In 6 min. ven. beating feebly, then motionless. Blood faintly acid	—	—	·154 gr. previously given without effect.

No.	Date.	Weight of frog in grains.	Nature of substance employed, and dose in grains.	Initial rate of pulse.	Effects in about 5 minutes.	In about 20 minutes.	In about 40 minutes.	FURTHER EFFECTS.
137	Dec. 7, 1864	341	5 gr. butyric acid with 3 parts water	48	In 2 min. P. 40, feeble. In 4 min. P. 34, almost extinct. In 6 min. P. 25, very feeble. Ven. during pause neither contraced nor dilated	In 12 min. ven. stopped, dilated. Frog crawls and leaps. Ven. in same state in 20 min.	—	—
138	Dec. 8, 1864	257	·154 gr. butyrate of sodium in water	51	P. 45	No effect. 1 gr. more given	No effect in 30 min.	Frog used for exp. 130 and 131.
139	Jan. 18, 1865	470	18·5 gr. of 20 per cent. methylated spirit	36	P. 40, regular	P. 34, regular	In 30 min. P. 38, regular. In 40 min. vol. p. much impaired	—
140	Jan. 16, 1865	458	18·5 gr. pure alcohol (20 per cent.)	26	P. 31, regular	In 15 min. P. 28, regular	In 30 min. frog torpid, crawls. P. 28, regular	In 50 min. P. 26, regular. Vol. p. much impaired. After 1¼ hour frog was in the same state.
141	June 29, 1865	460	10 gr. alcohol (same strength)	58	In 10 min. P. 48	In 15 min. P. 52	After 35 min. heart as before. Frog a little sluggish. After 50 min. leaps and struggles, though not forcibly	After 70 min. quite active.

142	July 1, 1865	577	15 gr. alcohol (20 per cent.)	42	In 10 min. P. 45	Slightly insensible to irritation, and vol. p. impaired for an instant, then struggled violently	Crawls and struggles after 30 min.	After 2½ hours P. regular, and frog active.
143	July 1, 1865	213	20 gr. alcohol (20 per cent.)	64, feeble; hæmorrhage	In 10 min. P. 52, regular, but feeble	In 15 min. P. 46. Frog lies helpless on back	—	After 63 min. P. regular. Frog struggled feebly.
144	June 29, 1865	426	20 gr. alcohol (20 per cent.)	62	In 10 min. P. 64. Draws up leg instantly on irritation	P. unaltered. Lies with legs extended, but can struggle violently	In 36 min. lay helpless on back. P. 60, regular	After 57 min. leaped rather feebly; after 1¼ hour in same state.
145	July 1, 1865	208	30 gr. alcohol (20 per cent.)	60	In 7 min. P. 56. Frog lies helpless on back, but crawls and moves limbs feebly	—	—	After 54 min. P. regular; frog quite paralysed.
146	—	496	60 gr. pure alcohol, of 42 per cent.	—	In 10 min. P. feeble, regular. Vol. p. almost lost	In same state after 25 min.	—	—
147	Dec. 13, 1864	570	7 gr. undiluted methylated spirit	33	In 7 min. P. 27, rather feeble	P. 28, regular, rather feeble. Frog can just crawl	—	In 50 min. P. 30. Ven. purple. Frog lies passive on back. In same state 20 min. later.

TABLE XI.—*Showing the Effects on Frogs of various Alcoholic previous to*

No.	Date.	Weight of frog in grains.	Nature of extract used, and maximum dose of digitaline possibly present.	Initial rate of pulse.	Effects in about 10 min.	In about 15 min.	In about 20 min.
148	Nov. 10, 1864	295	Ext. of human post-mortem gastric fluid. Maximum of dig. ·055 gr.	36	—	P. 39	—
149	Nov. 12, 1864	304	Same dose of same ext.	39	—	—	P. 37, regular
150	Nov. 10, 1864	335	Similar ext. Maximum of dig. ·055 gr.	37	In 8 min. apex of ven. pale, base forming protrusions. White contracted part increased in area	P. 40, as before. Afterwards ven. dilated only with a slight crimson blush, nearly white during intervals	P. slow; intervals between the dilatations long and irregular
151	Nov. 12, 1864	389	Same extract. Maximum of dig. ·028 gr.	37	—	—	Every alternate diastole of ven. only full, the other consisting merely of slight protrusion at apex
152	Nov. 10, 1864	697	Ext. of urine. Maximum of dig. ·055 gr.	32	P. 29. No effect	—	Peristalsis after struggling
153	Nov. 12, 1864	373	Same dose of same ext.	50	—	In 12 min. crimson pouches and white patches succeed one another on surface of ven.; its action very irregular	Effects continue well marked

Extracts of Animal Fluids to which Digitaline had been added Extraction.

In about 25 min.	In about 30 min.	In about 40 min.	In about 1 hour	FURTHER EFFECTS.
Ven. dilating only at left border, the rest contracted and white. Left margin forms crimson protrusion	After marked protrusions ven. almost ceased to beat, quite pale	Heart beating feebly. Vol. p. not impaired	Ven. motionless; cut across, nearly empty. Vol. p. unimpaired	—
—	Peristalsis, then protrusions. 2 min. later ven. motionless, contracted; aur. gorged, beating	In 34 min. vol. p. slightly impaired	Heart motionless, pink, but much paler than the engorged auricles	After 77 min. frog could still leap. Ven. cut across contained no blood.
Heart beating more fully; ven. purple	Vol. p. not impaired	Ven. motionless. Vol. p. good	Ven. motionless, pale, not quite white. Vol. p. unimpaired	—
P. 36, more regular. Apex of ven. dilates imperfectly	Protrusions. Apex still pale	In 36 min. ven. motionless, white; aur. gorged; vol. p. at first impaired, then frog struggled violently	After 50 min. ven. redder, then again white. After 1 hour motor power unimpaired	After 72 min. ven. pale, motionless. Cut across, it contained no blood. Frog could crawl and leap.
Crimson protrusion at apex, marked off by a constriction. Afterwards extraordinary peristalsis	—	After 36 min. ven. beating slowly in same manner. Vol. p. good	—	—
Ven. purple, beating more slowly. Protrusions	Vol. p. slightly impaired. Ven. pale pink, dilating only at long intervals, with a slight blush	Afterwards ven. dilated, forming dark purple protrusions. Vol. p. much impaired	After 50 min. frog killed. Ven. then purple, and contained some blood	—

TABLE XII.—*Showing the Effects on Frogs of an Acetic Extract of Digitaline was added*

No.	Date.	Weight of frog in grains.	Maximum quantity of dig. present in dose of extract given.	Initial rate of pulse.	Effects in about 5 minutes.	In about 10 minutes.
154	Dec. 6, 1864	352	·2 gr.	56	In 2 min. peristalsis; in 6 min. protrusions. P. 34. Effects are as yet quite like those of dig.	P. slow. Ven. remaining dilated during pause
155	Dec. 1, 1864	305	·066 gr.	47	In 4 min. peristalsis. In 6 min. very irregular action of ven.	P. 32. Paleness of ven. and protrusions on its surface
156	Dec. 6, 1864	374	·066 gr.	50	—	—
157	Dec. 1, 1864	375	·031 gr.	50	—	—

TABLE XIII.—*Showing the Effects on Frogs of Extracts of Urine, had been added, and which were subjected to*

No.	Date.	Weight of frog in grains.	Nature of extract, and maximum quantity of dig. present.	Initial rate of pulse.	Effects in about 5 min.	In about 10 min.
158	Oct., 1864	361	Ext. of vomit: what passed through the dialyser. Maximum of dig. ·032 gr.	50	P. 51, laboured	—
159	Oct., 1864	450	Same ext.; maximum of dig. ·016 gr.; then a second dose containing a maximum quantity of ·064	57	—	—

a Fluid taken from a Human Stomach after Death, and to which previous to Extraction.

In about 15 minutes.	In about 30 minutes.	In about 1 hour.	FURTHER EFFECTS.
In 13 min. ven. dilated, motionless, except an occasional beat. No protrusions. Vol. p. of limbs somewhat impaired	In 20 min. ven. motionless, liver-coloured. In 25 min. ven. bleeds on section	—	—
In 20 min. P. 26. Ven. beating in manner very characteristic of dig.	Ven. motionless, not quite white, except at apex. Vol. p. decidedly impaired	In 40 min. ven. beating very feebly. In intervals is contracted and nearly white	After 90 min. ven. beating very feebly. Vol. p. greatly impaired.
In 15 min. P. 38. In 18 min. characteristic protrusions and white patches, with tumultuous beating of ven.	P. 24. Rhythm very irregular. Well-marked protrusions and white patches	Vol. p. unimpaired. Most marked protrusions with every beat of ven.	Ven. bled when cut across 80 min. after administration of the ext.
In 20 min. P. 50, regular	P. regular	No effect. Killed	—

Vomited Matters, and post-mortem Gastric Contents, to which Digitaline Dialysis before being extracted by Alcohol.

In about 15 min.	In about 20 min.	In about 30 min.	In about 50 min.	FURTHER EFFECTS AND REMARKS.
—	—	In 25 min. P. 48. No effect	—	In 1 hour no effect. This frog afterwards poisoned with ·014 gr. dig., which produced marked results.
—	—	—	—	In 55 min. after last injection characteristic peristalsis and protrusions appeared. Motor power unimpaired. After 70 min. heart again beating pretty regularly.

No.	Date.	Weight of frog in grains.	Nature of extract, and maximum quantity of dig. present.	Initial rate of pulse.	Effects in about 5 min.	In about 10 min.
160	Oct., 1864	437	Same ext. Maximum of dig. ·125 gr.	49	P. 42	—
161	Oct., 1864	420	Same ext. dried to a solid. Maximum of dig. ·222 gr.	53	—	—
162	Oct., 1864	527	Same dose, in same state, of same ext.	56	—	—
163	Oct. 22, 1864	379	Same vomit; ext. of what did not pass through dialyser. Maximum of dig. ·045 gr.	56	—	—
164	Oct. 22, 1864	185	Same ext. Maximum of dig. ·09 gr.	41 (hæmorrhage)	—	—
165	Oct. 22, 1864	379	Same ext. Maximum of dig. ·166 gr.	Same frog as in exp. 163	—	—
166	—	353	Post-m. gastric fluid; ext. of what passed through dialyser. Maximum of dig. ·077 gr.	51—38	—	P. 41
167	—	314	Same ext. Maximum of dig. ·01 gr.	56	—	—
168	—	314	Same ext. Maximum of dig. ·25 gr.	52	In 4 min. slight protrusion showed itself at apex of ven. Heart's action afterwards churning and very irregular, with momentary protrusions	P. very slow. Diastole of ven. imperfect. In 11 min. ven. contracted and reddish, dilated only at intervals of some seconds

In about 15 min.	In about 20 min.	In about 30 min.	In about 50 min.	FURTHER EFFECTS, AND REMARKS.
—	—	—	In 40 min. irregular rhythm of ven. and peristalsis. Ven. contracting at twice. In 50 min. heart regular; immediately afterwards, protrusions and peristalsis, not very well marked	Marked peristalsis in 1 hour; heart's action slow and laboured.
—	—	—	—	No effect in 1 hour; inertness of this extract perhaps due to its being in solid state.
—	—	In 25 min. ven. tends to remain contracted	In 40 min. heart beating naturally	After 1½ hour P. 40. Heart regular.
P. 48. Heart's action regular	—	No effect in 34 min.	After 36 min. temporary protrusions during a struggle; afterwards heart's action again regular	No effect in 1 hour. This frog afterwards used for exp. 165.
—	—	—	Ven. slightly pale, probably result of hæmorrhage. No other change in its action	—
Heart's action feeble, but regular. Vol. p. much impaired	Effects precisely similar to those of extracts containing no dig.	—	In 40 min. P. 24. Ven. livid. Vol. p. much impaired	After 55 min. ven. beating feebly at long intervals; and when ven. cut across, blood escaped. Frog could still leap.
Peristalsis and irregularity of ven. After 17 min. apex pale during diastole	Ven. motionless, contracted, reddish. Vol. p. not impaired	In 25 min. vol. p. impaired somewhat. In 30 min. hardly any evidence of impairment of vol. p.	Frog killed	Same ext. in dose containing a maximum quantity of ·002 gr. dig. had been given to frog without any effect.
—	—	P. 38. No effect	—	After 30 min. dose repeated (see exp. 168).
Ven. now quite motionless. Vol. p. unimpaired	Ven. becoming darker. Vol. p. failing slightly	—	In 40 min. vol. p. a good deal impaired. Heart more livid. Animal still draws up legs when irritated	Ven. when cut into found to contain scarcely any blood.

No.	Date.	Weight of frog in grains.	Nature of extract, and maximum quantity of dig. present.	Initial rate of pulse.	Effects in about 5 min.	In about 10 min.
169	—	293	Same fluid; ext. of what did not pass through. Maximum of dig. ·019 gr.	54	—	P. 56. In 13 min. apex contracted even during diastole, and left side of ven. formed crimson protrusion
170	—	598	Same ext. Maximum of dig. ·009 gr.	38	—	—
171	—	598	Same frog and same ext. Maximum of dig. ·019 gr.	—	—	—
172	Nov. 11, 1864	400	P.-m. gastric fluid; ext. of what passed through dialyser. Maximum of dig. ·062 gr., after ·031 had had no effect	46	—	—
173	Nov. 3, 1864	525	Same fluid; ext. of what did not pass through dialyser. Max. of dig. ·038 gr.	44	In 7 min. protrusions and white patches. Diastole gradually affected less and less of ven. In 9 min. ven. motionless and contracted. Aur. gorged. Frog could crawl but feebly	—
174	Nov. 10, 1864	596	Urine; ext. of what passed through dialyser. Maximum of dig. ·033 gr.	54	In 4 min. ven. for a time almost ceased to dilate	In 11 min. well-marked peristalsis and protrusions. Ven. constricted at one part
175	Nov. 10, 1864	353	Same ext. Max. of dig. ·022 gr.	54	P. 44	Ven. corrugated, not dilating freely. In 13 min. peristalsis

In about 15 min.	In about 20 min.	In about 30 min.	In about 50 min.	FURTHER EFFECTS, AND REMARKS.
In 14 min. ven. white, contracted, motionless. Vol. p. perfect	Motor power more impaired	Ven. redder, still contracted. Frog can crawl	—	After 84 min. frog could kick vigorously. Ven. contracted, pale livid.
—	P. 40, regular	Slight protrusions during a struggle. (This often occurs when no dig. has been administered)	After 45 min. no effect	Dose repeated at end of 45 min. (See exp. 171.)
—	No effect. Dose repeated with ·033 gr. dose. Immediately afterwards protrusions showed themselves, and in 2 min. ven. motionless and white. Motor p. unimpaired. Aur. beating	17 min. later ven. contracted, but livid. Vol. p. somewhat impaired	—	—
Apex pale during systole; white spot afterwards became more distinct	—	In 25 min. dilatation of ven. now a peristaltic wave traversing surface. In 30 min. still marked peristalsis	In 40 min. ven. livid, contracted, scarcely beating. Aur. gorged. Vol. p. unimpaired. Afterwards ven. quite white	In 50 min. vol p. slightly impaired. After 3 hours aur. beating; when cut across, ven. quite empty of blood.
Frog can still jump a short distance	—	In 25 min. ven. liver-coloured, contracted; when cut across, almost empty. Frog still sits up, but does not turn over from back	—	Frog previously had ext. containing a max. of ·019 gr. dig.; its only effect in 20 min. being a temporary protrusion at apex with every other beat.
Marked protrusions. Base of ven. dilating before apex	Dilatation of ven. only a red protrusion at apex	In 25 min. ven. contracted; in 27 min. livid, a purplish blush occasionally travelling over surface	In 40 min. P. 10, regular in rhythm, very peristaltic, forming protrusion at apex. In 50 min. ven. motionless	After 100 min. ven. motionless, not absolutely contracted. Vol. p. unimpaired.
Most marked protrusions and peristalsis. P. 22	P. 18. Ventricular diastole very irregular. Pulse very slow	In 25 min. ven. motionless, contracted, livid; when cut across, almost empty. Pupils dilated	—	—

No.	Date.	Weight of frog in grains.	Nature of extract, and maximum quantity of dig. present.	Initial rate of pulse.	Effects in about 5 min.	In about 10 min.
176	Nov. 11, 1864	616	Similar ext. Max. of dig. ·011 gr.	56	Heart immediately feeble. In 3 min. ven. scarcely dilating at all. In 6 min. diastole peristaltic. All these effects probably due to compression of heart; they ceased on cutting across sternum	P. 44, regular
177	Nov. 11, 1864	616	Same frog and same ext. Max. of dig. ·022 gr.	31	Beats begin to be peristaltic	—
178	Nov. 11, 1864	429	Same ext. Max. of dig. ·022 gr.	71	—	P. 57

TABLE XIV.—*Showing the Effects on Frogs of Extracts*

No.	Date.	Weight of frog in grains.	Nature of extract, and dose in grains.	Initial rate of pulse.	Effects in about 10 min.	In about 15 min.	In about 20 min.
179	Nov. 14, 1864	—	12 gr. of ext. of liver of No. 1 dog	45	In 5 min. P. 31. In 10 min. P. 12, laboured. Ven. pauses semi-dilated	P. slow, but regular. Heart pale pinkish	Heart in same state. Vol. p. much impaired
180	Nov. 19, 1864	451	47 gr. of same ext. (see remarks at end of exp.)	56	In 5 min. P. 51	—	P. 40, feeble, but regular. Frog vigorous. After 22 min. vol. p. suddenly failed
181	Nov. 14, 1864	206	6 gr. of ext. of stomach and stomach-contents of No. 1 dog	56	P. 45. Apex of ven. white	P. 42. Apex of ven. pale, even during the diastole	Well-defined whiteness of apex. P. 42

In about 15 min.	In about 20 min.	In about 30 min.	In about 50 min.	FURTHER EFFECTS, AND REMARKS.
—	Peristalsis and marked protrusions. Apex shooting out into a red pouch	Systole and diastole consists of waves, one white, the other crimson; action very characteristic of dig.	In 40 min. P. 31. Heart still beating peristaltically	In 83 min. heart nearly resumed its normal action. Frog was therefore employed for another experiment.
Very marked peristalsis	—	In 25 min. ven. pauses, sometimes dilated, sometimes contracted. Feeble cardial action. Heart livid	Heart livid, beating very feebly	After an hour ven. dilated feebly at apex at long intervals only. After 130 min. frog found dead.
After peristalsis and protrusions ven. almost ceased to dilate	Ven. almost motionless; diastole consisting merely of a slight blush	Killed	—	—

of the Viscera of Dogs poisoned by Digitaline.

In about 25 min.	In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
Frog killed	—	—	—	—	—
—	Slightly recovered, so as to leap	P. 37	—	After 1½ hour frog dead. Ven. motionless, dilated	Ext. purified by precipitation with water, filtration, and re-evaporation.
Only small part of ven. now dilates, rest remaining white. P. 12, with intermediate [illegible]ble dilatations.	Apex now forms a crimson protrusion	Heart as before. Motor p. slightly impaired	After 45 min. ven. scarcely beating, pinkish, not very contracted	Frog killed	—

No.	Date.	Weight of frog in grains.	Nature of extract, and dose in grains.	Initial rate of pulse.	Effects in about 10 min.	In about 15 min.	In about 20 min.
182	Nov. 14, 1864	—	15 gr. of same ext.	34	In 5 min. P. 44. Irregular cardiac action. Protrusions at apex. In 7 min. P. rapidly falling from 36 to 26	Irregular rhythm continues. At end of 12 min. protrusions no longer observed	—
183	Nov. 14, 1864	—	18·5 gr. of same ext.	34	In 5 min. P. 50. In 10 min. P. 46. Red protrusion at base. Afterwards dilatation of ven. very feeble and transient, the organ mostly white and contracted	—	After contraction ven. relaxes and becomes pink; base then dilates by itself and instantly again contracts. P. 22
184	Nov. 15, 1864	409	10 gr. of same ext., previously purified	45	—	—	Line of permanent paleness on surface of ven.
185	Nov. 15, 1864	243	10 gr. of same ext. as in last exp.	44	—	Crimson protrusion at apex, gradually more marked	Distinct peristalsis and protrusions. In 21 min. ven. stopped, contracted; aur. beating
186	Nov. 16, 1864	270	9 gr. of same ext. as in last two exps.	61	In 3 min. P. 56. In 7 min. P. 48. In 10 min. peristalsis and protrusions; ven. dilates fully only 18 times per min.	P. 23. Peristalsis. Ventricle beating very feebly, now pink, now dark liver-coloured	Ven. dark. Vol. p. a good deal impaired
187	Nov. 17, 1864	246	9 gr., as in last exp.	56	In 5 min. P. 45, very feeble. Ven. tends to stop, contracted. In 10 min. P. 44. Heart pinkish; diastole imperfect; apex not dilating at all. Vol. p. much impaired	In 12 min. ven. pinkish, dilating only at long intervals	—

In about 25 min.	In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
—	Vol. p. considerably impaired	—	—	—	—
Ven. motion-[illegible], contracted; [illegible]ricles beating. [illegible]tor p. very [illegible]ch impaired	—	—	—	—	—
Apex and base [illegible]nson, rest of [illegible] white. After [illegible] min. one or [illegible] crimson pro-[illegible] form [illegible] only diastole	Ven. contracted, white, scarcely yielding at all to impulse of aur. Motor p. unimpaired	Ven. again dilating, but feebly and imperfectly. Motor p. slightly impaired	—	Ven. pale, contracted. When cut across, 13 min. later, empty. Aur. beating	Ext. purified by precipitation with water, filtration, and evaporation.
Ven. contract-[illegible] aur. Doubt-[illegible] whether mo-[illegible] p. is impaired	—	Ven. yields slightly to aur. impulse. Frog crawls and jumps vigorously, but rather sluggish	—	Ven. pale, small, contracted. When cut across, 15 min. later, empty. Even then motor p. not perceptibly diminished	—
—	—	The frog can still crawl feebly. Heart beating as before	—	Frog can crawl, but not leap	After 1¼ hour ven. motionless, liver-coloured; nearly empty when cut across.
—	After 34 min. ven. is very small, pink, almost empty. Frog dead	—	—	—	—

No.	Date.	Weight of frog in grains.	Nature of extract, and dose in grains.	Initial rate of pulse.	Effects in about 10 min.	In about 15 min.	In about 20 mi
188	Nov. 17, 1864	374	9 gr., as in last exp.	52	P. 52, regular. In 12 min. beats of ven. irregular and peristaltic	—	P. 23. A d purple protru forms at the s of ven. during diastole
189	Nov. 17, 1864	585	6¼ gr. of same ext. (see remarks at end of exp.)	50	After 5 min. P. 42, regular	—	P. 39, regu
190	Nov. 17, 1864	585	11½ gr. of same ext. in same state. Same frog	—	After 5 min. P. 33. Irregular action of ven. and peristalsis. Ven. seems inclined to pause dilated rather than contracted. After 8 min. P. about 14. Ven. mostly dilated, its beat consisting of peristaltic waves passing from base to apex	—	Heart in s state. Moto not impaired
191	Nov. 19, 1864	680	10½ gr. of same ext. in same state	54	P. 50	Marked protrusions and peristalsis. Rhythm also irregular	Ven. di only at long tervals. M p. unimpaired
192	Dec. 17, 1864	355	3½ gr. of ext. of fluid part of vomit of No. 3 dog	29	Marked protrusions and peristalsis. In 12 min. ven. motionless, contracted. Motor p. unimpaired	Ven. white, motionless; aur. still beating	—
193	Dec. 17, 1864	425	1½ gr. of same ext.	31	P. 40, regular	P. 40. Ventricular action sharp; ven. white during systole; aur. gorged	In 16 min. ristalsis and trusions; only beating for two of In 19 min. motionless, w aur. still beat

In about 25 min.	In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
Heart beating *ristaltically,* *...ly, and slow-* *During the* *...se the ven. is* *...ated and of a* *...rk purple co-* *...ur*	*Musc. p. is extinct*	—	Frog breathes, but has lain motionless since last report. Heart contains blood	—	—
—	—	—	No effect. Dose repeated (see next exp.)	—	The ext. was fir purified by pr cipitation with *large quantity* water, filtratio and evaporation
Ven. beating ...ly at long in- ...rvals with most ...rked crimson ...trusions. In ...se is purple. ...tor p. becom- ...g impaired	**In 34 min. apex of ven. contracted and nearly white**	Frog can still turn over. When cut across, ven. not empty of blood	—	—	—
—	**In 27 min. ven. motionless and contracted. Motor power unimpaired**	Ven. pale liver-coloured, still motionless	No blood escaped from ven. when cut across	—	—
Heart in same ...te. Frog ac- ...ve	—	Motor p. good	Heart in same state. Motor p. slightly failing	Frog now quite dead. A single drop of blood escaped when ven. cut across	—
Musc. p. not at all diminished	—	Ven. pale, just perceptibly beating. Motor p. good	—	—	In 1½ hou motor p. unin paired; ven. m tionless, whit When cut acros no blood escape

No.	Date.	Weight of frog in grains.	Nature of extract, and dose in grains.	Initial rate of pulse.	Effects in about 10 min.	In about 15 min.	In about 20 min.
194	Dec. 21, 1864	309	$\frac{3}{4}$ gr. of same ext.	43	P. 41, regular	P. 38. Marked peristalsis and protrusions. Afterwards apex remained contracted during diastole	Ven. pinkish white, motion less, and con tracted. Moto p. unimpaired
195	Dec. 19, 1864	239	7 gr. of ext. of solid part of vomit of No. 3 dog	28	—	P. 11. Ven. quite white and contracted, two crimson protrusions forming all the diastole. After 16 min. ven. white, altogether motionless. Motor p. a little impaired (probably from coldness of room)	Heart in sam state, exceptin an occasiona protrusion; ven perfectly white
196	Dec. 21, 1864	325	6 gr. of same ext. (see remarks at end of exp.)	41	P. 34, feeble	P. 17. Irregular rhythm of ven., which is pale during systole; diastole full. After 16 min. marked peristalsis, protrusions, and white patches	After 19 mi ven. ceased t beat. It was the at first purpl afterwards pale Frog much para lysed
197	Dec. 21, 1864	270	3 gr. of same ext. in same state	55	Marked peristalsis and protrusions. At one time ven. nearly stopped, contracted	Ven. scarcely beats, but slight crimson protrusions appear at the apex. Motor p. unimpaired	Ven. whit contracted; au beating
198	Dec. 21, 1864	236	3 gr. of same ext.	39	In 12 min. peristalsis and protrusions	—	Beats of ve now peristalti now regular
199	Dec. 21, 1864	298	7 gr. of ext. of empty stomach of No. 3 dog	45	The pause, during which ven. bright red, much prolonged; systole sharp, indistinct, and sudden; occasional protrusions	After 18 min. P. 13	No irregulari nor protrusions

In about 25 min.	In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
—	—	There has been nothing to note since last report	Ven. livid, not dilated, just perceptibly beating. Motor p. gradually becoming impaired	—	After 1½ hour frog dead. Ven. contracted, almost white; when cut across, scarcely any blood escaped.
—	—	—	—	Ven. contracted, white; cut across, al-almost bloodless. Frog moves limbs slightly, but its eyes are closed	—
—	—	—	—	Frog now appears dead. Ventricle, now livid, has not resumed its action since last report. When cut across, almost empty of blood	Ext. purified by precipitation with water, filtration, and evaporation.
After 27 min. Motor p. failing. Ventricle almost bloodless when cut across	—	—	—	—	—
Ven. mostly crimson, nearly motionless, diastole consisting merely of a slight peristaltic wave, with protrusions	Motor p. not impaired	Ven. white and motionless, looking like a mere appendage to distended auricles. Musc. p. somewhat impaired	Heart as before. Motor p. more impaired	Ven. quite empty when cut across	—
Ven. stopped suddenly, dilated, then beat again very slowly	—	P. 14, regular and full	P. regular, very slow. Motor p. almost extinct	P. 15, regular. Frog lies passive	—

No.	Date.	Weight of frog in grains.	Nature of extract, and dose in grains.	Initial rate of pulse.	Effects in about 10 min.	In about 15 min.	In about 20 min.
200	Dec. 22, 1864	607	7 gr. of same ext.	27	P. 20, regular	—	—
201	Dec. 19, 1864	335	3½ gr. of ext. of fluid part of gastric contents of No. 2 dog	About 28	P. 24, regular	—	Well - marked peristalsis and protrusions
202	Dec. 17, 1864	370	3½ gr. of same ext.	29	P. 29, regular	White constriction round ven., above the apex	A protrusion at apex. P. 33
203	Dec. 17, 1864	282	1½ gr. of same ext.	36	P. 43, regular	—	—
204	Dec. 21, 1864	160	7 gr. of ext. of solid part of vomit of No. 2 dog	36	Peristalsis and protrusions at apex, at first indistinct, then marked and characteristic	—	In 18 min. ven motionless, contracted, white; auricles gorged Motor p. slightly impaired
205	Dec. 21, 1864	244	3½ gr. of same ext.	50	After 5 min. heart's action very feeble	Marked peristalsis and protrusions. Ven. small, purple	Ven. pale liver-coloured, beating at rare intervals with an occasional protrusion Motor p. much impaired

In about 25 min.	In about 30 min.	In about 40 min.	In about 50 min.	In about 1 hour.	FURTHER EFFECTS, AND REMARKS.
P. 24, regular	P. 28, regular	P. 30, regular. Motor p. somewhat impaired	Heart's action regular. Frog lies passive on his back	—	In 77 min. frog apparently recovering. After 2 hours ven. motionless, dilated. After 197 min. ven. again beating; animal quite active.
Each alternate systole of ven. a mere protrusion at apex	Ven. motionless, contracted, white; aur. gorged, beating; musc. p. unimpaired	Ven. beating, but very slowly, and with a protrusion	—	—	After 70 min. ven. again motionless, white, contracted; motor p. unimpaired. When ven. cut across, a little blood escapes.
Ven. motionless, white; aur. gorged, beating; motor p. unimpaired	—	After 44 min. ven. as before; motor power slightly impaired	—	Heart as before. Animal still jumps	In 84 min. frog dead. A little blood escaped when ven. cut across.
—	Ven. white during systole. Marked temporary peristalsis has been several times observed	Heart's action regular	P. 38. Renewed peristalsis and protrusions; ven. livid	P. 17, regular. 5 min. later systole peristaltic, attended with slight protrusions	After 80 min. motor p. unimpaired.
—	—	—	Frog dead. Ven. contracted, pale red, yielding blood when cut across	—	—
—	—	After 43 min. ven. of same colour, but pointed. When cut across, yielded some blood; auricles gorged. Frog appeared dead	—	—	—

TABLE XV.—*Experiments on Frogs wit*

No.	Date.	Weight of frog in grains.	Quantity in grains of poison.	Initial rate of pulse.	Effects in about 5 minutes.	In about 10 minutes.
						Experiment
206	Nov. 19, 1864	307	·02	55	—	P. 56, regular. Heart bright red
207	Dec. 6, 1864	485	·048	47	—	Heart very pale during systole; not red as in other experiments
208	Dec. 6, 1864	245	·1 (some lost)	36—40	Ven. bright florid red. In 8 min., during struggle, a bright red protrusion at apex. During some of heart's beats base also showed a tendency to form protrusions	P. 40. Apex protrudes and dilates sometimes before base. Afterwards heart quite regular
209	Dec. 23, 1864	304	·1	48	P. 44. Irregular rhythm. Heart pauses dilated every few beats. After 7 min. P. 24	Heart stopped dilated, making an occasional beat, with slight protrusion at base. Vol. p. almost lost
210	Dec. 22, 1864	376	·143	51	P. 52. Heart's action more feeble	—
211	Mar. 31, 1865	355	About ·167	49	P. 53	Ven. dark liver-coloured. Peristalsis suddenly set in, with pallor of apex, but no absolute protrusions. A few seconds later P. 28, regular. After 12 min. only one beat of ven. to two of aur.

Emetina and with extract of Staphysagria.

In about 15 minutes.	In about 20 minutes.	In about 30 minutes.	FURTHER EFFECTS.
with Emetina.			
—	—	P. 64, feeble. Heart bright red. Frog leaps vigorously	No further effect. Killed.
—	P. 46. Ven. slightly constricted across its centre during diastole. A minute later again beats fully	P. 42, regular	In 1½ hour P. 34, heart's action regular. Motor p. unaffected. 3 hours afterwards heart beating normally.
Ventricular diastole distinctly peristaltic, with tendency to protrusions, but not well marked. Ven. bright red, tending to remain dilated. In 17 min. irregular rhythm and only 22 beats of heart per min.	Muscular power very much impaired. Heart beating pretty regularly, 18 per min.	—	After 45 min. dilatations occur at twice after a single systole. P. 11. Musc. p. as before.
—	—	—	The animal carefully watched, but no further change occurred. At the end of an hour frog dead and stiff; heart distended with blood.
Heart's action regular. Motor power unimpaired	Ventricular beats 26, auricular 52, per min. Systole regular. After 25 min. P. about 17	After 35 min. P. 19, irregular in rhythm. Ven. not contracting quite uniformly, but no definite edge to redness or pallor, so that appearance unlike that after dig. Frog lies with chin on table, but can struggle	After 50 min. P. 18; contractions of ven. alternating with those of aur. Frog then killed.
P. 18. Ven. still contracting once for 2 beats of aur., remaining dilated during intervals. Heart very dark. Motor p. much impaired	Ventricular beats 10, regular; aur. beating rapidly and regularly. Ventricle dilated during pause	After 35 min. aur. beating regularly; ven. only at long intervals. Frog can just turn over; musc. p. very much impaired	After 1 hour heart was in same state. Frog could not turn over when placed on back.

No.	Date.	Weight of frog in grains.	Quantity in grains of poison.	Initial rate of pulse.	Effects in about 5 minutes.	In about 10 minutes.
212	Nov. 24, 1864	304	·2 altogether, of which ·077 gr. in solid state	44	P. 42	Irregular rhythm. Ventricular contractions form waves, with tolerably distinct protrusions at different parts. Whole heart bright red. Ven. tends to remain dilated

Experiments with Alcoholic

No.	Date.	Weight of frog in grains.	Quantity in grains of poison.	Initial rate of pulse.	Effects in about 5 minutes.	In about 10 minutes.
213	Jan. 17, 1865	196	Extract of 2 drachms of seeds	24	P. 29	—
214	Jan. 26, 1865	531	Extract of 2 drachms of seeds	39	P. 44, regular	P. 41, regular

Experiment with Ethereal Washings

No.	Date.	Weight of frog in grains.	Quantity in grains of poison.	Initial rate of pulse.	Effects in about 5 minutes.	In about 10 minutes.
215	Jan. 28, 1865	—	—	46	In 3 min. rhythm of heart irregular; action of ven. sometimes quickened, sometimes slow, or altogether ceasing for a time. Heart dark liver-coloured. No peristalsis or protrusions. In 6 min. muscular power extinct	After 8 min. heart dark purple, beating peristaltically and feebly, and often with well-marked protrusions

In about 15 minutes.	In about 20 minutes.	In about 30 minutes.	FURTHER EFFECTS.
Heart's action still much like that due to dig. Systole only a white wave traversing ven. from base to apex, at long intervals. P. only 18	After 25 min. frog struggles actively, leaped on to floor. Ven. afterwards small, pale, scarcely beating	Heart dilating more fully, but peristaltically and feebly. After 35 min. heart livid red, beating feebly	After 45 min. P. 48, feeble, but without tendency to stop contracted. Frog still turns over.

Extract of Staphysagria.

P. 15	After 25 min. P. 9, regular. Heart dilated during pause, very pale during systole	P. 9. Systole not quite uniform, apex and base remaining red a little after the rest of ven. has become pale	After 45 min. ven. motionless, red, contracted rather than dilated; aur. beating. Motor p. now first examined, extinct. After 65 min. frog quite dead. Ven. motionless, purple, moderately contracted. Aur. beating slowly.
P. 36, regular. Heart liver-coloured. Both motor p. and sensation seem completely extinct	P. 33, regular. For some time no respiration observed	P. 30, regular. There is still complete paralysis	After 45 min. P. 28, regular. After 53 min. marked peristalsis and protns., first noticed after touching heart with forceps. After 68 min. cardiac action feebler, markedly peristaltic. After 87 min. heart's action regular, feeble, 16 per min.

of Alcoholic Extract of Staphysagria.

Heart beating pretty regularly, dark purple; protrusions and peristalsis when ven. touched with forceps	—	—	After 1¼ hour ven. red, still beating feebly.

TABLE XVI.—*Experiments upon Dogs, to each*

No. and Date.	Method of Experimenting.	Effects in about 15 minutes.	In about 30 minutes.	In about 45 minutes.	In about 1 hour.	In about 1½ hour.
216, Nov. 5, 1864. No. 1 dog	Chloroform given; poison injected into stomach through œsophagus; lastly, œsophagus tied. Poison dissolved in 5 ounces very dilute alcohol	Frequency of pulse appeared to vary, difficult to count, but estimated, after 4 min., at 124; afterwards 90. In 7 min. 106. In 15 min. 90. After 23 min. again 120	Respiration long drawn, shuddering, accompanied by quivering movement of lower limbs. Animal runs about staggering, as if drunk, falls on one side. Sensation seems lost in posterior limbs, which are more paralysed than anterior	Animal sits with hind legs doubled up under him; still trembling movement of limbs. Saliva trickles from mouth; no attempts to vomit. P. 112, regular	Animal regained its muscular power; has been led across the road; seems not much the worse for injection. Just before made violent attempts to vomit	—
217, Dec. 15, 1864. No. 2 dog	Operation and injection of the poison effected in same way	After 5 min. P. 127	Animal moves constantly, retches. Is weak, listless, and falls on to the side. Is this the effect of the chloroform?	Renewed attempts to vomit	P. intermitting, varying in rapidity, first 88, then 100, afterwards 128. Dog ill; can at times stand; retching again	Sits, swaying from side to side, and moaning. When pinched does not appear to feel
218, Dec. 15, 1864. No. 3 dog	No chloroform was given; poison was injected by mouth	Vomiting at first prevented by tying string over mouth	Dog vomited easily, and without effort, twice	Lies quietly on side. When roused stands up; fell once when standing	Again vomited easily. A large quantity of frothy mucus discharged, as well as stomach contents	Falls down when placed on legs. Lies slavering with mouth on ground

of which five grains of Digitaline were given.

In about 1¼ hour.	In about 1½ hour.	In about 2 hours.	In about 2½ hours.	In about 3 hours.	FURTHER EFFECTS.
—	—	—	—	—	Was not further watched. Found dead 3¼ hours after injection of poison. Death supposed to have occurred in about 2½ hours.
P. about 150	Renewed and violent attempts to vomit	Lies quiet; moans almost inaudible. Afterwards retches violently, and howls loudly	After being laboured, respiration calmer. Dog sits up	After 3½ hours P. about 160. Sometimes beats suddenly slower for about six beats. Heart's action seems to intermit; respiration sighing, audible. Animal appears not to feel pinching or pulling the hairs; lies quiet; constant trembling of limbs	After 3¾ hours dog suddenly started up, retched violently, and fell off table; afterwards appeared dead. Heart's sounds inaudible, but a quivering sound of muscles of chest heard. Chest at once opened. While this being done two or three deep inspirations made. Even after left pleura opened dog breathed several times and heart contracted once or twice after an inspiration. Touching heart with points of scissors caused it to become contracted for two or three min. Heart found dilated when chest first opened.
Attempts to stand, but reels and falls	Retches violently and repeatedly, but brings nothing up	Still staggers and falls when raised. Is quite quiet	Is now livelier, raises his head, and takes notice. It would appear that he cannot stand	Found dead 2¾ hours after administration of poison	Heart found, half an hour later, not contracted, containing blood in all its cavities.

OBSERVATIONS

ON THE

PATHOLOGY OF SOME OF THE DISEASES OF THE NERVOUS SYSTEM.

By SAMUEL WILKS, M.D.

The following remarks are in substance the same as those which I delivered to my class during the last winter session; but the cases which illustrate them are, for the most part, taken from my post-mortem records of the last twelve years. I wish it, therefore to be understood that the present paper is to be regarded rather as an hospital report than as a lecture.

My object has been to endeavour to find some scientific basis for a classification of nervous diseases, so that the organic affections, at least, might be arranged on a definite principle, and that we should not be obliged to isolate such diseases as apoplexy, softening, paralysis, dementia, &c., which, from the form of expression, would seem to have nothing in common. There may be, of course, a question as to the propriety of altering names; and whether, indeed, such general terms as those which I have mentioned may not be less objectionable for practical purposes than any founded on a more precise method. But at the same time it must be allowed that, for purposes of instruction, the grouping of cerebral affections on some more scientific basis must be of the utmost advantage. On considering whether we should follow a strictly pathological, or a physiological, method of classification, I think all will agree that the latter is to be preferred. For when it is remembered how complex an organ is the brain, consisting of parts having very different functions, it is evident that a particular morbid change cannot always be productive of the same symptoms. Thus, the effects of softening, or tumours, or abscesses, are different in different cases, and a treatise on these diseases without reference to precise situation would afford but indefinite information. On the other hand, since the symptoms are in the main dependent on the part of the cerebro-spinal centres affected, the physiological method presents us with accurate knowledge, and must therefore have greater attractions for us. It is certain that it is often of far more importance to know what part is affected, than what is the character of the disease; its site is what we generally first seek for, leaving the cause to be considered afterwards. Without, however, discussing further the relative merits of the two nosological systems, it is sufficient to say that the discovery of the connection between particular symptoms and definite nervous lesions is of the utmost importance in a clinical sense, and of the extremest interest from a physiological point of view.

Of late years there has been a wise endeavour to carry physiological knowledge to the bedside of the patient; but it is remarkable how recent has been the inquiry in this direction, for, even with the experience which they then possessed, our immediate predecessors seemed to consider that all attempts to localize the seat of brain diseases were futile. Indeed, even at the present day there are some, and those men of eminence, who, having paid no especial attention to this subject, declare that we are still working in an unknown territory, and that little can be really learned of cerebral affections, inasmuch as tumours, abscesses, and sloughings of the brain, and even gun-shot wounds, may exist without any apparent results. When such views prevail no surprise need be felt at the remarkable opinions sometimes expressed in our courts of law as to the effects of injuries; as, for example, that the plaintiff could not have received any hurt to the brain, seeing that he had no paralysis; or, again (if paralysis was said to be present), that it must have been factitious, because it was on the same side as the blow, which (they say) could not be, and so on. The work which has recently been done in this department of medicine proves, however, that such a statement of our ignorance is not correct, although it is true that it is only of late years that much accurate knowledge has been obtained in this direction. In our modern text-books we find scarcely an attempt to place nervous diseases on a scientific basis; and in the works which immediately preceded them, such as the valuable lectures of Graves, the authors appear rather to be endeavouring to ignore the efforts made to accomplish this object. Thus, Dr. Graves, alluding to the attempt made by the French school in the direction of positive medicine, says, after stating how little light is thrown on the symptoms of cerebral disease by the post-mortem appearances found in cases of such disease, "In fine, without detracting from the true value of morbid anatomy, these facts prove that the attempt to connect symptoms with diseased alterations of structure is attended with many difficulties, and is often impracticable." Far be it from me to speak lightly of such worthies as Graves; it is sufficient to say that the physiological study of nervous disease is a new pursuit.

It must be theoretically true that alterations in particular parts of the nervous structures are attended by definite and certain symptoms; and, practically, it must be equally true that, could such lesions exist as results of disease, corresponding phenomena must occur.

I think the last few years have added much to our knowledge in this direction,[1] the physiologist having assisted the pathologist, and *vice versâ*. The object has been to discover the seat of the disease rather than its cause; for it is clear that a softening, a sanguineous effusion, and an abscess, may produce exactly the same symptoms, if seated in the same part of the brain; whereas, when situated in different parts of the cerebral structures, affections of the same nature have little in common, so far as their effects are concerned.

[1] The workers in our country in this department are several, but I may refer more especially to Dr. Hughlings Jackson, Dr. Ogle, and Dr. Broadbent. An interesting essay by the latter in the 'Med.-Chir. Rev.' has been published since the substance of the present essay was written. As regards spine disease, I should mention the valuable papers by Dr. Gull in previous numbers of this work, and in reference to functional nervous diseases, which are equally worthy of study, I must not omit the name of Dr. Handfield Jones.

Our nosology at present possesses no method; and as regards the nervous system more especially, the terms used are expressive in one case of the morbid change, in another of the phenomena produced, and in a third of the cause, so that they have, indeed, nothing in common. On the other hand, analogous morbid states are widely separated. For example, an acute cerebro-meningitis, attended by active delirium, is found in the ordinary category of diseases treated by the general physician; whereas a chronic cerebro-meningitis, attended by a slower derangement of the bodily and intellectual faculties, is styled general paralysis of the insane, and is ignored by the same physician, and handed over to the alienist. Again, a patient with a tumour in his brain is sent to a lunatic asylum if his mind suffers. For many purposes (especially in reference to treatment) it may be advisable, and even necessary, to make a distinction between these cases; but when a patient with such a tumour comes before an ordinary practitioner, who keeps him at home or sends him to an asylum, according as the mind is deranged or not, it should be remembered that the two conditions do not differ pathologically.[1]

[1] Many terms in general use, such as apoplexy, epilepsy, &c., are wanting in definition. As regards the first, such an alteration has taken place in its meaning that this word is not only used in a sense differing from that in which it was originally employed, but is most commonly applied to those very cases to which it was formerly inapplicable. As described by Abercrombie and nearly all authors after him, the name of apoplexy was given to cases in which certain phenomena existed; but at the present day the majority of medical men use the term only for cases in which an effusion of blood has occurred. The phenomena mentioned by Abercrombie consisted mainly in a sudden loss of consciousness; but such cases are probably epileptic, and are not due to an effusion of blood, so that, according to modern usage, they are not cases of apoplexy. On the other hand, the term is now mostly used as synonymous with effusion of blood. But, when this occurs, there is often no loss of consciousness, nor, indeed, any of those symptoms which were formerly thought to characterise the disease. We are thus in this predicament—we may use the term in its original sense, remembering that the case to which we apply it is exactly that in which the majority of medical men at the present day would not employ it; or we may join with them and apply it to the case of effusion of blood, remembering that this is just the case where, according to its original meaning, the term apoplexy is inapplicable. That the term has long lost its original signification as applicable to the case of a person suddenly struck down, is clear from the use of the very absurd expression "apoplexy of the lung," by which we always mean effusion of blood.

The same difficulty arises with other medical terms, as, for instance, epilepsy. Is this to be defined by symptoms alone, or upon some theoretical pathological standard? If by symptoms, we must include (as was formerly done) cases of tumour,

Our knowledge of the physiology of the nervous system has depended in part upon experiments on animals, in part upon the experiments made for us in the human being by disease. That the field is still open for further investigation is clear from the new facts which have quite recently been displayed before us by Brown-Séquard and others. Among the experiments which have afforded the best results have been those on the spinal cord and the nerves, commencing with the celebrated discoveries of Sir C. Bell as to the distinction between the motor and sensitive roots. As regards the brain, I believe more has been done by the study of its diseased states; indeed, I consider the results so obtained to be more reliable than those derived from vivisection or direct experiment. In this place, without intruding on the province of the physiologist, I propose to state as briefly as I can, and in general terms, the connection between particular affections and diseased states of the nerve centres. We shall thus approach the consideration of the method of framing a scientific classification of nervous diseases on a pathological basis. I believe that by these means some generally correct conclusions can be arrived at, although it must be admitted that, with our present imperfect observation, there is scarcely a single statement which might not be met by the introduction of a solitary exceptional case. In the actual state of science we are, to a great extent, obliged to confine ourselves to general propositions.

In dealing with the functions of the nervous system, it is clear that the practical physician and the physiologist should be in accord; and when we consider how different are the methods which they employ, it is satisfactory to find that their results

exostosis, and other organic diseases; if we exclude these affections, we must apply the term to those cases only where no appreciable lesion exists.

Again, if a medical man be asked to define "croup," he will probably answer that it is a tracheitis accompanied by the formation of a false membrane; but, at the same time, he will declare that in practice he seldom proves the fact, and that in reality he means by croup a disease characterised by a certain set of symptoms. Any cause of impediment at the upper part of the larynx whereby a peculiar ringing respiration is produced is called croup; and thus we find simple laryngitis and diphtheria confounded with it. The medical man of whom we are speaking has one definition theoretically, another practically. So common is this that I regard all statistics concerning the operation for croup as worthless, if the term croup is used in the sense generally given to it in books. I have reason to believe that most of the cases which have recovered after the operation were really instances of simple laryngitis.

approach so nearly as they do. Moreover, the medical man, whilst treating the diseases of the brain, has very often at the same time to deal with the various operations of the mind which are intimately associated with it. Indeed, should he really investigate with full interest the various examples of brain disease which come before him, he can scarcely avoid being psychologist as well as physician; and I venture to affirm that already, by regarding mental operations in their physiological and medical aspect, the true explanation has been given of many of the obscure phenomena of mind. Pure metaphysics, in spite of their important revival by Sir W. Hamilton, appear to be becoming a subject of the past; and it is now seen that those who engage themselves in the study of psychology are fain to employ the true inductive method, and to derive their conclusions from observation and experience in the same way as in every other branch of positive science. Thus it is that the more advanced opinions of the later metaphysicians have tended in the same direction as those of physiologists, and that psychologists are now compelled to study mental operations as observed in their fellow-men, and no longer wrap themselves up in their own self-consciousness and evolve every conclusion from the inner self. It would be absurd for the metaphysician to adopt his own method, and arrive at different results from the anatomist and the physician, who are studying the physiology of the brain in health and in disease. The psychologist can no longer ignore the fact that the brain is the material organ of the mind, and that he must study its nature and its operations under the most varied circumstances before he can establish a true mental philosophy.

In studying the nervous system as physiologists or pathologists, we must first regard it in its material aspect, and then endeavour to discover its uses. We see, in the first place, every part of the body supplied with nerves which proceed from or to definite centres. The superficies of the body is provided with nerves of sensation, which pass to the spine, and by this channel to the sensorium within the cranium. From this centre, again, proceed other nerves which are distributed to the muscles for the purposes of locomotion, and which convey to them the necessary stimulus. But, besides these, the blood-vessels and the different organs of the body are supplied by nerves which pass to their own centres, and of which, except under certain circumstances, the sensorium takes no cognizance.

The whole working of the machinery is thus performed without the exercise of will or feeling on our part, and it is only under morbid conditions that we become conscious of derangement, by means of the connections between the organic and the spinal systems of nerves. The cutaneous or sensitive nerves pass to the spinal cord, and then upwards to certain ganglia or large masses of nervous structure in the middle of the brain. From these same parts proceed the motor nerves. Experiments have shown that special powers reside in the centres of the spinal cord, as well as in these ganglia, and that a stimulus conveyed by an afferent nerve will excite in it a power or force which shall pass back by a motor nerve. Thus, if the cord be severed, so that all connection between the sensorium and the limbs is cut off, movements may be excited in them by a direct stimulus to the skin. This is true of the whole spinal system, including the cerebral ganglia of which I have already spoken. For if the hemispheres of the brain of an animal be removed, many of the functions of life can still be performed during several weeks or months of existence. From these facts we learn that a large part of our actions are probably of an automatic kind; and just as a bird may fly away without its head, so in the act of walking we use a piece of machinery which may act quite independently of the influence of the brain proper and the will. The volitional power created in the hemispheres may start the machinery, just as an engine-driver by opening the valve gives the first impetus to the steam; but in either case the machine subsequently proceeds by itself, a superior force again stepping in only to arrest its movements. It is therefore probable that the spinal cord, whose summit is in the brain, contains the working power of the body; while the cerebral hemispheres have the power of control, and are also the seat of the intellectual operations and of the consciousness and the will. But these, again, may be separated, and, just as a silent working is pursued in the spinal centres, so also, in all probability, processes of intellect may be carried on in the cerebral hemispheres without our direct knowledge or consciousness. Having arrived at this point, we have got rid of many troublesome ideas and expressions concerning the operation of the will in all the various processes and operations of the body; and in studying the diseases of this complex organ, the brain, we shall be able to unravel many of the morbid phenomena attending them. For example, in a case of paralysis we shall be able to say whether the machinery or the controlling power is at fault. In studying such a subject in a purely scientific spirit, we must free ourselves entirely from effete metaphysical notions, the great errors underlying all systems of mental philosophy being such as require an intense effort to get rid of—the strong feeling of individuality and of the power of our will, and the belief that all bodily and mental operations are the results of our own consciousness. It may be true that, were we not impressed with a strong sense of individuality, we could not be active members of society, nor could we as men and women be appealed to as responsible and moral agents.[1] Since this is the necessary constitution of our being it is not remarkable that the mass of persons should believe that all operations, both of body and mind, proceed from their own consciousness, and that they should turn to this for all information regarding the mind and its operations. Indeed, it has been left to the present period to adopt a positive philosophy, and to find that, in order to study man and all the phenomena connected with him, we must give up the older metaphysical method, make observations on his being and attributes under all circumstances of change, and in disease as well as in health, and place him in comparison with the lower animals. Turning our observations in this direction, we soon find that very many operations in our body belong to us in common with inferior creatures, and that, in fact, a large part of the cerebro-spinal functions are carried on quite irrespectively of any rational influences. The spinal cord and the two large ganglia at its summit constitute a system which

[1] "Such materialistic writers as Miss Martineau would argue that, if the brain be the organ of the mind, and if it be acted on by outward impressions, we can have no control over our thoughts and passions; that we are, in fact, irresponsible beings. But, granted that this is true to the extent to which such writers would proceed, yet if it be also conceded that we have power of will sufficient to set out from our house or to place one foot before another, the argument fails; for even should an individual feel himself totally unable to repress certain emotions or thoughts when placed in particular circumstances, so as not to be master of his passions, he can at any rate avoid running into danger, and thus be in harmony with the divine prayer "lead me not into temptation."

regulates a large part of the bodily operations, and to which the terms "sensori-motor," and "ideo-motor" have been applied by Professors Carpenter and Laycock. Many of the so-called electro-biological and mesmeric phenomena have been explained by showing that these centres of activity may be played upon by external influences quite irrespective of the will. And, even more than this, there is every reason to believe that sensory impressions may be conveyed beyond these ganglia to the cerebral hemispheres, and there, setting in active operation that wonderful machinery, may produce purely mental results without any necessary consciousness on the part of the individual. This is certainly completely overturning the doctrines of Descartes and his school, that psychology is limited to the domain of consciousness, that the mind always thinks, that being and knowing are the same, and that even during sleep and stupor consciousness exists. Such doctrines are now being superseded by a more positive philosophy. It certainly would have astonished those of the older metaphysical school to have heard the opinion broached that, so far from consciousness being the basis of all intellectual operations, it might in some cases be a result of them. But even some of these schoolmen were unable to resist the logic of facts; for Dr. Laycock quotes Cudworth, who says it is certain "that our human souls themselves are not always conscious of whatever they have in them, for even the sleeping geometrician hath at that time all his geometrical theorems and knowledge someway in him. We have all experience of our doing many animal actions non-attendingly which we reflect upon afterwards." Also Tucker, who says, "It has generally been remarked by schoolboys that after having laboured the whole evening before a repetition, to get their lesson by heart, but to very little purpose, when they rise in the morning they shall have it current at their tongues' end without any further trouble." And Sir W. Hamilton—"I do not hesitate to maintain that what we are conscious of is constructed out of what we are not conscious of." Mr. Mill, in his examination of Sir W. Hamilton's philosophy, commenting on this, says, "I am myself inclined to admit unconscious mental modifications in the only sense in which I can attach any very distinct meaning to them, viz., unconscious modifications of the nerves. It may well be believed that the apparently suppressed links in a chain of association, those which Sir W. Hamilton considers as latent, really are so; that they are not even momentarily felt, the chain of causation being continued only physically, by one organic state of the nerves succeeding another so rapidly that the state of mental consciousness appropriate to each is not produced." I think every one must admit that we have no will to control the succession of ideas; we can only divert our thoughts into another channel by putting some new object before us; and if we are not capable of this our highest mental power is wanting—we are, in fact, mad. We have no control over our thoughts; during sleep they wander on just as they please, even in the sanest person.

"Sleep, kinsman thou to death and trance and madness." [1]

Mr. Mill has always maintained that experience is the foundation of all knowledge, and has ignored the so-called necessary mental condition by which conclusions of an *à priori* character are arrived at; and the same method must apply to the study of the mind. It is clear, then, that the doctrine of "unconscious cerebration" is not held by materialistic physiologists only, but is now regarded by psychologists as one of the laws of our being. It might appear that the subject to which I have been alluding has nothing to do with the pathology of nervous disease; but I take it that the two are intimately connected, and, moreover, that a just and scientific view of this interesting piece of philosophy is of great assistance in the comprehension of many phases of animal and vegetable life.

In speaking of the brain as the organ of the mind, I intend to say that we are ignorant of any mental manifestations except through the brain. I think I echo the opinion of the great majority of our profession in saying that we know nothing of the actions of the mind except through the interposition of a material organ, and that no mental process ever emanates except from such an organism, or is received except through a similar piece of machinery. Spiritualism is not in vogue with us as a profession. When speaking of the mind as resulting from the operations of the brain, I mean the *anima*. I have

[1] Tennyson's 'In Memoriam.'

nothing to do with the purely spiritualistic idea of the *animus*, or psyche, which may underlie it.[1] Thus the observations of physiologists, pathologists, and psychologists, are beginning to be in unison, and medical men need no longer have the fear of lying under the ban of teaching an infidel philosophy. We are all agreed that it is only by observation that right views can be obtained.

Summary of the functions of the Brain.—Looking once more at the nervous system, we observe the cerebro-spinal centres, with their sensory and motor nerves, and associated with these the ganglionic system. The latter is in close connection with the correct working of the whole animal machinery; but whether this result is due to some occult nervous influence, or merely to a regulation of the blood-vessels, is a point not yet positively determined. The ganglionic nerves are joined to the spinal; and thus, in abnormal conditions of the viscera, the sensorium can take cognisance of the derangement through the medium of the cerebro-spinal system proper. We find that there are two kinds of nerves, sensory and motor. Soon after the discovery of the double nature of nerves it was surmised that the motor nerves passed up the front of the spinal cord, and the sensory along

[1] To show that materialistic views with reference to the operation of the brain are not repugnant to a high spiritualistic idea of our being and of the universe at large, I might refer to the work of my colleague, Mr. Hinton, entitled, 'Man and his Dwelling-place.' In this the author maintains that we are living in a spiritual world, but that the soul in its present habitation is shut in, and has no other communication with the external than by means of our nerves. In the words of Lorenzo to Jessica—

> "Sit, Jessica, look how the floor of heaven
> Is thick inlaid with patines of bright gold;
> There's not the smallest orb which thou beholdest
> But in his motion like an angel sings,
> Still quiring to the young-eyed cherubins;
> Such harmony is in immortal souls;
> But whilst this muddy vesture of decay
> Doth grossly close it in, we cannot hear it."

It appears to be absurd that Cruikshank, in writing a book on the philosophy of ghosts should endeavour to prove their non-existence by drawing attention to the fact that they generally appear to their friends in human attire. For then, he says, we should have to carry our belief into a world of spiritual clothes, and to consider a spiritual hat, a spiritual coat, or a spiritual pair of stockings. He forgets that, even if our friends were immodest enough to appear in the guise of our first parents, flesh and blood and bones are no less material than the clothes themselves.

its back. But observations in cases of disease soon showed that such opinions were erroneous, and Brown-Séquard has recently proved by experiment that the motor tract is chiefly in the antero-lateral columns, that the sensory fibres pass more especially along the centre of the cord, first crossing to the opposite side soon after they enter it, and that even the gray substance conveys sensory impressions. Thus, a transverse section of one half of the cord produces a paralysis of motion on the same side of the body, but loss of sensation on the opposite side; while a longitudinal section through the central grey matter causes paralysis of sensation on both sides. Cases both of injury and disease have tended to corroborate these statements of the experimentalist. Besides containing these strands of fibres for sensation and motion, the spinal cord possesses a property of its own, known as the *excito-motor* function, by which a stimulus conveyed to the cord through a nerve is able to excite a corresponding motion without the animal being aware of it or having any voluntary share in the result. This is often seen in man when the spine is crushed by accident. It is a power which, under ordinary circumstances, is under the control of the will, and thus is not seen to come into play.

As regards the sensory and motor tracts, these pass along the cord, as before said, as far as the medulla oblongata, at which point the motor tracts decussate or cross to the opposite sides; they then pass on in part to the cerebellum and in part to the pons Varolii. Those which enter the pons then proceed through the crura cerebri to the thalami optici and corpora striata. Although the sensory and motor tracts run side by side, yet, in tracing them, we speak of the one, the motor, as running downwards, and of the other, the sensory, as running upwards, because the former tract carries a stimulus in the one, the latter in the other direction. Commencing at the top of the spinal system, at the ganglia, or "head-centres," we should say that the motor tract originating in them passes downwards through the crus cerebri and the pons Varolii, decussates in the medulla oblongata, and so passes on to the spinal cord. The sensory tract has been made the subject of special investigation by the physiologist, who appears to have less trouble in distinguishing the sensory than the motor portion; but, clinically speaking, the reverse has been the case, for all lesions seem to affect more particularly the parts concerned in motion. This may possibly arise, as we shall see hereafter, from the fact that there is greater difficulty in measuring loss of sensation. Hence the disagreement may not really be so great as it appears to be; a loss of motion is apparent to any one, a loss of sensation requires a test to prove it.

The physiologist tells us that in tracing the motor fibres upwards he finds them pass along the cord to the pyramids, where they cross and pass through the lower part of the pons Varolii and the crura cerebri, and then along the lower border of the thalami to end in the corpora striata. The sensory fibres pass up in like manner to spread out in the thalami optici. It is evident, therefore, that disease in the course of these fibres will produce a loss of sensation or of motion, complete or partial, according to the amount of damage done. Theoretically, we ought to meet with some cases in which the paralysis is of motion alone, and with others in which it affects sensation alone; but practically, as before said, loss of motion is much more common than loss of sensation, or, at all events, it is much more easily appreciable, and has received most of our consideration. Since the motor tract on each side of the body crosses to the opposite side in the medulla, and then separates into two distinct portions, it is clear that disease of this tract will produce paralysis of the opposite side of the body. Now, as regards the summit of the tract in the cranium, it is very unlikely that both sides should be simultaneously affected; and therefore the probabilities are greatly in favour of hemiplegia rather than complete paralysis occurring when disease originates within the brain. Without further investigation it might seem that, under these circumstances, there would be a complete paralysis of one half of the body; but physiological and pathological observations show that the motor tract, in passing up from the medulla to the ganglia above, contains more especially the fibres from the extremities, and that the nerves to the chest and those which are styled cranial nerves have distinct centres of power of their own. Thus it happens that when disease occurs in the sensori-motor ganglia the arm and leg of the opposite side are paralysed, whilst the face and other parts remain unaffected; or if they are paralysed, the loss of power appears to arise from a mere transient influence exerted upon the origin of the nerves. The independent power in the grey centres of these nerves is sufficient to preserve the tone of the facial muscles. As before said, the motor machinery of the ganglia is set in action by the influence of the will, which descends from the cerebral hemispheres; but when these ganglia are deranged by disease the influence of the will is ineffectual. Even in the case of the cranial nerves it is manifest that there is no longer a transmission of the volitional power, for it may be observed that, although the tonicity of the muscles is preserved in virtue of their independence, yet when an effort of the will is made there is an evident contrast between the affected and the opposite side of the face. Thus, owing to the independent action of the third nerve, winking and the ordinary closing of the eye may go on as usual, even when the large central ganglia are diseased; but if an effort of the will be made to close the eyes the screwing up of the healthy side is in marked contrast with the simple closure of the other; on the affected side the volitional power cannot pass.

Thus it is that in a case of paralysis of the arm and leg we diagnose disease of the ganglionic centres, those masses of nerve-matter which lie at the summit of the cord. If any special cranial nerves are involved, then we know that disease must exist in the sensori-motor tract lower down, so as to involve their roots. The spinal system may therefore be considered to extend upwards into the centre of the brain, its summit being formed by the corpora striata and thalami. The nerves of the body pass to the spinal cord, where they find their centres of action, although some of their fibres, no doubt, pass up to the sensorium. The nerves of the chest concerned in the respiratory function have their distinct centres of power, and so have also the several nerves of the face, whilst the nerves of the arms and legs especially pass up to terminate in the corpora striata and thalami. Just as two symmetrical halves make up the body, so do the nerves of the two sides meet and form one strand of fibres in the spinal cord, while their centres of power are blended in the middle as an apparently single grey mass; and so, again, do the centres of the nerves of the upper part of the body (which have no distinction in character, but are styled cranial simply because they arise from that portion of the cord which occupies the cranial vertebræ) likewise meet in the mesian line. But the extremities on each side of the body are independent members, having their own separate movements, and thus, as might be expected, the centres from which their nervous supply proceeds are also distinct and preserve their independence. I take it that had the arms or the legs respectively been united into a single limb, the corpora striata and the thalami would likewise have been united into one mass like the pons Varolii. From a consideration of the structure of the nervous system and the effects of disease, this has always seemed to me one of those self-evident propositions which scarcely wanted more proof. It is certain that these large ganglia are especially connected with the extremities, and that, although a small commissure connects them, they are remarkably independent. This isolation is made more complete by the ventricular cavity around them, a cavity almost necessitated for the fulfilment of such a purpose.

It would seem that there resides in the summit of the sensori-motor tract a power sufficient to preserve the movements of the body, and that in the ganglia there is a force which is all sufficient for such purposes as walking, eating, &c. Experiments in the lower animals have distinctly proved that such movements may go on when the cerebral hemispheres have been removed, and it is theoretically possible that, if the machinery was set in motion, a man might continue to walk although deprived of the greater part of his cerebral hemispheres. Dr. Carpenter has already fully treated of this subject, showing, for instance, that the acts of winking and coughing during sleep must be quite independent of the will.

The Will, closely connected with the intellectual operations, has its seat in the cineritious structure of the brain, and influences the ganglia below by means of the white fibres of the medullary matter. Thus it exerts a control over the machinery, very similar to that of an engine-driver over his locomotive. It has not yet been positively ascertained whether any of the nerve-fibres pass directly to the hemispheres, or whether they all terminate in the central ganglia, the further communication between these ganglia and the hemispheres being in this case effected by independent fibres. It is likewise doubtful whether all the fibres of the medullary matter do or do not converge to these centres, or whether, as would appear to be the case, some fibres pass more directly down towards the pons and lower part of the tract. It is almost certain that a very large part of the body is influenced by the will through these bodies, even if its nervous supply does not proceed directly from them. The influence which a nerve imparts differs altogether from that conveyed by the will. The distinction is one not only of physiological but of great pathological importance. The influence of a nerve centre on the part to which fibres proceeding from it are distributed is to preserve a healthy nutrition and tone. This must be allowed even if the doctrine of Dr. Radcliffe respecting the functions of nerves and muscles be admitted, for the appearance of a paralysed part is familiar to every one. A limb really paralysed is easily distinguishable, by its flaccidity and want of tonicity, from one which is simply held powerless by the side; thus, in hysteria, where the influence of the centres is good but the will is wanting, it is notorious that the natural character and the plumpness of the limbs are preserved. Dr. Marshall Hall showed that by destroying the spinal cord a flaccidity of the body is at once produced, just as when the nerves themselves are severed; this is not seen in sleep.

In saying that paralysis ensues when the influence of the nerves upon the muscular system is wanting, we must not forget the fact that, at the same time, contraction often exists. It was thought by Dr. Todd that when rigidity occurs at the onset of paralysis it shows that there is an irritation of the centres, caused by the effused blood or other disease, and that when, after a length of time, a contraction of a paralysed limb ensues, this arises from a cicatrix having been formed. I do not see any objection to the first proposition, that early rigidity is produced by irritation, or, as is so frequently seen, by the movements of the opposite side of the body causing a stimulus to be conveyed to the ganglia of the side affected; but whether the final contraction can be due to such a cause I feel in doubt. It would seem that the limb withers and contracts in most cases in which a destruction of the centres has taken place. Thus, in persons affected with a congenital contraction of a limb an atrophy of the central part of the brain has often been found, whilst, on the other hand, in those instances of infantile paralysis in which there is no proof of the existence of central lesion the limb simply wastes, is flaccid, and dangles by the side of the patient.

It may be said, then, that the cerebral hemispheres are connected with the intelligence, consciousness, and will, so that impressions made on the surface of the body are transmitted through the sensory ganglia and conducted to these parts, while, at the same time, the influence of the will passes down to act on the motor ganglia, and so on the body at large. An important practical question arises whether the volitional power can act only through these ganglia, so that, when they are destroyed, it has no longer any power over the body; this is true of the limbs, but is not positively ascertained as regards those nerve centres which exist in the lower part of the motor tract. The nerve centres of the pneumogastric and other cranial nerves are so far independent that they act even when the central ganglia are destroyed; but whether the will can exert an influence upon them except through these is uncertain. Anatomists would make fibres pass from the pons to the surface of the brain without touching the central ganglia, and it might seem from some observations in diseased persons that the will could act irrespective of the intervention of the ganglia. Probably this is a point to be settled as much by the physician as by the anatomist; for if in cases of disease of the central ganglia, when no movements can be produced in the limbs by the strongest effort, the will yet retains a power over the muscles of the chest, face &c., it follows that there must be a direct communication between the cerebral hemispheres and the grey centres in the pons and medulla. But if we, on the other hand, suppose that the only connection between the cineritious surface and the grey origins of the motor nerves is through the central ganglia, it ought to follow that when these are diseased the body must be paralysed, although the will is good. We shall also have to consider the consequences of the surface being diseased while the central parts are healthy. When this has occurred to any extent the mental power and consciousness are gone, and therefore the will with them. No doubt the analysis of the effects of such disease is often difficult, but we certainly sometimes see cases in which the power of the will is gone sufficiently to afford a contrast to those instances in which the motor machinery alone is at fault. Again, we shall have to inquire whether, when one hemisphere is affected, the volitional power is impaired, and whether paralysis, or anything equivalent to it, results. We shall, no doubt, find that when the whole surface of the brain is chronically diseased the powers of the body may remain intact for some time, although the controlling will is wanting. We shall also see that when the will fails in functional affections, such as hysteria, the effect is characteristic, for the limbs are well nourished, and the paralysed leg is dragged behind the patient, no effort being made, as in the ordinary genuine paralysis, in which we see the limb thrown forward with a jerk. We shall find, too, on reading our cases, that an injury to or disease of the surface is productive of convulsive movements, and it may be of interest to inquire whether this is due to a direct influence upon the muscles exerted by the hemispheres, or whether it arises from an excitation of the ganglia immediately below. As regards the medullary substance, it might be supposed that if this were destroyed paralysis would occur, because all communication between the surface and the centres would be cut off, but, practically, we seldom see such a result. The cause of this may be that a destruction of the white fibres sufficiently complete to isolate the centres rarely occurs, the diseases in question being mostly diffused through the medullary substance.

Disease of the central ganglia; hemiplegia.—The fact that these parts of the brain are very frequently diseased is the reason why hemiplegia is one of the commonest forms of paralysis. It is observed, whatever the nature of the alteration in them—whether this be softening, effusion of blood, or embolism of the middle cerebral artery. The cause of the great frequency of these changes is probably the large vascular supply to these bodies, and in the case of embolism the direct course of the artery from the heart to the brain.

An alteration in structure leading to a loss of function of the corpus striatum and thalamus, or of any other part of the motor tract above described, produces a hemiplegia of the opposite side of the body, but no disturbance of the intellectual faculties. An effusion of blood from the sudden bursting of a vessel may produce a temporary concussion and unconsciousness, but this is by no means constant. Hence patients who have become paralysed in the night are so little aware of the circumstance, that it is only on attempting to get out of bed that they find one side of the body to be powerless. It is a fact of everyday experience, that when called to a patient who has suddenly become hemiplegic we often find him perfectly conscious. It is true that thickness of speech may cause his answers to be unintelligible, but even this frequently passes off in a day or two. So, also, when the disease proceeds towards a cure by the absorption of blood and the formation of a cyst or cicatrix, the patient may remain permanently paralysed and yet be able to fulfil important duties, the discharge of which, perhaps, requires considerable mental effort.

As is well known, by hemiplegia is meant a paralysis of the arm and leg only, with a partial affection of some of the muscles of the face. The trunk of the body is not affected. At the onset of the attack decided paralysis of the face is observable, but this is soon recovered from; hence, when the patient is not seen till after the lapse of some hours, or even of a day or two, all that we observe may be that the tongue is thrust to the paralysed side, and that the face is slightly fallen and the speech thick. This partial paralysis of the face seemed to Dr. Todd to prove that the facial nerve was not affected, and he sought an explanation in the distribution of the motor branch of the fifth nerve. He believed that the buccinator muscle was supplied by this nerve, and thus, on the supposition that paralysis of this muscle would account for the falling of the face, he escaped all difficulty in reference to the seventh nerve. There are, however, many reasons against this explanation, as well as several in favour of the view that the facial is the nerve which is slightly or transiently affected. This is the opinion of Dr. Hughlings Jackson and of Dr. Saunders, of Edinburgh, who has got rid of Dr. Todd's explanation by showing that the buccinator muscle is mainly, if not altogether, supplied by the facial and that the branch of the fifth which passes through it is a sentient nerve. My colleague Mr. Bankart informs me that he teaches this in the dissecting room.

The circumstance that the chest is not paralysed is generally attributed to the fact that the pneumogastric and other respiratory nerves have distinct centres of force in the medulla, and this I take as a sufficient reason. I know not why we should use a phrase denoting that one half of the body is paralysed, and then endeavour to explain why certain parts on that side are exempted from the change. We should simply take the facts as we find them. Noticing certain paralytic conditions and finding definite organic lesions associated with them, we should connect the two as cause and effect. If we do this we see that disease in the central ganglia produces a paralysis of the arm and leg, with slight temporary or transient paralysis of the face. In explanation of the fact that other parts are not affected, we can only say that their nerve centres are independent of the central ganglia, and this we know to be a fact. The difficulty arises rather in connection with the question whether the will has any influence over these non-paralysed parts, acting through their nerve centres, or whether it is transmitted only through the large ganglia. Now, this has to be ascertained both anatomically and clinically. If the volitional force acts only through the corpora striata and thalami optici, and yet, when one of the central ganglia is diseased, there is power to raise at will both sides of the chest, the explanation must be that the respiratory tract is placed centrally, and that the two halves of the grey matter are blended so that the stimulus to one half is sufficient for the excitation of the whole. On the other hand, the power of the will to move the chest and other parts of the trunk when the central ganglia are diseased may be simply due to the fact (which anatomists teach us) that there is a direct communication between the pons Varolii and the hemispheres, by means of which the grey ganglionic matter in the pons and in the medulla oblongata can receive an immediate influence. These explanations are only required should it be shown as a clinical fact that the will *can* still exert an influence over the trunk. In ordinary hemiplegia the chest and abdominal muscles act as usual, but the power of a forced inspiration has not, I think, been sufficiently studied. I have certainly seen cases of hemiplegia in which the movement of the two sides of the chest were quite equal until the patient was requested to breathe deeply, but in which the ribs on the hemiplegic side were then seen not to be raised so perfectly as those of the healthy side. I have, unfortunately, not recorded cases in evidence of this, and therefore, so far as my own written knowledge goes, I must leave it an open question. In a man now under my notice the chest is not nearly so much raised on the paralysed side as on the other when he is requested to take a full breath; but this case may be an exceptional one, since there is reason to believe that there is disease in the pons Varolii. In a word, it may be said that if the will continues to influence the movements of the chest in a hemiplegic person, either there must be some connection between the seat of volitional power and the centres of the chest nerves, independent of the large ganglia, or else the regulating centres must be so associated that a stimulus to one side can affect both.

As regards the slight paralysis of the face and the implication of certain cranial nerves to the exclusion of others, I apprehend that this difference is due to some simple anatomical fact, such as the special position of the centres of these nerves in the motor tract. If all the cranial nerves originated in this tract they would be paralysed at the same time as the arm and leg; but having independent seats of power, they are influenced only transiently, and chiefly when an effort of the will is directed upon them. I should say that a certain inhibitory influence is exerted on these nerves in connection with the motor tract, and more especially on the seventh and ninth.

The lateral diversion of the eyes sometimes met with in hemiplegia has recently been under discussion, but this circumstance has long been remarked. My colleague Dr. Gull many years ago used to draw attention to it.

With regard to the loss of speech in right hemiplegia, I need scarcely say that my observations accord entirely with those of Dr. Hughlings Jackson, although the true explanation of this remarkable circumstance has yet to be discovered. It cannot be believed that the organ of speech is originally situated on one side of the brain only, and thus the explanation must be sought in some secondary cause. My colleague Dr. Moxon has offered a very good theory, which probably has much truth in it; it is to the effect that of the two halves of which the body is made up one is more especially educated, and that the other follows the movement by consent. This phenomenon, which appears so remarkable, is probably merely one instance of a general law of our bodies which has been hitherto overlooked; so far from being exceptional, I believe it to be only one example amongst numerous others, which shows how partial is the education of our muscles. In the case of writing the fact is so evident that we have never thought of its importance in reference to the physiology of the brain; as most persons write with their right hand, a hemiplegia on this side deprives them of the power to write, whereas a similar affection of the left side has no such effect. Now, when we consider that the mechanism by which writing is performed is entirely the product of education, and that the various movements are guided by nerves which are themselves under the influence of the brain, it becomes evident that one side of the brain only obtains the guiding power. Hence, were it possible for the halves of the brain to be changed over, so that the limb should retain its activity, the power to write would, I take it, still be wanting. It might, of course, be acquired, and thus our hemiplegic patients again learn to talk as they did in infancy. It is probable that for those operations in which we can use both sides of the body equally both sides of the brain have been educated alike; but since in many acts one side rather than the other has been put into use, it has followed that one half of the brain has been specially educated. As regards the movements of the limbs, this is self-evident, but it now seems to be equally true of the power of speech, and it may be true, likewise, of many other operations, as, for example, musical performances. So, again, with reference to the eye; I have heard of a man who was in the habit of using a theodolite with the right eye, and who could not employ the left for this purpose, although the sight was equally good.

There seem to be different degrees of loss of speech. Thus, in some cases there is a mere inability to articulate, whilst the patient is able to write; in others, a loss of memory of words until they are suggested; and in yet others a total forgetfulness of names, the patient giving everything a wrong appellation. It would be important to know whether these different symptoms are associated with distinct lesions; at present it is thought that loss of speech is associated especially with some disease in that part of the brain known as the island of Reil; and yet, at the same time, it is said that the loss of speech is nearly always connected with a hemiplegia. It follows, therefore, that, unless disease in the spot just named is sufficient to produce a hemiplegia, there must be an affection of the centres, extending towards the external parts.

In all the cases which I have myself seen, and, if I remember rightly, in those which have been recorded by others, there has been disease in the central ganglia, and I take it that this is necessary for the production of a hemiplegia. But the question remains, is it necessary that disease should advance beyond these parts in order to cause the loss of speech? According to the theory of the education of the one side to the disparagement of the other, a simple loss of power is all that would be required to produce this symptom; and if, as a matter of fact, loss of speech accompanies all right hemiplegia, then, assuredly, a small spot of disease in the central ganglia is sufficient. It may, indeed, be thought that a further extension of the mischief outwards to the grey matter of the hemispheres (especially in the region of the island of Reil) is essential to the further loss of all memory of words; and, on that supposition, we should be on the watch for cases in which this part is injured independently of the centres, and in which loss of speech would, perhaps, exist without any corresponding hemiplegia. Cases of loss of speech certainly occur without a paralysis of the limb, but I am not aware that they have been shown to be due to disease in the island of Reil. I have on more than one occasion seen loss of speech with right hemiplegia where the lesion was confined to the centres, but in these cases I believe that the failure of speech proceeded no further than an inability to articulate. Aphonia may also occur from disease in the pons Varolii, and I have thought that, by noting accurately this symptom in cases of disease lower down in the motor tract, we might obtain a proof of the importance of the anterior lobe in the production of speech. For example, a lady had disease in the pons Varolii; her tongue and soft palate were paralysed, so that she could not utter a word, but she could write down accurately all her wants. On the other hand, I am now seeing a patient who has somewhat similar symptoms, denoting a disease of the pons, and who has loss of speech; but before this was complete, and when she spoke intelligibly enough to make herself understood by her husband, she called things by their wrong names. The cause was clearly not in the brain proper, for the temporary paralysis which she at first had was on the left side.

If we believe that, for the production of a persistent hemiplegia, some part of the motor tract must be involved—if we find that in nearly all these cases of loss of speech with paralysis of the right side one of the central ganglia is affected—and if we also adopt the theory that this peculiarity is due to the education of one side alone—these facts would imply simply that the muscles of the tongue and palate had never learned to act in a certain definite manner; but whether the memory of words, being independent of the mechanism by which they are formed, requires another locality for its action, is very doubtful. This anatomical and physiological question merges into the old metaphysical one as to how far the idea must correspond with the outward sign. I apprehend that, according to Dr. Jackson's supposition, a disease of the left corpus striatum would necessitate a loss of the power of articulation, and, if it also involved the neighbouring cineritious structure, would likewise destroy the faculty of speech; while if the latter were the only function affected it would be surmised that the surface about the island of Reil was alone affected.

Respective functions of the ganglia.—There can scarcely be a doubt that each of the two great ganglia has its own peculiar office. The anatomist, by his dissections, shows us that the motor tract passes into the corpus striatum and the sensory tract into the thalamus; and all theoretical considerations would lead us to infer that there must be a special termination for the. motor tract, just as there must be a commencement for the sensory; although we should quite expect that the two would be intimately blended together. By clinical observation no such distinction in office can be made out. It has long been observed that, wherever the lesion is situated, paralysis of motion is more marked than paralysis of sensation. It must, however, be remarked that it is much easier to recognise loss of motion than loss of sensation. In fact, I have no doubt that the latter is often overlooked. When a man is seen in a half unconscious state, soon after a paralytic attack, it is very difficult to ascertain what amount of sensibility exists. In a large number of reports of cases of hemiplegia no mention is made of any alteration in the sense of feeling, but in several it is stated that sensation is wholly or partially lost. Hence, while there is sufficient proof that the motor and sensory tract are closely incorporated, and that a very slight lesion is sufficient to involve both, the very fact that the paralysis of sensation varies in different cases shows that the two are in parts distinct. A few well-observed cases, in which the exact site of the lesion should be carefully noted, ought to throw some light on this interesting anatomical and pathological question. At present there is, so far as my experience goes, no evidence to connect the ganglia separately with the motor and the sensory tracts.

We might suppose that, just as a destruction of any part of the tract produces loss of function, so irritation would produce an exalted action; and, as regards sensation, we see, in practice, not only a loss of feeling, but sometimes an exaltation or hyperæsthesia, or even a painful condition of the affected side. So, also, besides paralysis of motion, we observe, in some cases, convulsive movements of the limbs, both on the affected side and on the healthy one. In fact, a constant restlessness or jactitation is not infrequent, and may even be so marked that, as I have more than once seen, a case of apoplexy from effusion of blood may be mistaken for one of epilepsy.

It was long ago conceived that the corpus striatum rules especially over the lower extremity and the thalamus over the upper. With reference to this point, Bright was somewhat disposed to confirm the opinions of Serres, Foville and others, but I believe that clinical experience has not in any way tended to corroborate this view. It is probable that the fibres are so incorporated that only the most minute and the most perfectly isolated lesion could throw any light on the subject; and as such a lesion would not produce death, a rare accident alone could give us the opportunity of seeing this question tested. It might well be supposed that the power which we possess of moving each limb separately, shows that the mechanism for each must be distinct; and, on the other hand, the mutual connection of the centres by means of their commissures might be well conceived to be associated with the process by which the limbs act alternately and yet in unison, as is seen in the act of swinging the arms during walking. I have no doubt that there is in these centres a special piece of mechanism which would, for instance, explain the fact that birds belonging to some orders walk, like man, by alternate movements, while those of other orders hop with both legs at a time.

Cases of disease of the central ganglia.—It is scarcely necessary to cite cases exemplifying the ordinary phenomena of hemiplegia: the momentary dizziness, the complete restoration to consciousness which very soon follows, and the termination in more or less prolonged paralysis of one side; or, on the other hand, if the case is soon fatal, the gradual extinction of consciousness as the blood extends from the ganglia into the ventricle, and the conversion of the hemiplegia into a paralysis of the whole body, associated with stertor, &c., and very frequently with a contracted state of the pupils. This latter symptom is, I believe, of very bad portent, and most generally indicates an effusion of blood at the base of the brain. I have very frequently observed a slight arachnitis on the surface of the brain where there has been a large effusion of blood.

The following cases came under my care in the Clinical Ward; they may be taken as examples of the ordinary form of the complaint.

CASE 1. *Hemiplegia; no loss of consciousness; Bright's disease.*—A cook, æt. 52, whilst engaged at her occupation, was seized with a fit. She was bled and sent to the hospital. She was paralysed on her left side, and her face was slightly drawn. She retained her consciousness (which had never left her) until shortly before death. The right ventricle was found full of blood, which had proceeded from the right corpus striatum.

CASE 2. *Right hemiplegia and loss of speech; Bright's disease.*—A girl, æt. 23, said to have been previously in good health, was suddenly paralysed. She was perfectly hemiplegic on the right side, and could not utter a word; she could write on the slate with her left hand, so that her memory of words remained. She partially recovered and left the hospital, when, about a year afterwards, she was seized with a fit, and died in a few hours. This was found to be due to an effusion of blood, which proceeded from the site of old disease on the left side of the brain. The corpus striatum and thalamus were indented and of a gray colour, from an old apoplectic clot. This extended downwards and outwards immediately above the island of Reil. Kidneys extremely degenerated.

CASE 3. *Hemiplegia; no loss of consciousness; seat, the thalamus opticus.*—A man was seized with a fit in the street, and when brought in was found to have complete hemiplegia of the left side, including the face. He spoke thickly, but gave his name correctly and appeared quite sensible. Afterwards coma came on, with stertor; it was then supposed that the blood had burst into the ventricles. He lived fourteen hours. The post-mortem showed that the blood had proceeded from the right thalamus and burst into the ventricle. The surface of the brain was slightly greasy, as in the first stage of arachnitis.

CASE 4. *Hemiplegia; no loss of consciousness; corpus striatum.*—A man employed at the hospital suddenly fell whilst at work, and was immediately brought into the ward. His right side was paralysed; he could put out his tongue and by signs showed that he perfectly understood what was said, although he was not able to speak. I remarked that blood was effused into one of the central ganglia; but whether it would cease to flow or extend I could not say. He gradually fell into an unconscious state, and died in ten hours. The blood was found to proceed from the left corpus striatum, and had burst into the ventricle; the surface of the brain was greasy, as in recent arachnitis.

Many patients are admitted in this final stage when coma exists, and, unless a good history be given, it is then impossible to form a diagnosis as to the exact seat of the disease. Of this the following case is an example:

CASE 5. *Apoplexy; supposed drunkenness.*—A woman, æt. 34, was brought into my ward in the evening in a state of perfect insensibility, and soon died. She was a woman of bad character, and had been in a coffee-house in the Westminster Road, where in the morning she was found insensible. She was thought to be drunk, and the police were called in. Afterwards it was considered that she was drugged, and an emetic was given to her. A large rent was found in the corpus striatum, and the ventricles were full of blood.

It may be remarked that death occurs in a few hours when the blood is poured into the ventricles and flows to the base, but that those cases are much more protracted in which the blood extends into the hemisphere.

CASE 6. *Apoplexy; death in ten days.*—A servant was found lying insensible in her bedroom; when brought in a few hours afterwards she had stertor and paralysis of the left side. The right side was frequently moved. She partly recovered her consciousness, and the palsied side became somewhat rigid. She lived ten days. The right hemisphere contained a large clot of blood of a brownish colour; there was no blood in the ventricles.

Any lesion of these central parts will, of course, be productive of the same paralytic symptoms as are caused by effusion of blood, and, therefore, I need only quote one example of softening. I may here remark that we cannot connect the condition of the pupils with any definite lesion, for their state is very variable, and is liable to be influenced by very slight causes.

CASE 7. *Softening of corpus striatum; hemiplegia.*—This man two years before had received a blow over his nose, to which no immediate ill consequences were attached. For some months before his decease he had a discharge from his nose, and often felt giddy. Eleven days before admission he was found in bed asleep, and could not be roused to go to work. He got up, but soon after went to bed again, and sank into a half-conscious state. When first seen he appeared just to recognise his friends, and could just speak; eyelids half closed; left pupil more contracted than right; eyes directed to right side; left side of face fallen; left leg and arm motionless, and apparently paralysed; right hand constantly scratching the body. He sank into a state of coma and died three days after admission. On the day of his death both the pupils were contracted. After death the pupils were unequal, the left more dilated than the right. There was disease of the ethmoid bone; the anterior lobes were adherent to it; and the disease extended backwards on the right side to the corpus striatum, and along its lower part to the crus cerebri.

In cases of chronic softening, or wasting of the centres, there has been found a corresponding atrophy with rigidity of the limbs. In congenital contraction and paralysis of the limbs an almost complete destruction of the centres has several times been present.

CASE 8. *Atrophy of brain; contraction of limbs.*—A man, æt. 50, was admitted into the hospital in a fit, which was styled apoplectic. It was anticipated that the attack would be quickly fatal; but, instead of this, he gradually recovered, remaining paralysed on the right side. He was soon able to leave his bed and sit up; but there was no improvement in the limbs; he gradually became weaker both in body and mind, until, in course of time, he spent the whole day in his chair, having a vacant look and with his arm drawn up to his side. From the commencement of the attack he had never been able to speak, but he could put out his tongue. He afterwards lost the power of sitting up, and became confined to his bed, with his arm drawn across his chest and rigidly contracted. He continued in this vegetative state until his death, fifteen months after the attack. The autopsy showed that nearly the whole of the left hemisphere was destroyed, its place being taken by a bag of fluid, which was confined only by the pia mater and arachnoid, and which burst when touched. It contained at least half a pint of fluid like lime-water. The softening process had destroyed the left thalamus, nearly all the corpus striatum, and all the neighbouring convolutions towards the surface, but the fluid had not broken into the ventricles.

Some cases are recorded in the 'Pathological Transactions' of wasting and rigidity of the limbs, associated with an atrophy of the brain on the opposite side. One such case is recorded by Dr. Ogle. It is that of a man of advanced age, who had been paralysed on the right side since he was two years old. He dragged his right leg and had partially lost the use of the right arm; this was flexed, and the fingers resembled the claws of a bird. After his death there was found a large cyst occupying the corpus striatum and slightly encroaching on the thalamus also.

In affections of the cerebral centres themselves the paralysis is hemiplegic, by which I mean that it is a loss of power of the arm and leg, and, to a slight extent, of the face also. By disease situated lower down in the tract particular nerves are also implicated, and thus, besides the paralysis of the extremities, other special nervous symptoms exist. One of the best observed and most accurately recorded cases of disease of the crus cerebri has been brought by Dr. Weber before the Medical and Chirurgical Society. The patient was seized with hemiplegia, and, at the same time, had paralysis of the third nerve, shown by ptosis, dilated pupil, &c. The diagnosis that there was a clot in the crus cerebri turned out to be absolutely correct.

Cases of disease of the pons Varolii.—As regards this part of the brain, since the motor-sensory tract passes through it, a disease of one side of the pons Varolii must produce a hemiplegia, and a disease of its centre a total paralysis. I believe I have noticed that when disease has originated in this part there has been more impairment of the sense of feeling than in the form of hemiplegia already spoken of. If this be so it may be due simply to the greater concentration of the sensory fibres in this region. In disease of the pons there is not necessarily any loss of consciousness. Since numerous nerves arise from this part, the implication of these often affords evidence as to the part of the sensori-motor tract affected. In ordinary hemiplegia we have seen that there is merely a temporary inhibitory influence on the facial nerve centre; but in actual disease of the pons the nerve root may be involved, and thus, with the hemiplegia, there may be a decided facial paralysis on the same side. In such a case, according to Brown-Séquard, the upper part of the pons is affected. In other instances, however, paralysis of the face occurs on the side opposite to that of the limb, and he says that we may then infer that the lower part of the pons is the seat of disease: for it is believed that the fibres of the seventh nerve decussate in the pons, and that in the cases last supposed the centre of the nerve of the same side would escape, but the disease would involve the fibres of the nerve of the opposite side. I cannot, from actual observation, say that the lesion has been higher in the one case than the other, but it is evident that it must have differed in position in the two cases. I should, however, say that for many years past we have, at Guy's Hospital, been in the habit of referring hemiplegia in which there is marked facial paralysis to lesions of the pons Varolii. Quite lately I had a man in the ward who, on getting out of bed, found himself paralysed on the left side of the body, while at the same time the right side of the face was as completely affected. He spoke tolerably well, and had not the slightest mental trouble. He rapidly improved.

It might be expected that an affection of the root of the *portio dura* would implicate the *portio mollis* also; I have no doubt this is often the case. Hitherto examples of disease in the pons have not generally been very accurately noted, but the occurrence of sudden deafness in association with facial paralysis has been substantiated by Dr. Hughlings Jackson. In a speedily fatal case the presence of deafness on one side might not be remarked.

Disease of the pons may also affect other nerves, as, for instance, the fifth or the sixth. Cases of this kind have been related by Dr. Hughlings Jackson. In a child who was under my care with a tumour in the pons Varolii, there was, besides weakness of the limbs, paralysis of the seventh nerve of one side of the face and of the sixth nerve on the other side.

The late Mr. Barlow, of the Westminster Hospital, brought before the Pathological Society a case in which a clot in the pons had given rise to hemiplegia of the right side of the body with paralysis and *anæsthesia* of the left side of the face.

I have a man now in the hospital with partial hemiplegia and numbness of the left side of the body, in whom there is also loss of feeling on the left side of the face and in the eyeball and tongue. In this patient, too, the head is drawn backwards and to one side, and when an effort is made the two sides of the chest expand unequally.

By disease still lower down towards the medulla oblongata the fibres of the lingual and glosso-pharyngeal nerves are involved; and thus, again, special symptoms are produced. I have given three instances of such an affection. In the first case there was no lesion of the motor tract, in the second this was temporarily affected, in the third it was decidedly implicated.

Case 9. *Disease of pons; loss of speech.*—A lady fell in a so-called fit during dinner; she was taken up speechless and put to bed. She lay with her mouth open, and the saliva running from it, and she was unable to swallow or to speak. There appeared to be no paralysis of the limbs, and from her gestures and expression there was every reason to believe that she was perfectly sensible. She was soon able to leave her bed, and recovered her usual health, but she never lost the paralysis of the tongue and palate. She wrote down all her wants on a slate. She swallowed with difficulty, and the saliva was always flowing from her mouth; but she was able to walk three or four miles a day, and was accustomed to join in a game of cards. About two years after the first attack she had another apoplectic fit, in which she died. On post-mortem examination there was found to be a great amount of disease of the cerebral vessels; much blood, which had escaped from the pons, was effused at the base. Within the pons there was an old brownish cyst. The central ganglia were healthy.

Case 10. *Disease of pons; loss of speech.*—A woman of middle age, about twelve months before I saw her, suddenly became speechless; she gradually recovered from this attack, but during the subsequent four months her speech was thick and hesitating. She then had a fit, and on the doctor being called he found her hemiplegic on the left side, including the face. She had again entirely lost the power of speech. This never afterwards returned; but she rapidly recovered the use of her limbs, so that when I saw her, there was but the slightest suspicion of weakness of the left arm. She could walk out for two or three miles, but could not utter a word. She could not protrude her tongue nor open her mouth wide except by gaping, when it became thoroughly opened; so that she was obliged to be fed with a spoon. The saliva was constantly flowing from her mouth. Her principal trouble, besides the inability to speak and the flow of saliva, was a restlessness which she could not control. The right pupil was more dilated than the left. There was no affection of the hearing. Her husband said that, after the first attack, when the power of speech was returning, she called things by their wrong names, or altogether forgot the names until they were suggested to her.

Case 11. *Disease of pons; loss of speech; fatal choking.*—A woman, æt. 56, five weeks before admission into the hospital had a fit whilst getting out of bed, and had not been well since. She walked to the hospital, but could not speak. It was, however, observed that her right arm and leg were more feeble than those of the left side. She could not protrude her tongue; her sight and taste were good, and her mind was clear. She had some difficulty in swallowing, and liquids ran out of the right side of her mouth. Whilst at dinner a large piece of meat stuck in her throat and choked her. After death the brain was found shrunken, and contained spots of softening in various parts; some yellowish-brown ones were especially noted in the interior of the pons.

I have no recorded cases of disease confined to the medulla oblongata. All affections of this part require very accurate noting. In a remarkable case which occurred at Liverpool, of a man who had a complete severance of one side of the medulla through the restiform body, there was partial paralysis of motion of the arm and leg of that side, but no loss of sensation. The origins of the glosso-pharyngeal and pneumogastric nerves were involved, and he had complete inability to swallow and constant hiccough. He died suddenly after some hours.

We see, then, that disease of the pons or medulla oblongata, by affecting special nerves, is productive of a distinctive form of paralysis. As regards the pons itself, a disease on one side produces hemiplegia with or without some other local paralysis; a disease in the middle of the pons may involve both tracts and cause a complete loss of power of the whole body. A sudden effusion usually produces profound coma; but unconsciousness is not necessarily a concomitant of either acute or chronic disease of this part of the brain. Another symptom often observed in these cases is contraction of the pupils, just as we see this produced by effusion of blood at the base or into the ventricles. This state of the pupils has often led to the suspicion of opium poisoning. In many instances, in which the pons has been the seat of disease, sensation has been markedly affected, as evidenced by a feeling of coldness in the extremities; and, in one case, in which a tumour pressed on the pons, a sensation of numbness and various odd feelings over the body were experienced. If complete coma were present it would, of course, be impossible to say whether a loss of feeling was due to want of perception or to an actual lesion of the sensory tract. I have recorded several cases of apoplexy of the pons Varolii accompanied by profound coma and by contraction of the pupils.

CASE 12. *Apoplexy of pons Varolii.*—A man, æt. 41, was admitted completely insensible and unable to move a limb; there was no stertor, but the breathing was slow; pupils contracted. A clot of blood was found occupying the pons.

CASE 13. *Apoplexy of pons Varolii.*—A very similar case, in which the blood burst into the fourth and passed up into the lateral ventricle.

CASE 14. *Apoplexy of pons Varolii.*—A woman, whilst riding in an omnibus, suddenly fell insensible. When brought to the hospital, she was quite unconscious and motionless, except that some occasional involuntary twitchings were observed. Pupils much contracted; breathing stertorous, irregular, and catching. She died in twelve hours. The brain on removal appeared healthy, except that the pons looked bulky and soft. On a section a clot was found within it.

Case 15. *Apoplexy of pons.*—A woman, æt. 44, fell in a fit whilst in a shop, and was brought to the hospital. She was in a perfectly helpless and unconscious state; when her limbs were lifted they fell powerless; there was no rigidity. The expression of the face was gone; the mouth was open, with the saliva running out. Pupils minutely contracted. Breathing deep, but not stertorous nor attended by any noise. Pulse labouring and full. The urine was albuminous; and thus, apart from the history of the sudden attack, there was no reason why the case should not have been regarded as one of uræmia. The brain presented no abnormal appearance until the pons was cut across, when a clot was found within it. Kidneys small and granular.

In the journals there have been reported several cases in which the symptoms of apoplexy of the pons resembled those of opium poisoning. One great distinction between ordinary sanguineous apoplexy and poisoning is that in the latter the whole brain is involved, whereas in the former there is often paralysis of a particular part, indicating a local lesion. But when blood is effused into the centre of the pons this distinction does not hold, because the whole sensori-motor tract is involved.

The 'Transactions of the Pathological Society' contain such cases recorded by Dr. Bristowe; also one by Dr. Hare, in which opium poisoning was suspected. The woman was 60 years old, quite comatose, with no distortion of features; the breathing laborious, but not stertorous; the pupils contracted.

Mr. Nunneley, again, has given the case of a woman who was perfectly insensible; the countenance calm and natural, as though she was in a deep sleep. The breathing regular and natural; the pupils minutely contracted; both sides of body powerless; no sign of feeling when touched.

In partial disease of the pons, affecting one side, there is hemiplegia, often accompanied by paralysis of the face; but in such cases there is no necessary loss of consciousness.

Case 16. *Apoplexy of pons.*—A man, æt. 50, was seized with a fit, and brought to the hospital. He was conscious and could speak, although with hesitation. He was paralysed in the left arm and leg, and on the right side of the face. After death the brain was found to present no peculiar appearance until the pons was cut through, when a clot of blood was seen in it towards the right side.

Dr. Ogle has related a case in which there was a softened spot in the pons. The man had been seized with a fit, from which he soon recovered; but partial hemiplegia remained, there being loss of both power and feeling; the limbs were lax. He was conscious, but unable to articulate.

In another of Dr. Ogle's cases a cyst was found in the right side of the pons. There had been incomplete hemiplegia of the left side, with numbness and partial loss of sensation, and with paralysis of several of the nerves which pass through the pons Varolii. The mental powers were intact.

In Mr. Barlow's case of right hemiplegia from effusion into the pons, with complete anæsthesia, the consciousness and intelligence remained throughout the five days after the attack, and to within two or three hours of death.

Case 17. *Tumour pressing on pons; hemiplegia.*—A child had a tumour at the base of the brain, pressing on the left side of the pons and on the neighbouring parts. The first thing noticeable in the symptoms was a drawing of the head to one side; subsequently there was a partial paralysis of the right arm and leg, and vomiting was present. She at last lay coiled up in bed, not caring to move, and the paralysis became complete. The pupils were variable.

Paralysis in connection with disease of the surface.—In tracing the motor tract downwards from the corpus striatum through the pons to the pyramid, we see that paralysis is produced whatever part of this tract happens to be the seat of disease. But, we may ask, is the converse of this true? Is hemiplegia always due to an altered condition of some part of this tract? If we mean by paralysis an actual loss of the motor power of a limb, I should say that the proposition is true; but if we include under paralysis the inability to move a limb from want of volitional power, then the statement is probably not absolutely correct. The simplest case of the condition to which I refer is where a patient is perfectly unconscious, and his limbs fall senseless when raised; this might be the result either of simple coma taking away the consciousness or of a real paralysis produced by disease in the centres. Thus, as we have already said, the effect of a destruction of the motor tract (as in apoplexy of the pons) resembles the coma produced by opium. If we analyse the two states, we shall have to suppose that the want of volitional power associated with the loss of consciousness, and the failure of the will to influence the central ganglia, produce the same result as if these bodies themselves were diseased. Hence we might ask how would it be if one hemisphere only were diseased, the functions of the other side remaining perfect, and, perhaps, with them, consciousness on the part of the patient? Would the effect be seen in a paralysis of the side of the body opposite to that affected? I can give only one case which distinctly bears on this question; but this, if correctly reported, enables me to answer it. Cases are, indeed, not uncommon which may, perhaps, by some be considered sufficient to determine this point. These are instances in which an arachnitis on one side has existed, and in which a paralysis of the opposite side of the body has been observed. If this really be a fact—that a disease of the surface of the brain can produce paralysis—we shall have to distinguish different forms of the complaint. In the first place, there is the case of disease of the centres, in which some influence is evidently removed from the limbs, so that the muscles lose their tonicity. One of the best examples of this is where both facial muscles are paralysed and the usual expression is gone. But even in paralysis of a limb the same want of tone is observed; and thus this form of disease contrasts strongly with what is seen in the hysterical woman, who simply has not the will to move her limb, for in this case the form and nutrition of the part are preserved. In the one instance the attempt to move the leg is futile in spite of the greatest effort; in the other no attempt is made, no influence is exerted by the will upon the central ganglia which have the management of the mechanism. In this case the failure of action on the part of the hemisphere is functional; but supposing its cause were organic, would the result be the same?

The difficulty of solving the question has arisen mainly from the rarity of disease of one side, except as a result of accident. Thus, in those common cases of rupture of the middle meningeal artery leading to an effusion of blood, by which the brain is flattened, paralysis, if any has existed, has been explained by the pressure exerted on the central ganglia below. But may not this effect be really due to the compression of the surface?

So, again, with arachnitis; the only instance in which this is unilateral is where an injury has been received, and in very many of such cases the brain substance likewise has been deeply implicated. If an effusion of blood on the surface, or an arachnitis, can produce paralysis, such an affection would be distinguished from that in which the centres are at fault by the presence of coma, convulsion, or delirium, which would in the latter case be absent. But the truth of this is not absolutely certain, for an affection of the surface of one hemisphere only might, perhaps, be sufficient to produce a loss of consciousness. As my purpose is rather to furnish a clinical report, I shall not dwell upon this subject, and shall only say that, while I have seen many cases of unilateral arachnitis without paralysis, I have also witnessed its occurrence; but since, in most of these cases, there has also been an injury to the brain, I have frequently endeavoured to explain the presence of paralysis by an extension of disease to the centres.[1]

Some years ago there occurred at Guy's Hospital the following case, which, if the account of the post-mortem examination can be accurately relied upon, affords a most interesting and important example of the effects of disease of the superficial grey matter of the brain.

CASE 18. *Disease of cineritious surface of the brain; hemiplegia.*—A woman, æt. 36, struck her head against a beam of the ceiling several months before death; she subsequently became ill and had two or three attacks of vomiting. About three months before her death she became very ill, and took to her bed; she had febrile symptoms, sickness, &c., and was evidently suffering from cerebral disturbance. After the urgent symptoms had passed off she lay quiet in bed, and appeared quite sensible of what was said to her. She gradually became weaker in her limbs, and especially in those of the left side, until, in three weeks, her left side was quite paralysed. After this she lay quiet, apparently sensible, and able, although with difficulty, to answer questions and to put out her tongue. On being asked a question she would wait and deliberate, and then slowly answer. The most remarkable symptom was the fact that she never seemed aware that she had lost the power of the left side. On being asked about her arm she always declared she could move it, although it lay

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[1] It is only an act of justice to Mr. Jonathan Hutchinson to state that, since I have read some of his cases reported in the 'Med. Times and Gazette,' in which he speaks positively as to the connection of hemiplegia with arachnitis, I have regarded it as a fact, and have not endeavoured to explain it away in the manner mentioned. Should further observations corroborate the statement, it will be one of the most important points, not only in pathology, but also in physiology, in connection with the functions of the brain and the operations of the will.

I will take this opportunity of reiterating an oft-expressed opinion as to the disadvantages attending the study of special diseases. Without alluding to the grosser instances of persons who, from having taken an interest in the physiology of nervous or other diseases, have gained a special repute in the subject, and yet forgotten that embolism or Bright's disease may be productive of the symptom which they locate primarily in the brain, I will say even of the division of our art into medicine and surgery that, although it has its advantages, yet the fact of a most important affection of the brain being met with only in the surgical wards shows that the pure physician could never arrive at a full knowlege of the subject of nervous diseases. Pathology, as I have often said, knows nothing of the distinction between medicine and surgery.

helpless by her side. She gradually got weaker; mucus collected in her bronchial tubes, and thus she died.

The post-mortem examination showed nearly the whole of the surface of the right hemisphere of the cerebrum to be diseased; it was in a very peculiar condition, as if the whole cineritious substance were undergoing disintegration. This was not so apparent on the surface as on taking off a thin slice, when the cortical part of the brain was seen to be of a yellowish colour, worm-eaten and soft. The disease, in some places, affected the deeper layers more than the external. The disintegration reached the medullary matter, but did not appear to penetrate deeper. This morbid condition extended all over the right hemisphere with the exception of the base. The left hemisphere was quite healthy.

Here is the case of a woman who had disease of the surface of one hemisphere, and in whom there was associated with this hemiplegia of the opposite side. So far the case is comparable with that of paralysis from unilateral arachnitis; but in the latter case the inflammation is seldom confined entirely to one side, and it is necessarily accompanied by disturbance of the intellect. In the present case one hemisphere remained healthy and the consciousness was preserved, and thus the remarkable difference between such a case and one arising from disease of the centres. In the latter case the will is good to move the limbs, but the machinery is inefficient. In disease of the surface, as from compression, the consciousness is gone together with the will; but in the remarkable instance just related, where consciousness remained, the patient did not feel the inability, especially as the limbs were receiving their usual nervous influence. In disease of the centres the patient knows that he cannot move the limbs; he would like to do so, but feels he is unable; but in this case of disease of the surface the power to stimulate the apparatus was wanting; and yet, although consciousness remained, the woman did not feel the inability. Coleridge believed that the will could be separated from the understanding; for, alluding to his own melancholy addiction to opium, he says, "My case is a species of madness, only that it is a derangement, an utter impotence of the volition, and not of the intellectual faculties." More than one eminent writer has defined insanity as consisting in this want of will.

In the two following cases hemiplegia was observed to be associated with arachnitis.

CASE 19. *Injury to the head; unilateral arachnitis; hemiplegia.*—A boy had a blow on the head from a stone, causing a scalp wound. He had at first no symptoms, but subsequently became very ill, with great febrile disturbance, cerebral symptoms, &c. He afterwards fell into a semi-conscious state, and was found to be partially paralysed on the right side. He was trephined, but only a few drops of pus escaped. The post-mortem examination showed that there was no fracture, but that the bone at the seat of injury contained pus in the diploe. The left side of the brain was covered with purulent lymph, some of which was free on the surface, while some occupied the subarachnoid space; the cineritious substance also was infiltrated with inflammatory products of a pale yellowish colour.

Case 20. *Injury to the head; unilateral arachnitis; hemiplegia.*—A man fell from a loft several feet high on to a piece of wood, which produced a compound comminuted fracture on the right side, a piece of the bone being driven in. He at first suffered from concussion, but soon recovered; he remained in a doubtful state for a day or two, when arachnitis came on, and he died a week after the accident. Before death the right side was observed to be paralysed. The post-mortem examination showed a fracture, beneath which the brain was reduced to a pulp, and on the opposite side there was an arachnitis, the whole surface being covered with lymph.

In another case also the arachnitis was more severe in the side opposite to the injury, but in this instance no paralysis was noticeable.

I have said that a doubt has often arisen in my mind as to the arachnitis being a sufficient cause of the paralysis, because it was possible that a morbid process might have been set up by direct injury to the centres. In fact, in the three following cases disease of the central parts existed, and in the third of them the substance was involved, and yet no arachnitis existed.

Case 21. *Injury to the head; arachnitis; contusion of brain; paralysis.*—A young man, æt. 19, had a brick fall on his head, and died a week afterwards. The blow rendered him only partly unconscious, and from this he soon recovered. There was a scalp wound on the left side of the head. Two days after the injury he complained of pain in the right arm and leg, and especially in the latter. This limb was also very sensitive, and caused him actual pain when touched. On the following day sensation was partially lost, and the limbs were weak. This increased until he seemed to be completely paralysed on the right side. There was some distortion of the face, and there was also some difficulty in protruding the tongue. He afterwards fell into an unconscious state, and was trephined. No pus was found, but the dura mater was opened, when lymph was seen on the surface of the brain. An incision was made into the brain, but no abscess was discovered. The post-mortem examination showed acute arachnitis on both sides, but more especially on the side of the injury. A fracture was found, beneath which the brain was pulped, the medullary matter at this spot being red and soft. This condition extended downwards as far as the thalamus, but this body itself was healthy.

Case 22. *Injury to the head; arachnitis; contusion of brain; paralysis.*—A boy fell from a loft on to the top and right side of his head. He soon recovered his consciousness, and on admission was quite sensible. Some pieces of broken bone were removed. After a few days the brain came into view, and a large hernia cerebri was formed. He afterwards became very drowsy, and at length insensible. During the last twenty-four hours it was observed that the left side was paralysed, and the right occasionally convulsed. There was found general arachnitis of both sides, but more especially of the right. The softening of the brain at the injured part had extended to the outer surface of the thalamus.

CASE 23. *Injury to the head; contusion of brain; paralysis.*—A man had a brick fall on his head from the top of a house. He was stunned, and was brought to the hospital. He was able to walk to the hospital, but it appeared as if his left arm and leg were weaker than the others; right pupil contracted. Two days afterwards some pieces of bone and clot were removed, and it was thought that this led to a gain of power on the part of the limbs. For some days it was observed that he could move his leg, but that his arm was motionless. On the tenth day pulmonary symptoms appeared, and at the end of another week he died. During this time the dura mater sloughed, and a hernia cerebri appeared. The post-mortem examination showed that the man died of pyæmic pneumonia; there was no arachnitis. A section made vertically through the fungus and the hemisphere showed the medullary matter to be soft and mixed with blood. The change had extended down to the thalamus, in the middle of which a spot of red softening was found.

Paralysis from compression by blood or pus.—Having alluded to paralysis in connection with disease of the central ganglia and of the surface of the brain, it will be advisable to consider next that common class of cases in which an effusion of blood on the surface of the brain takes place, producing compression, and sometimes hemiplegia. In these cases, in which there is a fracture of the parietal bone with a wound of the middle meningeal artery, blood is often effused until a large clot is formed, which causes a depression on the side of the brain. If the pressure is excessive, stertor comes on, and the whole body is powerless; if it is less, a partial paralysis of the opposite side is produced, and what is very remarkable is that I have seen the paralysis without the loss of consciousness. From what has been already said, either of two conditions might be considered equal to its production—an implication of the superficial grey matter or pressure on the centres. There being, for the reasons already given, a difficulty in the first explanation, I have generally adopted the second, especially as great pressure has evidently been exerted, and as, in spite of this, the paralysis has not been complete. I think that the importance of endeavouring to arrive at a right conclusion is seen in the fact that such symptoms have suggested many successful operations. This is above all others the class of cases in which the surgeon trephines with a hope of success. So far as my experience goes, a partial hemiplegia, occurring soon after an injury, always points to a clot on the opposite side. At all events, I have observed only one case in which an injury to the brain substance has been productive of this symptom, and in this instance the surface was lacerated. It is true that I have sometimes seen hemiplegia result from a lesion of the central parts of the brain, but these cases are rare, and in them the hemiplegia is complete, and there may be no loss of consciousness.

The paralysis which comes on some days or weeks after an injury is altogether another matter, and is due to very different causes. We have seen that hemiplegia, coming on a short time after an injury, especially if associated with coma, is supposed to signify an effusion of blood. Now the appearance of such symptoms after some days is often thought to indicate the formation of an abscess, which, it is supposed, may, like the former, be relieved by the trephine. I do not wish to encroach on the province of the surgeon, but I can state that I have never seen an abscess in the situation in which a clot of blood is thrown out so as to be accessible to an operation. The unconsciousness which comes on in these cases is produced by inflammation of the surface of the brain; and hemiplegia, if present, is either due to the lesion of the brain having extended into the deeper parts or a symptom of arachnitis. If this be so we cannot but remark on the inutility of secondary trephining, and this theoretical objection is borne out by practice, for it has fallen to my lot to examine the bodies of many in whom the operation has been performed, and though I have made repeated inquiries, I have never been shown in the wards of the hospital any cases in which recovery has followed such an operation. The only cases I have seen in which it seemed possible that relief might have been afforded by secondary trephining have been one or two where a secondary hæmorrhage had occurred on the surface of the brain; but this condition was not suspected during life, and I apprehend that the expectation in cases of secondary trephining is that an abscess has formed in connection with the bone, and that this may be exposed; but such an event is certainly of the rarest possible occurrence. Failing to find this, many surgeons would proceed no further, whilst others would open the dura mater and even plunge a bistoury into the brain itself. The only case, however, in which any expectation of relief from this practice can be entertained is that of which I have spoken above, and such a case is almost an imaginary one. The only instance which has come before my notice, in which an operation was temporarily successful, is that of a man operated on by Mr. Sydney Jones, of St. Thomas's Hospital. In this case a sufficient quantity of matter had formed between the bone and dura mater to be removed with relief to the patient. In these remarks I, of course, allude only to cases resulting from injury, for I have seen an abscess within the skull arising from chronic disease of the bone. I should, from my own experience, say that unconsciousness coming on some days after an injury indicates arachnitis, and not an abscess, and that if hemiplegia also is present this must be regarded either as another effect of the inflammation or as an evidence that there is some lesion of the central parts. Hence the condition is, in neither case, such as could be remedied by operative measures.

Whilst writing down my own experience I have referred to two great authorities, Sir A. Cooper and Mr. Guthrie, and I must confess to some surprise at finding the positive manner in which they speak in condemnation of an operation which is at the present day so often performed. There may, however, be some recent argument unknown to me which can be suggested in its favour. Sir A. Cooper[1] says, "When pus is situated between the dura mater and skull trephining for its removal would be attended with complete success, but the chances of finding it there are against you, as it is generally situated between the pia mater and surface of the brain, for which an operation would prove worse than useless." "When an abscess has formed beneath the dura mater I have never seen a case recover from trephining for it, although that membrane has been opened for its discharge." And Guthrie[2]—"Inflammation of the dura mater, proceeding to suppuration or the formation of matter between it and the bone, appears to have been a much more common consequence of injuries of the head in the time of Dease and Pott than at present. I have rarely seen cases of the secondary tumour they have described, and

[1] 'Lectures on Surgery,' edited by Lee. [2] 'On Injuries of the Head,' p. 122.

on inquiring of the surgeons of the different hospitals in London who are on the Council of the College of Surgeons, consisting of what may be called the élite of the surgery of London, I find it is almost equally unknown to them."

I shall give the outline of a few cases bearing on the subject of compression from effusion of blood, the symptoms being, as a rule, unconsciousness, stertor, and incomplete paralysis.

In the three following cases the coma was complete as well as the paralysis, but there was every reason to believe that if the case had been seen at an earlier period a distinct though partial hemiplegia might have been noticed.

Case 24. *Compression; coma.*—A sailor was brought from on board ship at 9 o'clock in the morning, and died within an hour. It was afterwards learned that on the preceding night, between 11 and 12 o'clock, he had been struck on the head. He walked about after the injury, but subsequently fell into a state of stupor. A large clot of blood was found compressing the brain.

Case 25. *Compression; partial hemiplegia.*—A man, whilst driving a cart, was thrown out and pitched on his head. He got up and walked on for about half an hour, when, becoming confused, he went into a shop, where he was supposed to be intoxicated. He soon became insensible, and was brought to the hospital. He had a scalp wound on the left side, and had constant twitchings of the limbs; the pupils rather dilated; breathing stertorous. The right arm was more twitched than the left, which was thought to be paralysed. It was supposed that effusion of blood existed; but from the convulsive movements it was inferred that the brain might be otherwise injured, and thus no operation was performed. The post-mortem examination, however, showed that the brain was uninjured, and that there was a large clot of blood on the right side, causing a great depression of the brain.

Case 26. *Compression; temporary hemiplegia.*—A man, æt. 46, was admitted under my care in a state of perfect unconsciousness. It was said that, on the morning of admission, he went to work as usual in a brewery, and he was found lying at the foot of three steps which led from a room above. He was insensible and bleeding from the head. The history of his subsequent condition was contradictory, some saying that he remained perfectly insensible, and others that he spoke; it was also stated that he appeared to be paralysed on the left side. On inquiry about epilepsy it was said that he had had fits at long intervals. When he was brought in stertor, dilatation of pupils, &c., were present. The arms and legs, when raised, dropped helpless, there being no more marked weakness on one side than on the other. The arms were often stretched out and moved convulsively. My surgical colleague was sent for, who had the man's head shaved and carefully examined the cranium, but could find no fracture. He did not feel justified in operating, and the man died in a few hours. A slight fracture was found, which involved the right middle meningeal artery; there was a large clot of blood compressing the brain.

In some instances distinct hemiplegia exists, and in the first of the following cases there was also loss of sensation. In the second the paralysis was sufficiently well marked to determine the surgeon to operate, and the result of this was successful.

Case 27. *Ruptured meningeal artery; paralysis of motion and sensation.*—A man, æt. 30, had a fracture of the left parietal bone caused by a poker. This led to the effusion of a large clot of blood on the dura mater, compressing the brain. When admitted he was in an almost unconscious state, with contraction of the pupils (particularly of the right), and was very restless. Two days afterwards, though he had not spoken, it was thought that he had some degree of consciousness, and on examining his limbs it was considered that he had loss of motion and sensation in the right arm and leg, and particularly in the former. Two days after this he seemed to be roused out of his lethargic state, and appeared to feel in the limbs when they were touched. After this he again became comatose, and gradually sank, with dilated pupils, dying on the eighth day.

Case 28. *Ruptured meningeal artery; hemiplegia; trephining; recovery.*—Some years ago a man was admitted under Mr. Cock's care in an insensible condition, having fallen from a height. He gradually recovered from the concussion, and remained sensible for some time, but in the night he was found in a deep coma, with stertorous breathing and with insensible contracted pupils. The man seemed on the point of death, when Mr. Cock determined to trephine, being guided in the choice of locality by the fact that the left arm and leg were freely moved when they were pinched, whereas not the slightest motion could be excited in either of the limbs of the right side. A large clot of blood was removed; the stertor almost immediately ceased, and on the following day the man could move his right arm and leg freely. He shortly after resumed his work and remained well for thirteen years, except that he had some fits towards the close of his life. He at last died of apoplexy.

I have already alluded to convulsive movements as being produced by injury of the surface, and I shall shortly consider the same effect as the result of an irritation of the cineritious matter, producing an excitation of the centres. In the following case probably the motionless side was partially paralysed, whilst the sound side was convulsed.

Case 29. *Effusion of blood; compression; convulsive movements.*—A man fell from a height, and, besides receiving various injuries to the body, struck the right side of the head. After having his wounds dressed he was allowed to go home, but was requested to return if any fresh symptoms appeared. This was in the afternoon, and in the evening, about four hours afterwards, he was brought back to the hospital insensible. The right pupil was dilated, the left contracted; both insensible to light. Breathing quick and stertorous. Occasional spasms of the whole body, but more especially of the right side. An exploratory incision was made, but no fracture was found. On the following day there were frequent paroxysms of spasms affecting the right side, and the respiration was difficult, the chest scarcely moving. On the following day he was better, and the stertor had passed off. In the evening, however, the breathing was more embarrassed, and mucus was collecting in the tubes. There was found a fracture through the right parietal bone, and a large clot of blood pressing on the brain.

CASE 30. *Effusion of blood; compression; convulsive movements.*—A man received a blow from a heavy hammer, which fell on the left side of his head. He came to the hospital to have the scalp wound dressed, not suffering any bad symptom. A fracture was, however, detected, and he was put to bed. He then had no brain symptoms. After five hours he fell into an unconscious state. The left arm was then in constant movement, whilst the right was rigid. One pupil was contracted, the other dilated. He was trephined over the seat of fracture, and some blood escaped, but he died twelve hours after the accident. A large clot of blood was found external to the dura mater on the left side. The brain was uninjured.

The following cases should be recorded as bearing upon the question as to the causes of paralysis. In similar instances the pressure of the blood has been regarded as a sufficient explanation; but then, as might be expected, the patient was perfectly unconscious. If the explanation holds good, the fact that consciousness remained must be attributed to the function of the other half of the brain remaining intact.

CASE 31. *Ruptured meningeal artery; effusion of blood; paralysis; no loss of consciousness.*—A man was taken out of an area, apparently in a state of intoxication, and was put into the police station; but not having thoroughly recovered himself, he was on the following morning brought to the hospital. No fracture could be detected, but there was bleeding from the ear. The left arm appeared weak and almost helpless. This partial hemiplegia remained, although he recovered his consciousness. He died ten days afterwards. A fracture of the base was found, and external to the dura mater there lay a large clot of blood, which had indented the brain into a large hollow. Outside the clot there was some fluid blood, which appeared to have been more recently effused, and to have been the immediate cause of death.

CASE 32. *Ruptured meningeal artery; effusion of blood; paralysis; absence of coma.*—A man fell from a plank, striking his head on a heap of bricks. He was brought in with incomplete paralysis of the right side. He was not unconscious, but was in a lethargic state, and when spoken to was very irritable. He passed his evacuations voluntarily. There was found a fracture of the skull on the left side, and a large clot of blood lay external to the dura mater, compressing the brain.

In considering how paralysis is produced after injury we have, then, to deal with two classes of cases. Sometimes it occurs speedily and in connection with effusion of blood. In other instances it is a secondary symptom, and is often associated with arachnitis, although in many of these cases (as I have already shown) the disease set up in the cerebral substance has extended down to the central ganglia. In the cases belonging to each of these categories two opinions as to the cause of the paralysis might be given; some persons would say that the centres of motion are actually reached by disease or compressed, others that the implication of the surface by inflammation or by the pressure of blood is sufficient to produce the paralysis.

It might be asked, what, in this respect, are the effects of direct injury to the surface? As a rule, convulsions and analogous symptoms occur in these cases, but paralysis has occasionally been observed. It might be suggested that in the following instance the central ganglia were excited to over-action, causing the rigidity which afterwards, as in epilepsy, terminated in loss of power. The case was that of a man who received a fracture of the skull, and had his brain lacerated. He had convulsions, after which the left arm and leg became rigid, and in three or four days they were paralysed. The brain on the right side was found to have been torn to some depth, but the central ganglia were apparently uninjured. With reference to this point I may again mention the case already alluded to, in which a man, who had been injured by a brick falling on his head, was affected with partial paralysis of the right arm and leg; a fungus cerebri formed, and softening extended into the substance of the brain as far as the thalamus, but there was no proof that this body itself was involved.

Injury to the central parts of the brain from violence.—In discussing the question as to the connection of symptoms with certain definite lesions, it is important to know what changes may be found in the brain as a result of disease and violence respectively. I will, therefore, refer to a few cases in which some very remarkable conditions were found. I believe it to be a rule which generally holds good that injuries to the head involve the external parts of the brain, so that the effects are clearly seen when the organ is exposed; and that a lesion affecting the internal parts, without any sign of external injury, points to disease. If, however, the latter condition should be complicated with some outward signs of violence—produced, for instance, by a fall—the inquiry becomes more difficult. The opposite case, too,—that in which an effusion of blood on the surface is associated with marks of external injury—may give rise to considerable difficulty.

I have already alluded to cases of meningeal apoplexy,[1]

[1] 'Guy's Hospital Reports,' series 3, vol. v, p. 119.

remarking on the difficulty which sometimes exists in determining whether the rupture of the blood-vessel has been spontaneous or due to violence; but now we will inquire whether a blow can seriously injure a central part of the brain, and, further, whether it is possible that this should occur without the external parts being wounded. This question is, from a medico-legal point of view, of the highest importance.

In the following three cases, in which a fracture of the skull existed, there could be no doubt that the internal lesions were due to injury. In two of them the septum lucidum was torn; in one the mischief was exactly of the same kind and occupied the same locality as that observed in disease.

Case 33. *Fracture of skull; injury to corpus striatum.*—A man fell on to his head from a height of several feet. When brought in he was a little confused, but had no other symptoms. In a few hours coma came on, with stertor and paralysis of the left side of the body. He was trephined, but no blood was found, although the dura mater was incised. He died on the following day. There was found a fracture through the occipital bone, and the brain beneath was bruised, as were also the anterior lobes. The ventricles were full of blood, which had proceeded from a rent in the corpus striatum. The arteries were much diseased.

Case 34. *Fracture of skull; laceration of septum lucidum; old disease.*—A man, æt. 59, fell from a ladder and struck the ground with his head. He was taken up insensible, and remained with symptoms of concussion for twelve hours; stertor then came on, and he died. The skull was found fractured, and blood was extravasated both outside and beneath the dura mater. The ventricles contained a bloody fluid, and the septum lucidum was torn through. On the surface of the ventricle there was some old discoloration, and the interior was granular, so that a morbid state had previously existed.

Case 35. *Fracture of skull; laceration of septum lucidum.*—A man, æt. 37, was knocked down by an engine. He was admitted with concussion under Mr. Hilton. In a few days he regained his consciousness, but remained in a very confused state. At the end of a week febrile symptoms came on, attended with delirium; he afterwards suffered from cough, and gradually wasted away, dying at the end of three weeks from sloughing pneumonia. The post-mortem examination showed that death arose from the condition of the lungs. There was a fracture through the floor of the skull at the front part. There was only the slightest evidence of contusion. The ventricles showed ecchymosis, and the septum lucidum was torn longitudinally, the rent being three quarters of an inch in length. (See Prep.)

Injury to the surface occasionally, though rarely, extends directly to the interior; thus, in one of the cases before me, a man fell on to the top of his head, pulping the under surface of the brain to such an extent that the bruising reached down to the ventricle. In the following case, in which a similar condition was found, there was no history to guide to a right conclusion as to its nature.

CASE 36. *Fracture of skull; contusion extending to the interior of the brain.*—A man, æt. 54, was brought into the hospital insensible, and soon died; no especial paralysis was discovered. It was said that he had gone to bed the night before intoxicated, and that, being found insensible in the morning, he was brought to the hospital. On removing the dura mater we found a large quantity of blood, proceeding from the anterior lobe on the right side. This part of the brain was quite soft, and the same condition extended back as far as the external part of the corpus striatum. It was a question whether this arose from effusion of blood making its way outwards, or was a softening produced by injury and extending from without inwards. It was not until a slight fissure of the skull was found that the latter opinion was adopted as the correct one.

In the three following cases the central ganglia were the seat of injury; but it is remarkable that in the first two instances there was evidence of old disease, and that there was no trace of contusion on the external surface of the brain. In the third case, although there was some account of a blow, the condition found so exactly resembled the result of spontaneous disease that I had no hesitation in giving instructions that at the inquest death should be ascribed to this cause.

CASE 37. *Injury to head; contusion of interior of brain; old disease.*—A man, æt. 34, whilst drunk, fell a height of twenty-six feet, striking the back of his head. On admission, four hours afterwards, he was quite insensible. He lived only eight hours. No fracture could be discovered. On post-mortem examination there was no evidence of injury in the shape of fracture or contusion. The left ventricle contained bloody fluid, which flowed from the left corpus striatum. The right corpus striatum and thalamus were also ecchymosed on the surface, and contained blood within them. There was also a small old serous cyst.

CASE 38. *Sanguineous effusion in interior of brain; history of blow.*—A man, æt. 55, was found lying insensible near a crane, the handle of which had evidently struck him on the head, for there was a scalp wound. He had hemiplegia of the right side, including the face. He recovered his consciousness, and attempted to speak. He lived ten days. It was afterwards learned that he had not been well for some time, and had complained of weakness of the right arm. Excepting the scalp wound, no injury to the head or brain could be discovered. The left thalamus and corpus striatum contained blood; at the anterior part of the corpus striatum was a cyst passing into the anterior lobe.

CASE 39. *Sanguineous effusion in interior of brain; history of a blow.*—A man was brought into the hospital one morning in a state of perfect insensibility. His friends said that on the previous evening he had returned from his work, complaining of his head. He then stated that, whilst engaged in lifting a package from a cart, he had been struck on the head by the hook which hung from the chain. After giving this account, he became drowsy, and at last insensible; in this state he remained during the night, it being also observed that he was quite helpless on the left side. The subject of trephining was discussed, but, there being no signs of injury, this operation was not performed. The post-mortem examination showed a large clot of blood occupying the right hemisphere, and extending into the lateral ventricle, the corpus striatum being torn through. Not the slightest trace of injury to the head, skull, or brain, could be discovered.

I have, in my books, notes of several other patients who were insensible when brought in, and in whom a clot of blood was found within the interior of the brain. In these cases a decision was given in favour of spontaneous apoplexy, even though a scalp wound existed, and the patient had fallen down. Some of the preceding cases, however, show that no positive rule, with respect to the causes of lesions of the internal parts, can be laid down, for even in cases of injury the centre of the brain may occasionally receive all the violence of the shock.

Extension of disease from the brain to the cord, and vice versâ.—I will, for a moment, diverge from my course to remark that several of the cases now before me suggest the consideration that probably many erroneous impressions as to the cause of particular nervous symptoms arise from the fact that the post-mortem examinations were formerly imperfect, and from its not being known that disease of the brain may extend to the spine, and *vice versâ.* In former times it was probably not taught that the ventricles of the brain communicate with the subarachnoid space of the brain and spinal cord; at least I am under the impression that the demonstration of the fact not many years ago by Mr. Hilton was quite original. I may say, then, that an inflammation at the base of the brain is constantly associated with a similar affection of the ventricles, and, again, that an inflammation of the spinal membranes may pass up to the base and even to the interior of the brain. Thus, in a person suffering from spine disease, and whose head was not examined after death, cerebral symptoms would very likely be attributed to the spinal affection; indeed, I know that this has occurred. On the other hand, after an injury to the head an examination of the spine is often omitted, and thus all the symptoms are ascribed to the primary lesion, whereas there may have been, in addition, an inflammation of the contents of the spinal canal, extending down to the cauda equina. The same thing may be said of effusions of blood, which may pass from the ventricles downwards, or from the surface of the brain either downwards around the spinal cord or upwards into the ventricles.

I could quote numerous examples of these facts, but a few will suffice.

Cases.—A man was struck on the nose by a piece of timber, which fractured the ethmoid bone, &c. This accident caused his death in three days. An arachnitis was found, passing along the base, extending up into the ventricles, and down the spinal canal to the dorsal region.

In a case of tubercular meningitis lymph was found to extend down the spine as far as the dorsal region.

A man had a compound fracture of the skull, injuring the brain; an inflammatory softening took place, which reached the ventricle, and there set up an inflammation. This subsequently extended downwards through the third and fourth ventricles to the spinal canal, so that lymph was found enveloping the cauda equina. Thus, a blow on the head gave rise to an inflammation, which reached to the sacrum.

A boy had a blow on his back, injuring the spine; an inflammation of the membranes took place, which extended up to the brain, producing an arachnitis, of which he died.

A young man, long ill with caries of the vertebræ and lumbar abscess, at last died from the extension of a spinal meningitis to the brain.

A woman, with a bed-sore after fever, suddenly became very ill, and died in a few days. The spinal cord had become implicated in the disease of the sacrum; the canal was laid open, and an inflammation was set up which extended to the brain.

In a case which recently occurred, and in which blood was effused within the cranium from an aneurism, the fluid passed downwards in the membranes of the spinal cord.

These examples show the necessity of a thorough examination of the cerebro-spinal centres before drawing any conclusions concerning the value of a particular symptom.

Compression of the brain by depressed bone.—There is one subject which I cannot discuss, because I am deficient in well-attested facts which bear upon it. I allude to the effects on the brain of circumscribed pressure, such as would be produced by a piece of depressed bone. In the commonest class of cases, those in which the skull is comminuted, and pieces of bone are driven in, there is, at the same time, severe injury to the brain itself, and it is difficult to decide how many of the symptoms ought to be attributed to concussion, compression, or actual laceration of this organ; and, moreover, the fragments of bone are removed before their direct influence can be determined. The effects of depressed bone would be best seen in cases in which recovery from the first shock had occurred; but, on inquiring of my surgical friends, I find that they cannot provide me with any cases such as are recorded by some of the older surgeons, in which insensibility or paralysis arose from the compression of the brain by bone, and in which these symptoms departed as soon as the bone was removed. I think these cases require further elucidation by the examination of fresh instances. There is, indeed, the oft-repeated case related by Sir A. Cooper, of the sailor who was trephined by Mr. Cline for depressed bone, and who immediately recovered the consciousness which he had lost for thirteen months.

Mr. Guthrie mentions some remarkable instances of the symptoms produced by depressed bone, and of their departure after its elevation. One is the case of a girl who had a stone fall on her head, producing insensibility; she also gradually lost the power of the right arm and leg, so that, in the course of some months, she was paralytic. Besides other symptoms, the hearing and eyesight also were affected, and the memory was bad. She was in this condition when Mr. Guthrie trephined, removing a healthy piece of bone, and in three days the paralysis had disappeared, and the sight and hearing had returned. She left the hospital recovered. I am at a loss for any explanation of the cause of the symptoms or the result of the treatment.

The cases in which the trephine has been used at an earlier period are not so far removed from some possible explanation, although even these are somewhat obscure.

For instance, there is the case of the soldier who, at the battle of Waterloo, was struck by a piece of shell over the left parietal bone. He remained insensible for half an hour, when he found himself unable to move the right arm or leg. Four days later he was trephined, and some bone was removed; immediately afterwards he recovered in part the use of his limbs, and he gradually got well, except that some weakness of the arm remained.

There is also the case of a man who, at the same battle, was struck by a shot, which fractured and depressed portions of both parietal bones. When he recovered his senses he found be had lost the use of both legs, and was benumbed from the loins and lower part of the chest downwards. Ten days afterwards he was trephined, and the use of the legs returned, though he was not able to walk for some weeks.

Disease of the cineritious substance.—Although there may be a difference of opinion as to whether disease of the surface of the brain produces paralysis, there can be no doubt that this portion of the brain is intimately connected with the intellectual operations, so that disease of any kind affecting it produces mental disturbance. In acute disease we witness delirium, which ends in coma; in chronic disease we observe an equally certain though slower dissipation of the intellectual faculties. It is a fact, also, that injury or disease of the surface is productive of convulsive movements. After an injury such symptoms generally indicate contusion or laceration of the surface. In the course of disease continued convulsion is often the effect of acute change, whilst an occasional convulsion is due to chronic change; but this is by no means necessarily the case.

Having now sufficiently considered the spinal cord and its prolongations upwards, known as the cerebral ganglia, we pass on to the brain proper, which is associated with the intellectual powers, the will, and the consciousness. If the brain be violently shaken, all power of movement or feeling is gone; and we also observe minor degrees of this commotion resembling those which are met with in patients recovering from the severest form of concussion. In these cases we constantly have an opportunity of seeing that the whole nervous mechanism may be at work, and yet that all mental power and even consciousness may have disappeared. Sir A. Cooper mentions the case of a gentleman who received a blow on the head. Although this patient could say nothing, and was apparently unconscious, he nevertheless got out of bed, bolted the door, made water just as if he had been in perfect health, and returned to bed again; and yet when the door had to be broken open the noise did not rouse him. Sir A. Cooper, too, gives another case of a gentleman who, although suffering from concussion, attempted to shave himself.

If the function of the hemispheres is in such cases completely in abeyance, we can suppose that the same automatic movements might be performed even were these parts altogether absent. While speaking of concussion, I may allude to the fact that I have seen cases in which, from the effects of a blow, patients have become violent and almost maniacal; and, just as the perfect insensibility of concussion has been mistaken for drunkenness, so also have screaming and violence, which have really been the immediate effects of injury.

With reference to affections of the cineritious substance of the brain, it might be imagined that meningitis would, above all others, be the disease in which this substance would be involved, seeing how intimate is the connection between the pia mater and the convolutions. It may, therefore, be well to explain the meaning of the terms *meningitis* and *arachnitis*. The serous cavity of the arachnoid being looked upon as analogous to that of other serous sacs, the word arachnitis has been used in the same way as pericarditis, peritonitis, &c. But it has been observed that the inflammatory products are often confined to the meshes of the pia mater, and thus the term *meningitis* has come into use as a more general expression, and one less strictly defined. Any little difficulty which may be felt by students is, I believe, easily removed by the simple explanation that the surface of the brain is convoluted instead of being smooth, and that, therefore, the inflammatory exudation finds room between the organ and its enveloping membrane. Hence, the case is different from that of an inflammation of the surface of an organ like the lung, where the lymph necessarily transudes. It should, however, be remembered that serous cavities are formed by two membranes, a visceral and a parietal. Now, either of these may be primarily inflamed; but since the disease soon involves them both, the distinction is overlooked. The cause of the inflammation in the two cases is, however, different, and consequently the pathology of the affection may likewise vary widely. For instance, we say that pleurisy is an inflammation of the serous membrane of the chest, but that it may affect either the parietal or the pulmonary membrane. Now, in a case of injury to the chest it would be parietal, but when associated with inflammation of the lung it would be pulmonary. It might very often be important to distinguish between these forms of disease, but this is impossible. Suppose, however, that the lung surface were irregular, and the pleura tightly stretched over it; if this were so, it is clear that in cases of inflammation primarily affecting the pulmonary membrane the lymph would be found between the lung and the investing membrane, whereas in a pleurisy originally parietal the inflammatory product would be within the serous sac. Just so is it with the brain. If the lymph is found free in the arachnoid cavity, we know that the source of the inflammation is the parietal arachnoid or the dura mater; whereas, if the lymph is beneath the visceral arachnoid, the source of the inflammation is the pia mater or the brain itself. Thus, the idiopathic meningitis is subarachnoid, whilst that which arises in connection with the dura mater and bone is interarachnoid. Herein lies the explanation of the statements of the older French writers as to the rarity of idiopathic arachnitis. The practical consequence of the distinction is that simple arachnitis (the affection in which the exudation is found in the arachnoid space) must be regarded as having its origin in the bone without, and that therefore disease or injury of the cranium must be looked for. An idiopathic meningitis shows itself by an exudation which is altogether subarachnoid, or by the presence of a comparatively small amount of transuded lymph. The latter form may, however, be set up by an injury to the brain; and thus it is not uncommon to find that both the subarachnoid inflammation and the interarachnoid exist in the same case. One consequence of this is that in idiopathic inflammation the whole surface is affected, whereas after injury it often happens that one hemisphere alone is covered with lymph. A unilateral arachnitis, then, is met with only after injury; and, as I have already remarked, this is one reason why there is a difficulty in tracing the consequences of such an affection. Another result which probably follows from the fact that the disease arises in the one case from without and in the other from within, in connection with the pia mater and cineritious surface of the brain, is, that the symptoms associated with the former affection may be not nearly so severe as those arising from the latter. This would afford an explanation of the surprise which the surgeon sometimes evinces when he sees a quantity of purulent matter covering the brain in a case which has presented very few symptoms. I believe, too, that the fact may be accounted for by the inflammation being confined to the surface, whereas in idiopathic inflammation the whole substance is often involved. It is clear, then, that it is especially in the idiopathic form of meningitis that we must look for disturbance of the function of the grey substance of the brain, for in any affection of the pia mater this must necessarily suffer. In fact, we find that in these cases the cineritious matter is appreciably softened and adherent to the membranes, and that it often contains inflammatory products. In some instances there may even be a marked discoloration between the various layers, and, by pinching up a portion of the brain substance, one layer may be separated from another. Indeed, it is probable that in idiopathic meningitis the whole brain suffers; it is certain that the ventricles are involved, for lymph may be found within them, and the central parts are softened. Since, then, the surface without is affected as well as the surface within, and since the brain substance beneath each of these surfaces is involved to some depth, it is clear that the disease known as meningitis would be more correctly termed *meningo-cerebritis.*

Hence, as regards its symptoms, inflammation of the membranes of the brain is in reality a more complex condition than we should at first have supposed. We might imagine that in such a disease both the mental and bodily powers must rapidly fail; that the surface being involved, delirium and convulsions would result, and that these cases would end in coma. This termination is, in fact, accelerated by the production within the ventricles of fluid which, by its pressure alone, would tend to produce coma. The exceptional cases of chronic hydrocephalus, in which no symptoms exist, are just those in which the pressure is relieved by the expansion of the bones of the skull. I have always thought that the hemiplegia which sometimes occurs at the termination of a case of hydrocephalus is to be ascribed to the softening of the central parts of the brain; and I have, in some cases at least, considered the fact that one side is affected more than the other to be fairly attributable simply to the position of the patient. In these cases, too, the base of the brain being especially implicated in the effusion, the cranial nerves suffer, and thus we meet with various conditions of the pupil. Amaurosis is so common in many affections of the true brain, or of the cerebellum, that I will not venture to conjecture its immediate cause. It may be remembered that the optic tract is of considerable length, and that the vascular supply to the retina is liable to be affected by tumours or other diseases situated at a distance.

The fourth ventricle and the neighbouring parts are also involved in most cases of meningitis, and the pneumogastric and other nerves arising in this region are consequently implicated. It may, perhaps, be by the irritation of this nerve that the respiration and the heart's action, as well as the stomach, are affected. A sighing or irregular respiration is well known to be a brain symptom, and, at the same time, the heart is "slowed" (to use the engine-drivers' term), and the stomach ejects its contents.

The symptoms, then, due to implication of the cineritious substance of the brain are sufficiently well distinguished from those caused by disease of the central parts. In the latter case paralysis is the chief effect, while in the former we observe convulsions and an affection of the intellectual powers.

By chronic changes affecting the cineritious substance analogous symptoms would be produced, although more slowly. If this part of the brain should be especially affected, the mind will suffer; but, just as the whole brain is involved in the acute diseases, so is it also in those which are chronic. The effect of a chronic change in the brain is, in very many instances, an atrophy; and thus this condition has been found in a great variety of disorders, which have all tended to the same end. Hence, as might be supposed, the symptoms have been of a very similar kind in all these cases. I have elsewhere[1] entered somewhat fully into this subject, alluding to the atrophy which is found in old age, in dementia, in alcoholismus, in lead poisoning, &c. In such cases of atrophy of the brain as a whole it might be expected that there would be a failure of both the physical and the mental powers. This is seen in the dotage of the old man, who, sinking into second childishness, totters as he walks, falters as he writes, and by his garrulity and foolish talk evinces his bodily and mental condition; the very thoughtlessness and mirth of childhood often again assume their sway, and the aged sire may thus be the happiest and merriest of the family circle.

The brain, under these conditions, becomes actually smaller than it was before; it has shrunken, the sulci between the convolutions are deep and are filled with water, and the arachnoid over them is thickened and opaque. The shrinking is partly compensated for by effusion into the ventricles, and the choroid plexus contains cysts and earthy matter.

The same changes occur in those who become prematurely old, who live debauched lives, and, becoming haggard in body and worn out in mind, early "run to seed." This is especially

[1] 'Journal of Mental Science,' October, 1864.

seen in those who are intemperate in drinking, for in such persons, as the body degenerates, the brain shrinks.

That the latter organ has suffered is shown by the tottering gait, the tremor of the hands, the vacant look, and the hesitating speech. Whether the brain affection is a simple atrophy, or an inflammatory change, need not be discussed here, especially as it would open up a large question which also arises with reference to the analogous changes in other parts. This is the case, for instance, with the kidney of Bright's disease, where both the parenchyma and the vessels undergo a change, which some would call inflammatory, but of which the ultimate result is that the tissue is degenerated.

In like manner, the thickening of the membranes on the external surface of the brain, and the granular condition of the ventricles on the internal surface, might suggest that the change is of an inflammatory nature. But whether this is so or not, the result is the same.

Mercury and lead appear to produce a similar condition, and to give rise to the same symptoms as are observed in cases of dementia, paralysis, epilepsy, &c. The brains of demented persons who die in asylums present a wasted appearance quite like that just described. The reports of such institutions sometimes seem to imply that this form of atrophy is peculiar to such persons; but in its general features, at any rate, it is not distinguishable from that seen in the brains of old people or of those who have died of delirium tremens.

But although the appearances observed in the brain after death do not evidently differ in these cases, it is very possible that the early conditions of the organ may not be the same, and that the accompanying symptoms may, in these different affections, present peculiarities of their own.

In illustration, I may refer to the diseases of the kidney; any one of these may end in that stage in which the functions of the organ are simply lost; but during their progress these various diseases differ sufficiently to enable us to distinguish one of them from another.

So also, for example, in the disease known as the general paralysis of the insane (dementia paralytica or paralysie ambitieuse) the whole brain, no doubt, eventually suffers, the powers, both of body and mind, being completely lost. This result is due to a peculiar inflammatory process in the cineritious matter, originating sometimes in purely mental or moral causes, but probably often set up by injuries.

It has been most satisfactorily determined that, in this disease, a very definite morbid process takes place in the cineritious substance.

Salamon, Wedl, and others, have drawn attention to the production of connective tissue in this affection, and to the alterations in the blood-vessels, which become thickened and varicose. Dr. Sankey has confirmed these statements, although not in every particular. In a case of general paralysis of which I published an account the cineritious substance had undergone an important degenerative change, and the ganglionic cells were much altered in form and colour, and apparently contained earthy matter. The superficial grey matter was also full of large amyloid bodies, and the blood-vessels (although the patient was comparatively young) had undergone a most remarkable degeneration. The smaller ones were more rigid than any I had ever seen, standing out from the cut surface of the brain like so many bristles from a brush. In this disease the pia mater is involved; indeed, the membranes are often closely incorporated with the cineritious substance. Such adhesions are in themselves a sufficient proof that the brain structure has suffered. Hence the term peri-encephalitis or meningo-encephalitis has been used to express the pathological condition which exists in these cases, and the disease has been regarded as standing in the same relation to acute meningo-encephalitis or cerebritis as other chronic to the corresponding acute affections; the same relation, in fact, which an acute synovitis of a joint, going on to involve all the tissues of the part, bears to a chronic rheumatic arthritis affecting all the same structures. The general paralysis of the insane is, then, a disease which has an appreciable morbid anatomy; and, in consequence, when cerebral diseases are classed on a pathological basis, it comes under the domain of the ordinary physician; but since the mind suffers in a more chronic manner than in most of the other affections which are seen by such practitioners, this complaint is, in practice, treated chiefly by the alienist.

There are, however, many reasons for drawing a line between this and other mental affections. Thus, it sometimes arises from a definite cause, such as an injury, in a person not predisposed to insanity; it runs a certain course of not many years' duration, and it may attack a brain previously sound. On the other hand, purely mental diseases are generally dynamic or functional; they depend upon some natural and inherent failing, and they show themselves by peculiarities of manner, habits, and feelings. We therefore must not infer, from the supposed analogy between general paralysis and the various forms of insanity, that they also arise from a structural alteration similar to that which exists in this disease.

I have said that the result of the changes which occur in peri-encephalitis or general paralysis is a destruction of the tissues and a corresponding loss of function, but that it is very probable that the peculiar symptoms which accompany this special form of disease are characteristic of it.

This, however, raises questions which I should like to have more distinctly answered by alienists. Do they rely on the symptoms which accompany the disease, as characteristic of it? Or do they, on the contrary, consider that the only cases which deserve the name of general paralysis are those in which they believe that there is an affection of the cineritious substance, such as I have spoken of above? Would such a condition, in the absence of many of the usual symptoms, be sufficient to constitute the disease? Are a certain number of the symptoms sufficient to mark it, even though one or two of those usually regarded as most characteristic should be absent?

The symptoms of this disease, as detailed by writers, appear to consist rather in a general failure of nervous power than in any special affection. To this, however, there is an exception in the peculiar form of mental aberration, tending to an exaltation of the ideas. But the presence of this is not absolutely constant.

Authors speak of inequality of the pupils, and of the progressive paralytic symptoms, shown by a peculiar gait, a quivering of the lips, a trembling of the tongue, and an alteration in the speech, resembling that due to intoxication. Now what I wish to know is, whether such symptoms can be distinguished from similar ones arising from a disease of the brian brought about by intemperance or by other causes. It is quite true that, in many cases, there are circumstances which will enable the physician to distinguish general paralysis from other conditions; but these circumstances are, for the most part, extraneous to the brain disease, and have reference to the habits of the patient. Thus, there may be marked hepatic symptoms and a history of intemperance; and these facts, and the sottish look of the individual, would contrast strongly with the absence of such symptoms and history in patients affected with general paralysis. But every one must have seen instances of general paralysis among men who have led very intemperate lives; and, in such cases, it may require considerable acumen on the part of the doctor to discriminate between the two affections.

In desiring more information as to the peculiar symptoms which characterise the various forms of atrophy, I am indicating the belief that they are, as a rule, distinguishable. For what leads medical men into error is, I think, the fact that they pay more attention to the study of one class of diseases than to that of others. Thus, I have, on two occasions, seen a distinguished hospital physician mistake general paralysis for delirium tremens; one of these cases was that of a medical man whom I knew to be of temperate habits, but who had had a severe fall from his horse some time before. The mistake arose from the fact that the physician ignored all mental complaints, whilst delirium tremens was a malady which was daily before his eyes.

Difficulties, however, do present themselves even to the most practised alienists. Thus, not long ago, I witnessed the case of a gentleman who, in the opinion of all those who saw the case during its later phases, was affected with general paralysis. But this gentleman was thought by an alienist physician to have a tumour in the brain. The opposite of this occurred in a patient at Guy's Hospital, who, getting maniacal, was sent to the county asylum. There the case was recorded as general paralysis of the insane, but after death a cholesteatomatous tumour was found at the base of the brain.

I lately took the trouble to test the powers of diagnosis possessed by different physicians who pursued each his own speciality. A gentleman came to me, as a patient, whom I recognised to be suffering from general paralysis. He tottered or straddled into my study; he spoke thickly, like a man intoxicated; he had lost all intellectual expression; he had tremor of the tongue, and his pupils were unequal. He said he was very well, and appeared cheerful. His reason for coming to see me was the fact that he had had two or three bilious attacks. His wife, however, said that for many months he had been forgetful and strange in his manner, so that he was quite incapacitated for business. I afterwards met a medical practitioner who had known him for years, and who was content to style the case one of softening of the brain. It so happened that chance brought to my house two gentlemen, one of whom had been making a study of Duchenne's paralysis, the *ataxie locomotrice* (as it is called), while the other had been connected with a lunatic asylum. The one, seeing my patient walk with a tottering gait and with his eyes constantly directed towards his legs, was at once impressed with his own idea of the case, and this notion was strengthened when I showed him the inequality of the pupils, which, he said, is one of the symptoms of Duchenne's disease. Some amount of reasoning on my part was necessary to convince this gentleman that these characters are not peculiar to his favorite malady. My other friend instantly recognised the case as one of general paralysis, and expressed the opinion that the profession generally are very imperfectly acquainted with this disease.

This instance is, I think, sufficient to show that the true characters of the different forms of chronic brain disease are not yet so accurately defined that every one can at once distinguish between them. Many of the symptoms at present described as peculiar to some are, in reality, common to many of these affections, and are due to a general rather than to a special derangement. It is said, for example, that general paralysis is characterised by quivering lips and by hesitation in speech, followed by want of power in the limbs; that it often terminates in epilepsy, and that its mental symptoms consist in a loss of control, in the existence of delusions, and, ultimately, in a state of dementia. The ideas are generally of an exalted or ambitious kind, but authorities state that the intellectual phenomena vary, and that, whilst some patients are demented, others are maniacal. It is clear that many of these symptoms belong simply to an atrophy of the brain, being present even when this arises from alcoholismus; hence, we must ask whether the peculiar form of delusion, which is certainly one of the most striking features in these cases, is sufficient to characterise the complaint, and whether its absence is enough to negative the diagnosis of general paralysis. Further, we must inquire whether a meningo-cerebritis, or some other definite pathological change, is found after death in every case of this disease. If a negative answer is given to the last question, then my friend who called a case of general paralysis by the name of softening of the brain, by which he meant a general decay of the brain, was not far wrong. I have already said that if, in a case of alcoholismus, we abstract the symptoms of abdominal visceral disease, the nervous symptoms remaining correspond closely with those of general paralysis. I have also alluded to the so-called "ataxie locomotrice," which is said to be a disease with characteristic symptoms; but the condition of the pupils, and the presence of paralytic symptoms affecting some of the cerebral nerves, show that the seat of this malady lies within the cranium, and not merely in the spinal cord. Hence, my colleague Dr. Gull, many years ago, alluded to such cases in his 'Gulstonian Lectures' under the name of *cerebral paralysis*, and said that they were brought about, not by any actual softening process in the spinal cord, but by more general causes, such as sexual excesses.

I have said enough to show that, in studying the chronic affections of the brain, we should endeavour to discover which symptoms are universal, and which are proper to particular forms of disease. These complaints, as a rule, can be distinguished by the circumstances attendant upon them; it is not difficult to determine whether the failure of bodily and mental power arises from poisoning by lead, mercury, or alcohol, from old age, or from chronic inflammation.

The post-mortem appearances found in general paralysis are so far peculiar that in this disease the brain is not always obviously wasted, although the normal tissue is really destroyed to the same extent as in atrophy. Moreover, as this morbid change progresses slowly through the brain, and does not affect it all at once, the symptoms of it may vary. Since it begins in the cineritious surface, the mental powers often fail whilst much bodily power remains. Thus, every lunatic asylum contains many patients suffering from this disorder who can walk well, and who enter into the games of the institution, or are engaged in labour. That the guiding will may be almost absent, although the motive powers connected with the central ganglia are perfect, is sometimes remarkably shown by cases of patients who have considerable difficulty in starting, but who, when once the machinery is set a going, will continue to walk until exhausted, having scarcely any power to stop. Such a case is the very opposite of one in which, from disease of the centres or spine, all power is lost, though the mind and will are intact.

In the general paralysis of the insane, then, we find a chronic change in the brain, and especially in the grey substance immediately beneath the membranes. The inevitable result of such an affection is the gradual decay of all bodily and mental power. By removing this morbid condition from the category of special disorders we are not taking from its peculiarities, but adding to its interest; for if a spontaneous change of the kind mentioned is productive of certain well-marked results, it is a point of great pathological and physiological importance to observe that like affections, induced by causes of various kinds, give rise to very similar symptoms. This we see, for instance, after injuries of the head and effusions of blood; and, under these circumstances, the post-mortem appearances also resemble what is seen in the idiopathic form of disease; indeed, it sometimes happens that the lesions last named are the immediate exciting causes of a disease which is said to be simply mental. Thus, I believe I am correct in asserting that, according to the reports of asylums, evidence of hereditary influence fails in this disease more often than in any other class of affections seen in these institutions. This is in favour of the view that general paralysis may affect a previously good brain. If my memory serves me right, a history of hereditary predisposition is wanting in a quarter of all cases of insanity.

The accidental nature of this disease, perhaps, throws some light upon the cause of its greater frequency in men than in women, and of its extreme rarity in ladies of the upper class of life. Ordinary derangement of the intellect is generally due to a defect in the original formation of the brain, but a bad organ may last the proprietor of it during the whole of a long life; on the other hand, general paralysis is a disease which is generally continuously progressive, and which is seldom protracted beyond four or five years.

As before said, the post-mortem appearances consist in the membranes being thickened and adherent to the cineritious substance, which has virtually undergone a complete decay. Persons affected with such a *peri-encephalitis* are liable to effusion of blood on the surface, and may thus die in an apoplectic attack. If a patient should survive such an attack for some weeks, so that the blood becomes organized, there may, in the absence of a good history, be a question whether any structural change had occurred before the effusion took place. That an effusion of blood is itself sufficient to produce both the changes found and the associated symptoms, is clear from the fact that these effects sometimes result from an injury. Hence we should not be surprised to find that in the class of cases (so well described by Mr. Prescott Hewitt), in which cyst formation follows effusion of blood, mental imbecility and fits have been observed. Again, in reference to the diagnosis from alcoholismus, it would seem that intemperance in drinking is actually capable of setting up in the brain the process which is considered to be the basis of general paralysis of the insane. Knowing, too, how intimately the mental operations are associated with the integrity of the structure of the cineritious substance, the fact that moral disturbing causes may induce the disease is not incompatible with the other fact that it is sometimes due to physical causes. Since, then, this form of peri-encephalitis, whether sanguineous false membranes are or are not present, is characterised by disturbance of the intellectual faculties, the results of pathological and clinical observation agree. In these cases, as a rule, the whole brain subsequently degenerates, so that there is loss of bodily power, and with it an atrophy of the spinal cord, which I have sometimes found visibly shrunken and containing amyloid bodies. Again, since irritation of the surface produces convulsive movements, epileptic fits might be expected in these affections; and we, of course, observe also occasional vomiting, and other symptoms which are common to many brain diseases.

My own acquaintance with the morbid anatomy of this affection is gained entirely from a few cases which I have seen at the hospital, and from examining specimens that have been sent me. I regret to find that in the reports which are abundantly heaped upon us from lunatic asylums the work of the mere secretary or superintendent so much overshadows that of the physician, and that the scientific value of these pamphlets is altogether sacrificed to their business character.

In the reports which are in my possession I fail to find any well-recorded cases with details of post-mortem appearances, and I am therefore fain to turn to Calmeil, from whom I will quote one or two examples in illustration of the disease. One of the symptoms noted in the first which I shall give is pain in the head, and this was present in other cases also. In the second case cited no mention is made of ambitious ideas, excepting the fact of one delusion: was, then, the change in the brain of a peculiar kind?

Case.—*General paralysis; peri-encephalitis.* — Madame E—, æt. 31, after a confinement began to suffer pain in the head; this recurred at intervals for three years; she then began to lose her consciousness, and had convulsive movements affecting the face and limbs. She became feeble in all her movements, and had difficulty in speaking (sa voix saccadée et trainante.) The intellectual faculties began to be obliterated, and she could no longer manage her domestic affairs, nor even herself. She often said she was queen or empress, that she was going to build herself castles, &c. The paralysis then became more complete, and epileptic attacks occurred. She afterwards had difficulty in deglutition, and died three years after being put in seclusion. The post-mortem examination showed that the pia mater was so adherent to the summit of the brain that it was utterly impossible to detach it without producing a complete separation of the cineritious substance; there was adhesion also at the sides and at the base. The whole brain, including the medullary as well as the grey matter, also appeared softer than natural.

Case.—*General paralysis; no ambitious ideas; peri-encephalitis.*—A man, æt. 48, had suffered much grief from loss of fortune. For the first year he experienced vertigo and occasional momentary loss of consciousness. He afterwards lost his memory, and became incapable of attending to his business; he remained immobile in the same place, cried or laughed without an object, and believed himself to be Marshal of France. His pronunciation became embarrassed; he could walk only in a tottering manner; and he ultimately became quite demented, not knowing his name or his age. He was even obliged to be fed and at last he could only just move his arms or legs. The post-mortem examination showed the grey matter to be infiltrated with a greyish substance, like concrete pus, and to be adherent to the vessels of the pia mater. The brain was wasted, and the corpora striata and thalami showed depressions on the surface.

Another case which Calmeil gives is interesting, as being one of acute peri-encephalitis, although in all probability this was merely consecutive to a more chronic change. Had it not been so, I should demur to an acute disease of the kind being placed in the hands of an alienist and transferred to an asylum. An acute meningo-cerebritis is seen every day in connection with tubercles.

CASE.—*Peri-encephalitis; acute upon old disease.*—A man, æt. 37, a year before the acute attack, had trouble in business, from which he sank into a profound sadness. On January 13th, he appeared suddenly altered in manner, began to talk with volubility, and spoke of extraordinary projects and speculations. On the 17th he was taken to the asylum. He then uttered cries and gesticulated with fury; the pupils were dilated, the eyes haggard, the face pale, the skin covered with sweat, the pulse quick, the tongue dry; there was constant agitation and sleeplessness. On the 20th he suddenly lost his consciousness, and had convulsions, with intense fever. These symptoms continued until the 24th, when he died. *Post-mortem.*—The pia mater was found much injected and infiltrated with fibrinous products, which were of a milky colour and ran along the vessels. The pia mater, as a whole, was thickened, and closely adherent to the cineritious substance; the convolutions were squeezed together, and the sulci obliterated. The grey parts of the interior were of a violet colour, and the surface of the ventricles was in a granular condition. The microscope showed small extravasations of blood, inflammatory products, and molecular and granular corpuscles.

Now, it is not improbable that an injury to the head might set in operation a chronic inflammatory process of the kind described in these instances, nor is it at all unlikely that many of the cases of blows on the head which constantly come before us in hospitals do terminate in this manner. Amongst our out-patients the effects of injuries are frequently seen. Besides those which I relate in this place I shall be able, when speaking of epilepsy, to mention several other cases which illustrate this fact.

CASE 40.—A boy, æt. 11, had been thrown off an omnibus six months before I saw him. He was stunned, and had a blood tumour on the scalp. He never recovered the shock, and five months afterwards had a fit. Subsequently, he had several fits. When he came under my notice, he was stupid; he had been obliged to leave school, and could not read nor remember; he could not apply himself; indeed, he was becoming imbecile.

CASE 41.—Another lad, æt. 14, two months before I saw him, fell and struck the back of his head. He was stunned, and afterwards complained of pain over his forehead; a month after he had a fit, and he lately had another.

In these instances the more immediate effects are seen; but I have another case, that of a man who received a blow nearly a year ago, and whose mental powers are going. He totters as he walks, and yet he is happy and contented, and talks of returning to his work. His state approaches that of general paralysis. Bayle, who was one of the first to write on the pathology of the general paralysis of the insane, insisted on the fact that chronic meningitis is the cause of the disease. He believed that sanguineous congestion or effusion was the primary condition; that this gave rise to inflammatory action, and to the pouring out of an exudation which was transformed into false membranes, and that from these resulted the epilepsy. Thus, he apparently left it an open question whether the false membranes arise from inflammatory lymph or from blood. The two latter so often coexist that difficulties constantly arise in determining their exact pathology. If a cyst should be found in a case in which symptoms suggestive of effusion of blood had followed a direct injury to the head, there could be little doubt as to the origin of this morbid appearance; but when such a cyst has been met with accidentally, and combined with periencephalitis, a question may arise as to which was the prior affection. In a case published by me[1] of a lad in whom a large hæmatoma existed, the surface of the brain beneath was of a white colour, owing to the great thickening of the pia mater, and to its intimate connection with the cineritious substance. The lad had spent his life in the workhouse, and had been regarded as half-witted; he was very fond of frolicking and tumbling about. Dr. Bacon, of the Cambridge Asylum, related to me the following case:

CASE 42. *General paralysis; effusion of blood.*—A man, æt. 37, was admitted December 14th, 1865. He had been a billiard-marker, and had a dissolute appearance. His father, who lived with him, asserted that there was nothing the matter with him until December 1st; but some of his friends said his manner had been altered. On that day, being evidently ill, he was sent to a hospital, but the case was considered a trifling one. On the following day he became noisy and excited, and was discharged as insane. When admitted into the asylum he was stupid and dull, and only spoke occasionally; his remarks gave evidence of stupidity and elation combined. He was unsteady on his legs, and his muscles were tremulous. The case was considered to be one of dementia, connected with general paralysis. During the next few days he gradually became more feeble. On the 22nd he took to his bed, and on the 23rd he was semi-comatose. He could not move his limbs, and those of the left side were rigid. He died on the 25th. The post-mortem showed, when the dura mater was removed, that the right side of the brain was covered with coagula and fluid

[1] 'Psychological Journal,' April, 1865.

blood, about six ounces in all, by which the brain was irregularly compressed. The effusion seemed limited by a tough layer of organized lymph, which was easily peeled off the dura mater. The brain appeared healthy, and there were no signs of external injury. (Was the effused blood the primary affection or a mere accident in the course of a chronic disease ?)

Calmeil gives accounts of cases in which ambitious ideas and a happy delirium had existed, and in which cysts were found in the cerebral membranes. The cineritious structure was, no doubt, involved in a chronic change, and such cases, perhaps, show that any form of chronic periencephalitis may be attended with the symptoms usually observed in the general paralysis of the insane. In the following case there was a history of injury several years before, so that if this was the exciting cause the cyst must have been of a considerable age.

CASE.—*General paralysis; exalted ideas; cyst in brain.*—The patient, æt. 44, was a highly intellectualand courageous man. At the age of twenty-nine, whilst in the marines, he received three wounds on his head, by which his jaw was broken, &c. Some years after he was again wounded. When forty-one and a half years of age his conceptions became disordered. He had highly ambitious ideas, and fancied himself to be possessed of enormous wealth, and of palaces and horses. He then had paralysis of the face, walked with tottering gait, &c. The subsequent progress of this case was that of general paralysis. The post-mortem showed on the left side of the brain a pesudo-membranous pouch, filled with a yellow fluid. One side of the cyst touched the dura mater, while the other was in contact with the brain. Other parts of this organ were covered with membranous patches, and the pia mater was adherent at several points to the brain beneath.

Now, Calmeil gives cases of peri-encephalitis of long standing, terminating suddenly by sanguineous effusion; and, therefore, there is a difficulty in determining whether the cyst sometimes found constitutes the original disease or is merely an accidental complication. I have no doubt that those pseudo-membranes which are coloured by hæmatine arise merely from blood; but a thickening of the pia mater and arachnoid, with close adhesion to a degenerated cineritious substance, must, I apprehend, be due to a different pathological process, even though it may have been produced by the same cause. When the affection results from injury, the same blow which caused the effusion may have so damaged the brain as to set in action a chronic inflammatory process. There can be no doubt that moral circumstances alone may set up a chronic peri-encephalitis, but it is most probable that a person who had previously received an injury would be more likely to be thus influenced. It is, too, quite in accordance with what we know that this disease should be more liable to occur in those who have indulged in alcoholic excesses. Although I have spoken of the most characteristic change as being found on the surface, yet it is shown by cases which have been recorded that the whole brain may be involved, and that even the central ganglia are sometimes evidently diseased.

Calmeil mentions the case of a lady, æt. 55, in whom there was no hereditary history of insanity, who complained of various pains about her, and was afterwards affected with slight paralysis of the tongue, and at times attacked by fits accompanied with loss of consciousness. She was sometimes in a state of happy delirium, sometimes furious and incoherent. She died in the second year of her illness. There was found in the arachnoid of the left cerebral hemisphere a large cyst, containing blood, fibrin, &c., extending from before to behind; the cyst was organized, and adherent to the brain; the convolutions on the surface were partly obliterated. (The author considered that this cyst was formed from an effusion of blood, which had occurred during one of the fits.)

Calmeil also gives the case of a man who had a large cyst on both sides, in contact with the dura mater and the brain. The patient, æt. 55, had suffered for two years, with mental disturbance, &c. He had been intemperate. Other instances of general paralysis, with ambitious ideas, have also been recorded, in which old cysts in the arachnoid cavity have been found.

This disease, then, consisting, as it would seem, in a chronic inflammation, leads to a destruction of those parts of the brain which are endowed with the highest functions; but, as I have already said, it does not follow that this condition is always brought about in the same manner. Thus, in *alcoholismus* the brain wastes by a more simple process, the signs of an inflammatory action being limited to a thickening of the arachnoid and to a granular condition of the surface of the ventricles. This progressive *atrophy* is necessarily attended by symptoms of increasing loss of power, affecting both body and mind, and thus the basis is laid for the attacks known as *delirium tremens.* It appears to me that, in considering the true character of these attacks, it is essential to recognise that their cause lies in a wasting of the brain, for this is the basis of both correct diagnosis and judicious treatment.[1] Delirium

[1] If we consider the condition of a patient affected with delirium tremens, and observe how weakened is his bodily frame, and how great the failure of nervous power, we at once see that the treatment should be directed towards repose. If he sleeps, all is well; and this result may be obtained by a judicious

tremens is essentially a chronic disease, and is the result of pre-existing conditions of the nervous system. Alcohol, acting on a healthy brain, produces, not delirium tremens, but drunkenness; under such circumstances its effects are simply those of a poison. But delirium tremens implies that the patient's nervous power has previously been impaired, and that his brain has already suffered from his vicious habits. His whole body may have suffered from his long-continued excesses, but his brain has undergone a sensible atrophy, so that his bodily and mental powers are weakened. This is, indeed, shown by his trembling gait, his shaky hand, and his childish conversation. Such a person, being excited by a fresh debauch or meeting with an injury, is thrown into the condition known as delirium tremens. But his brain is in as impaired a state in the intervals between such attacks as while he is actually suffering from them; and he may, indeed, be said to be always affected with a chronic form of the complaint. Spirit-drinking causes a decay of all the tissues, including the brain; and thus, having examined a large number of such cases, I have always found this organ to be atrophied. This condition may at once be recognised by the fact that the convolutions gape, and that only fluid is found in the place of good brain substance. The patient who has such a wasted brain suffers both in mind and body; he is known as he enters your room by his tremor, and his smile, and his foolish talk. He has, in fact, become a good-natured fool—no man's enemy but his own. So many good ounces of his brain are gone; and thus Shakespeare puts no figurative expression into the mouth of Cassio when he makes him exclaim to Iago—"I remember a mass of things, but nothing distinctly; a quarrel, but nothing wherefore. O, God! that men should put an enemy in their mouths to steal

use of drugs, and of other means. But it is absurd, and even hurtful, to suppose that we are to force him to sleep by means of opium. How many times has it happened that I have been called to see, perhaps, a publican, and have found him sitting in the middle of a large room, lighted up by fires and gas? He is surrounded by his friends, who are holding him; he is bathed in perspiration, and is incessantly talking and contending with them; the doctor, standing by, declares that he has given so many ounces of laudanum, that he dare give no more (for the patient's pupils are contracted to a point), and that he is now helpless. When such a patient has been taken into a small dark room and put to bed, and all attendants excepting one have been dismissed, I have seen him, in less than half an hour, fall into a sound sleep.

away their brains." It was but lately that the newspapers contained the account of an action at law concerning the will of a person who, by the fearful vice of intemperance, had sunk from a good position in life into the most abject state of bodily and mental decrepitude. He may now be made to "point a moral;" but I quote the description principally for the sake of calling attention to the happy phrase "brainless sot," which is the most just expression for such a wretched object as is here depicted. The word "brainless" is, in fact, literally correct, as implying a loss of the good substance of the brain:— "In 1847 Sir Edward ——, then in his forty-first year, succeeded to the title and estates, having received his education at Oxford, and having afterwards been an *attaché*, during many years, at Berlin and Vienna. He was a refined, graceful, and accomplished gentleman, though even at that time the continual dissipation in which he had long unhappily indulged had seriously impaired a robust constitution. The history of his subsequent life down to his death, in the autumn of last year, is a terrible example of the effects of vice in reducing a mind originally sound to a state in which its capacity to think, or form a purpose, becomes questionable. Sir Edward's time was principally spent at Carrigoran or in visits to Dublin; and he was proved to have been continually addicted at both places to habits of drunkenness, which, acting on an enfeebled frame, produced melancholy consequences. He never, indeed, was actually demented, or subject to that frightful disease which is the special penalty of intoxication; and there was much evidence that, even to the last, he displayed a certain amount of intelligence, that under his care his property improved, that he was sometimes able to receive guests and converse with them in an agreeable manner, and that, in the common affairs of life, such as paying debts or executing leases, he was, when sober, capable of business. But, on the other hand, there can be no doubt that during the last ten years of his life, and therefore long before the period at which the instruments in dispute were signed, he had sunk into a state of debasement, in which his whole nature appeared changed; he was held hardly responsible for his conduct, and imbecility seemed imminent. The graceful gentleman became a paralytic of wretched aspect and filthy habits; the gay *attaché* degenerated into a *brainless sot*, an indecent talker, a petty pilferer; the trained and accomplished man of the world became an object of scorn or compassion. Shunned by his equals, expelled from clubs, unfit for active or social life, Sir Edward, after fifty, was a mere decaying wreck, his body a mass of weakness and disease, his mind, with some remains of intelligence, declining into premature decrepitude."

It has been my object to show that, if we look at the pathology of brain diseases, it becomes evident that those affections which denote a failure of bodily and mental power are characterised, after death, by a wasted brain. These symptoms are seen in delirium tremens, and the cause is well marked. It is difficult to make a selection among cases so common; the following are taken from my note-book of the last year:

Case 43.—A man, æt. 56, long half-witted, got out of the wrong side of a railway carriage on to the line, and was killed. His brain was remarkably atrophied, and the pia mater and arachnoid were much thickened. The ventricles were greatly dilated, and their surface minutely granular.

Case 44.—A woman, æt. 34, an imbecile, died of diseased hip-joint. The brain was wasted; the convolutions shrunken; the ventricles enlarged; the surface of these cavities granular; the choroid plexus cystic; the blood-vessels healthy.

Case 45.—An old man, æt. 78, very feeble, fell in the street, and was brought to Guy's, where he died of pneumonia. The brain was much atrophied; the arachnoid thickened; the dura mater adherent to the cranium. The ventricles large, containing much serum; their interior slightly granular. (Senile changes?)

Case 46.—A woman, æt. 25, the wife of a publican, and very intemperate. For four months she had been complaining of nervous symptoms, such as dimness of sight, pains in the limbs, impairment of memory, strange fancies, &c. On admission she could only just stand; her mind was dull and her expression vacant, and she gradually became weaker and weaker, until she died. The brain was found remarkably shrunken; the convolutions were widely separated, with fluid between them. The arachnoid was opaque. (The question was, whether this condition was produced by alcohol, or whether the malady was to be regarded as purely mental, and to be styled dementia.)

It is probable that in many cases intemperance leads to disease of the blood-vessels within the cranium, and that a definite apoplectic or paralytic attack may be really due to this cause, just as much as a general wasting of the organ.

Case 47.—A man of very intemperate habits fell back in his chair about a month before his death, and was found to have lost the use of his right side. He recovered slightly, so as to be able to walk with assistance, but he still gave wrong answers to questions, and exhibited symptoms of failing mental powers. He gradually became more feeble, and his other side weaker, and he sank into an unconscious state. The post-mortem examination showed that the convolutions were shrunken, that there was much effusion on the surface, and that the left anterior lobe was much fallen in. There were several local softenings in the cineritious substance and also in the central ganglia.

Among the results which follow chronic inflammatory changes in the brain is the production of lymph in the ventricles, which causes adhesions between certain parts, so that portions of the centre may even be completely cut off. Esquirol thus refers to them. In the lateral ventricles "the adhesions of this membrane are constant; they are rare in the other ventricles, and obliterate the appendix known by the name of the digital cavity. This appendix is almost always separated from the remainder of the ventricles by adhesions, which allow of only one or two openings between the ventricle and its posterior cornu. This membrane often adheres to the portion which covers the striated body. These adhesions, which are more or less extended, cause the ventricles to lose their true character."

Some time ago Dr. Rolleston, of Oxford, was mentioning to me that this morbid adhesion would be likely to cause a resemblance between the interior of the human brain and that of the brain of some of the lower animals. Dr. Bright also alludes to the same morbid condition, especially to cases in which adhesions had completely cut off the posterior cornu. This writer likewise speaks of the above-described alteration in the cineritious structure of the brain, as being shown by its change of colour, and by the easy separability of its layers in many cases of disease. These facts are referred to in the two following cases.

In the museum there is a prep. (1564^{75}) of a slice of brain, showing the separation of the external layer of the cineritious substance.

CASE.—W. S—, æt. 48, under Dr. Bright, in 1830. He had been a mountebank, and was noted for the rapidity and extent of the movements of his neck. He was much addicted to drink, and for two years he had been in a state of mental irritability. He was too feeble in mind to give any account of his symptoms. He was with difficulty kept in bed, and was constantly making grimaces, &c. After his death there was found an increased effusion on the brain, and a remarkable ossification of the vessels of the pia mater. The central part of the brain presented a peculiar tendency to separate, as the external layer was readily detached, leaving the remainder still preserving the form of the convolutions. The membrane lining the ventricles was thick, and of almost cartilaginous firmness, and not smooth. It presented some partial adhesions at the anterior part about one of the corpora striata, and posteriorly on the right side the posterior cornu was nearly, if not quite, shut off from the other part of the ventricle; and, being somewhat distended with serum, reached nearly to the surface of the brain. Both corpora striata were somewhat irregular, and in both there was a yellow stain, as of old effusion.

CASE.—Jno. H—, æt. 34, had for some years complained of pains in his head, which were sometimes so severe that he expressed a wish to die. In 1827 he was attacked with an epileptic fit, and he afterwards had headache and sickness, and, after this several more fits. He subsequently began to exhibit symptoms of imbecility, with loss of vision. He was also very feeble, so that he walked with a tottering gait. He then seemed quite lost; he never spoke, and had more fits, and his right side was paralysed. There was found a wasted brain, with increase of serum and opacity of the membranes. Over a large surface, on the left side of the brain, the convolutions were of a brownish colour. The most remarkable appearance was a very firm adhesion of the surface of each corpus striatum to the opposite side of the ventricle, for the space of nearly half a quarter of an inch square, this being so firm that the membranes tore from the surface on attempting to separate them.

General cerebritis, or softening.—Having seen that disease of particular parts of the brain gives rise to special symptoms, we shall not be surprised to find that the remarkable cases sometimes observed of general softening or cerebritis, are attended by so many phenomena that one fails to predominate over the other; hence the symptoms become almost negative. A local lesion, such as occurs in apoplexy, being productive of paralysis, can never be mistaken for blood-poisoning, by which the whole brain would be involved. But in a general inflammation, an encephalitis, or a cerebritis, no such distinction can be maintained; and as considerable pyrexia is present, it will excite no surprise that such cases have been mistaken for fever. In the few instances I have seen in which the whole substance of the brain has been involved the symptoms have been little more than a gradual failure of all bodily and mental power, such as is witnessed in cases of typhus. If one part of the brain should be affected more than another, there will, of course, be a special paralysis; but otherwise the symptoms are little more than weakness of the limbs, strange feelings, and an interference with the intellectual faculties, with, perhaps, giddiness, sickness, headache, and other symptoms common to all febrile disorders. The patient may at last have convulsions.

Such an instance occurred in a young man in Dr. Barlow's ward. He was admitted with the history that he had been for some time getting into a torpid state. He lived three weeks after this, lying in bed on his back, perfectly motionless and still. He never spoke, nor seemed to understand what was said, although his eyes followed objects in the ward. The brain appeared, after death, to be universally affected by an inflammatory softening; it was pulpy, and in parts almost semifluid, and of a pinkish colour. The changes were most marked in the medullary substance, but at certain points the cineritious matter also was involved. The softening was most decided in the hemispheres, the central ganglia being much less affected. The cause of this universal softening was not evident. In another very similar case the man lived only a week.

Epilepsy; disease of the surface of the brain.—We have seen that the intellectual powers are affected by diseases implicating the surface of the brain. I must now refer to another symptom connected with irritation of this structure, namely, convulsion. This is a fact of general experience. Arachnitis, especially if tubercle is present, is accompanied by convulsion. When an accident is followed by convulsive twitchings, a laceration or other severe injury to the surface is supposed to have occurred. We have already seen that in the general paralysis of the insane (which appears to be a peri-encephalitis) convulsions or epileptic fits occur. But perhaps the most striking cases of all are those in which such symptoms result from a permanent adhesion of the membranes to the surface of the brain at some particular spot.

Epilepsy is so striking and remarkable an affection that most medical writers have made it the subject of their efforts to theorise. Many have been the explanations advanced in elucidation of its nature. Some authors have attributed it to a local disease of the brain; some to causes altogether external. Of this we have an example in the remarkable theory propounded by Dr. Marshall Hall. The study of the phenomena of epilepsy may, no doubt, lead to the most varied conclusions. But it is remarkable that post-mortem examinations have not thrown much light on this disease. If we endeavour to gain an idea of its nature by considering what are the conditions in which similar phenomena occur, we find that, on the one hand, a local lesion is sufficient for their production, and, on the other hand, that one of the most efficient causes appears to be an altered condition of the blood generally, from the presence, for example, of urea.

It is necessary, in the first place, to determine the meaning generally attached to the term epilepsy by members of our profession, or rather to ascertain how they define it. Formerly a certain set of symptoms denoted the disease; and if, after death, a tumour or exostosis was discovered within the cranium, this was regarded as the cause. At the present time, however, if any such local change should be found, we speak of the latter as the disease, and should describe the convulsions as epileptiform rather than epileptic, epilepsy being now usually considered as an affection of which the true pathology is not so easily discernible, and which, in fact, is not associated with any evident morbid changes. Again, as regards the symptoms, the definition implying a loss of consciousness combined with a general convulsion is, no doubt, applicable to the majority of cases; but all observers are not agreed as to which is the most essential symptom. It is certain that loss of consciousness may be the only phenomenon present in the early attacks constituting the "petit mal." But some medical men believe that cases of epilepsy have occurred in which convulsion has been the first symptom. Of this I have never myself seen an example, nor do I know that any well-marked cases of this kind have been recorded, and therefore I am content to regard loss of consciousness as the main and essential feature of the disease. In the ordinary form of epilepsy consciousness is lost during the fit, and with it, of course, all feeling, while at the same time there are violent general convulsions. In the intervals, too, it is clear that the mind is involved. It always strikes me with surprise to read, in medical works, of highly intellectual persons being the subjects of fits; for my own part, I always observe epileptics to be deficient in mental power, and not uncommonly find them maniacal. In fact, in the case of an epileptic we have to give a melancholy prognosis, namely, that imbecility will one day manifest itself. I will not say that, since mania is often associated with the fits, we are to agree with M. Falret, and declare that an epileptic is an irresponsible person; but I am inclined to think that there is much force in his argument.

My object, however, is, not so much to discuss the nature of epilepsy, as to record facts for the further elucidation of this subject, and therefore I may say at once that the morbid conditions of the brain which we find to give rise to epileptiform convulsions are remarkably uniform. They all point to the presence of local irritation of the surface. Thus, the most definite affection discovered in cases in which the symptoms have been undistinguishable from epilepsy is an old adhesion of the membranes to one spot on the surface of the brain. In removing the skull in such a case we find the dura mater firmly attached to the convolutions beneath by old exudative or inflammatory material over the space of one or more square inches. This condition may arise from disease of the bone, from injury, or from syphilis. As I have had to say in reference to other diseases, exceptional cases may occur; but even here there is always the possibility of incorrect observation. I remember there being brought before one of the medical societies a case in which a tumour was found in the pons Varolii, the patient having had epileptic convulsions. This was regarded as confirmatory of Van der Kolk's supposition that the cause of epilepsy is seated in this part; but I have no hesitation in saying that for one such case fifty might be found in which the morbid changes producing these symptoms occupy the surface. In searching through my hospital records, I find that the condition above described was present in the large majority of those cases of epilepsy in which any local changes were discovered as the cause of the symptoms. It is this which occurs as a result of syphilis or of injury. Moreover, I believe that I am correct in saying that the surface of the brain was always implicated, even in those other cases in which local affections not of this kind were found. It may be objected that the very fact of definite lesions having been found shows that these cases were not examples of true epilepsy; and, indeed, it may be said that the symptoms sometimes differed from those of that disease. There is some truth in this; but at the same time it appears to me that, if a known cause is sufficient to produce a certain set of symptoms, an analogous cause should be sought whenever similar symptoms are witnessed. As a distinction between the cases of which I have been speaking and those of true epilepsy, it has been noted that consciousness is not always absent. As far as I know, Dr. Bright[1] was the first to observe this fact, and upon it he often based the diagnosis that a case was not one of true epilepsy, but that local disease was present. His statement I have seen confirmed in many instances. Again, in the very rapid succession of the fits and in their termination in death these cases differ from what is observed in true epilepsy; and I believe that in these cases there is more marked hemiplegia. A temporary paralysis is often observed in true epilepsy; but we then find that both sides are affected on different occasions, whereas when a local source of irritation is present the hemiplegia remains for a considerable time after the fit, and is always on the side opposite to the seat of disease in the brain.

With facts like these before me, I cannot see any grounds for a theory which supposes the seat of epilepsy to be in the pons or in the central ganglia. Disease of these parts would produce paralysis; but in order to produce increased movements, they must be healthy and susceptible of irritation. In the case of feigned epilepsy we should say that the will, which is intimately associated with the cineritious structure, acts on the central ganglia beneath, and excites them to the production of violent movements in the limbs, exactly as in the real disease. Thus, we can believe that in true epilepsy there arises in the superficial parts of the brain an influence which is independent of the will, and, in fact, takes away the consciousness, by operating through the cineritious substance, and which also irritates the ganglia below and sets up the paroxysm. At the same time we may allow that the pons Varolii and medulla oblongata also are excited, and we may thus explain the affection of the respiratory nerves, and of the spinal accessory,

[1] "My reason, then, for supposing that the epileptic attacks in this case depended rather on a local affection than on a more general state of cerebral circulation or excitement, was *the degree of consciousness which was observed to be retained during the fits;* for although we meet with great variety in this respect, yet in two cases which have occurred to me the fact of the patient generally remaining conscious has been a remarkable feature, while in each the injury on which the fits depended was of a local rather than a constitutional or a general character." ('Guy's Hosp. Rep.,' series 1, vol. i, p. 39.)

which causes the distortion of the head. Theoretically, we should suppose that, if violent movements are produced, an increased influence must be exerted upon the central cerebral ganglia; and this would, of course, originate in the cineritious surface above. That this violent and morbid influence should be accompanied, during the time of the paroxysm, by a loss of consciousness, is what might be expected, and we should also think it probable that such violent commotions would, in course of time, impair the structure of the cineritious matter, and lead to imbecility. Then we have before us the fact that disease of the surface induces epilepsy, as is seen in the peri-encephalitis, or general paralysis of the insane, and in other instances in which blood is effused and the surface injured. Even the cases in which, a local morbid change having been present, the symptoms have somewhat differed from those of ordinary epilepsy, are confirmatory of this view of the nature of the disease, by affording just those exceptional symptoms which ought to be met with. For in these cases, the cause being definite and local, an irritation is set up in the corresponding ganglia beneath; and thus the occurrence of convulsion without loss of consciousness is explained. Again, since the disease is progressive, the fits increase until they become constant; and, in consequence of the irritation being on one side, the convulsions are often unilateral, and the weakness which follows by an inhibitory action is also confined to one side of the body. Thus, supposing that in ordinary epilepsy the whole surface of the brain is involved, we might imagine that, as is really the case, a permanent local change would give rise to a disease differing from epilepsy in these respects. Should the local irritation excite the whole surface, the symptoms would, as might be supposed, be like those of ordinary epilepsy. It appears to me that, from clinical and post-mortem observations, as well as from all analogy, we cannot but conclude that the *fons et origo mali* is in the cineritious substance of the brain. I believe that in this region a commotion occurs which would, perhaps, be analogous to a palpitation affecting the heart, and that this irritates the ganglia below, which form the summit of the motor tracts. A laceration of the surface, of course, produces the very same phenomena; and, indeed, the implication of the intellectual functions proves that the seat of the disease is in no unimportant part of the brain. The difficulty felt by some of the older writers on this disease was, that the loss of consciousness showed the brain to be paralysed, while the convulsions proved the functions of the spine to be exalted. This difficulty disappears altogether when we admit the explanation given above. The aura experienced in some cases, and the shriek sometimes uttered, are entirely subjective, and are due to some sudden alteration in the action of the brain. That a poisonous matter like urea passing through the brain should interfere with its functions, also accords with the theory I have given. So far as I know, Dr. Todd has been the only author who has insisted on the fact that the seat of epilepsy is in the cerebral lobes; but, if I remember rightly, this view was not generally adopted at the time of its propagation, on account of objections based on physiological grounds; nevertheless, it appears to me that everything points to the correctness of this opinion. I am myself so convinced of it as to feel sure that the improved method of examination used by Mr. Lockhart Clarke will show a well-marked change in the cineritious substance of the brain in cases of long-standing epilepsy.

If permanent paralysis exists, I believe that something is superadded, and that the motor tract is involved; the hemiplegia met with in ordinary epilepsy being, in my experience, generally of an inhibitory nature, and therefore temporary. In a late number of the 'Psychological Journal' a writer attempts to controvert this opinion, which he quotes from the work of Dr. Russell Reynolds. But I am not convinced by the arguments of this writer, and believe that he will find some local disease of the brain in any epileptics in his asylum who may have been permanently paralysed.

I may take this opportunity of alluding to the operation which I have more than once seen performed for the relief of epilepsy. This was formerly done in the hope of being able to remove an exostosis or a piece of thickened bone; but as the local cause generally consists in an adhesion of the membranes, an operation can be useful only by allowing a bulging of the dura mater, or a growth of granulations, by which the brain within might be relieved. Trephining cannot be done with much hope of success.

It is remarkable that such remedies as the iodide and bromide of potassium[1] have often been used with more success than the so-called nervine tonics; we may, perhaps, infer that they act as absorbents, and thus actually produce a change in structure.

In conclusion, I believe that no symptom is associated with disease of a particular part of the brain more certainly than convulsions with an irritation of the surface.

I will first speak of those instances in which chronic local adhesions existed, and gave rise to epileptic symptoms; outlines of a few cases recorded in my books during the last half dozen years will suffice. It will be observed that in some of them the disease was on the superior surface, in others at the base of the brain.

Case 48. *Epilepsy; adhesion of membranes to brain.*—A man, æt. 31, had fits, and at times was almost maniacal. His left arm and leg appeared weaker than the limbs of the opposite side, but he could walk about until within a few days of his death. The convulsions then occurred in continual succession, his arm being constantly in rapid motion. The post-mortem examination showed the membranes to be closely adherent to the inferior surface of the middle lobe of the brain on the right side. The exudative material was present which is found in syphilitic cases. In the right corpus striatum there was a slight spot of softening.

Case 49. *Epilepsy; adhesion of membranes.*—A man, æt. 34, a soldier. He said he had had a sun-stroke in India, and also one in the Crimea, and that since then he had had fits. He was admitted on account of severe pain, which he styled neuralgic, on the left side of the head. He had several fits, one or more, a day. He at last died in a fit. His mind was not very clear, and the history is therefore doubtful. The post-mortem examination showed the middle lobe inferiorly to be adherent to the dura mater by a quantity of tough new material, which spread over the temporal bone. This was just such as is seen in syphilis.

Case 50. *Epilepsy; adhesion of membranes.*—A man, æt. 40, came to the hospital one day, asking for admission. He said that he had been in better circumstances, but was now in an impoverished condition and out of health. He was in a dull apathetic state, and immediately went to bed. Soon afterwards he was

[1] Having never yet taken to myself credit for the adoption of any new remedy in disease, I may here state my belief that the publication in the 'Med. Times and Gaz.' of my cases of epilepsy treated by bromide of potassium, in the year 1859, caused the remedy to be used more largely by the profession. The drug had been recommended by Sir C. Locock in the nervous diseases of girls supposed to be suffering from ovarian irritation. In books it was mentioned amongst other remedies, but was condemned as valueless. My using it arose from my having found epilepsy, whether originating in syphilis or not, to be often due to local causes. Hence, I began to treat all cases by the iodide. On the bromide coming into use for bronchocele and other affections, I substituted it by way of trial, and then I witnessed results more remarkable than I had ever anticipated.

found insensible, having stertor and convulsions; in six hours he died. It was afterwards learned that he had led a very abandoned life, and that he had had syphilis. He had complained of his head, and felt dizzy; it was not known that he had ever had a fit. Over the right temporal region the dura mater at one spot was found closely adherent to the brain, a tough yellow mass of new material uniting them together.

Case 51. *Epilepsy; adhesion of membranes.*—A man, æt. 34, came in with fits, from which he died. No history was obtained. On the left side the dura mater was firmly adherent to the brain, and also externally to the skull at the same place. The posterior lobe on the left side also was firmly adherent to the dura mater, there being a yellow amorphous matter between them.

Case 52. *Epilepsy; adhesion of membranes.*—A man, æt. 50, had received a blow on the head from a poker twelve years before, and had since suffered from epileptic fits. At the seat of the injury the bone was evidently thicker. He was trephined, and two portions of bone were removed, but with no benefit. Near the injured spot the dura mater was connected firmly to the brain by a quantity of tough yellow material, which penetrated through the whole depth of the cineritious substance.

Case 53. *Epilepsy; adhesion of membranes.*—A woman, æt. 31, had had syphilis, affecting her eyes and other parts. She had various nervous symptoms and fits. The anterior lobes of the brain were firmly and inseparably united to the dura mater. (This case has been fully detailed in a former volume of the 'Reports.')

Case 54. *Epilepsy; adhesion of membranes.*—A man, æt. 35, had had for some months abscess and disease of the left parietal bone. He then had a fit with partial paralysis of the right side. On the following day he had another fit. An incision into the scalp was made; but the convulsions returned with great frequency and violence, affecting chiefly the right side of the body. He was then trephined, and some dead bone was removed. Immediate relief followed. He said he was quite conscious that he was being operated upon, and that he was always conscious during the fits. At the end of a week the fits returned, and they subsequently recurred at various intervals. He ultimately died of pyæmia. Much dead bone was found, beneath which the dura mater was adherent to the brain.

Case 55. *Epilepsy; adhesion of membranes.*—A man had three years before been thrown out of a cart, receiving a severe blow on the head. Some time afterwards he had fits, which increased upon him until his admission, and after this occurred in continual succession until his death. On post-mortem examination the calvarium was found thickened. The dura mater was likewise thickened, and was closely adherent to the anterior lobe of the brain on the right side, the cineritious substance also being involved in the disease.

The museum contains several old specimens of parts of the brain with the dura mater adherent, from patients who had been the subjects of epilepsy (Prep. 1584[50], 1586[33], 1602, &c.). In one case the patient, who had fits in rapid succession, declared that he knew what was passing around him during the paroxysms.

Convulsions are observed also in cases in which there is no adhesion of the dura mater at any particular spot, the surface of the brain having undergone a chronic inflammation from other changes. I have already alluded to this subject when speaking of peri-encephalitis, in reference to the mental change which results from this condition.

CASE 56. *Epilepsy; old injury to the brain.*—A man, æt. 43, was admitted suffering from epilepsy. Between two and three years before, he had fallen on the back of his head, and, a year before, he had had a fit for the first time. On the morning of his admission it was observed by his fellow-workmen that his appearance underwent a sudden change, that his face became contracted, and that blood flowed from his mouth. He afterwards had another fit, and was brought to the hospital. He was then quite insensible, the pupils dilated, the respiration labouring, the cheeks blown out, &c. The fits continued with scarcely any intermission; he afterwards appeared to be paralysed on the left side. The post-mortem showed clearly that the brain had at one time received a severe contusion; the surface of the anterior parts (which would suffer from *contre-coup*) presented a brown or ochry colour from effused blood. This condition extended into the cineritious substance. The anterior lobe was adherent to the dura mater, and so was also the under surface of the left lobe.

A very similar case was, some years ago, brought before the Pathological Society by Dr. Bristowe. The patient had been subject to epileptic fits, and after death the anterior lobes were found to be adherent to the orbital plates by a delicate areolar tissue of an ochry tint. This morbid change was believed to have arisen by *contre-coup* from a fall on the back of the head.

At a meeting of the same Society Dr. Quain showed a specimen taken from a boy, æt. 13, who had been struck on the head by his schoolmaster four years before his death. He afterwards always complained of pain, and after some months became the subject of epileptic fits. On post-mortem examination the bone was found to be thickened and carious, and below this, in the substance of the right hemisphere, there was a cretaceous mass.

The 'Transactions of the Pathological Society' also contain accounts of several cases in which epileptic attacks were associated with cysts on the surface of the brain.

Again, tumours involving the surface of the brain give rise to fits, and so do morbid deposits of any kind affecting the cineritious substance, as in the second of the two following cases.

CASE 57. *Epilepsy; tumour.*—A man, æt. 34, was admitted for fits. He knew when they were approaching; he felt his sight going, but the retinæ were sensitive to the impressions of objects during the whole of the paroxysm; indeed, he never lost his consciousness. On the day preceding his death he became unconscious for the first time; he appeared to be paralysed on the left side; and with these symptoms he died. A large growth, the size of a duck's egg, was found occupying a great part of the right hemisphere. It involved the medullary matter, and also the cineritious substance of the surface. Below it reached as far as the central ganglia.

Case 58.—A man, æt. 32, whilst gettiug out of bed one morning in August, 1862, found that his right side was partially paralysed, and his mouth drawn up; he also felt pain in his head. He recovered, and at the end of three months resumed his employment, when he had another fit which affected the opposite side. In February he recovered from this attack. In September, 1863, he was again admitted in a half-conscious state, having frequent fits; he lay in bed in a stupid condition, with his limbs contracted, and he was partially paralysed. He was blind from old disease. The post-mortem examination showed the dura mater to be adherent to the bone. Imbedded in the cineritious substance were several small white deposits. The central ganglia were softened, and the pons Varolii had a deposit in it. The liver was cicatrized, and contained similar deposits.

Case 59.—Another case is that of a man who had a tumour on the surface of the brain, which grew from the dura mater. He had fits, which towards the close of his life occurred in constant succession, but during which he appeared to recognise his wife.

In cases of injury to the surface of the brain convulsions are constantly observed, and perhaps particularly when blood is effused, which becomes entangled in the meshes of the pia mater. Even a simple and spontaneous effusion of blood is sometimes attended with convulsions. My note-book contains the following cases:

Case 60. *Convulsions; injury to the brain.*—A man, æt. 46, was thrown out of a cart, receiving a scalp wound, by which the bone was injured. This became carious, and afterwards epileptic attacks came on. The skull was trephined, and the dura mater was incised. After death the brain was found to be torn, and there was arachnitis around the lacerated spot.

Case 61.—An old man received a fracture of the skull, with laceration of the brain. He was in a state of coma, and had constant convulsive twitchings until he died.

Case 62.—A man was knocked down by a cart, and his head was injured. The scalp was torn and the bone exposed. He continued without any marked symptoms for three weeks, and it was thought that he would recover, when he was seized with epileptiform fits; after this he had several attacks, which ultimately merged into a state of continuous convulsion. He died eight days after the appearance of these symptoms. There was found a local injury to the brain, beneath which was an abscess; this had burst into one of the ventricles.

Case 63.—A man was run over and was injured in the head. He died in twelve hours. There was found an effusion of blood into the arachnoid cavity. He was unconscious, but his limbs were perfectly rigid, or were affected with jactitation.

Case 64.—An old woman was found insensible in the streets, and was brought to the hospital. She had fits in constant succession until her death. No injury to the skull was found; but the brain was bruised, and there was blood on its surface.

Case 65. *Fits; effusion of blood from injury.*—A man, æt. 25, was admitted on October 25th, and died on November 15th. Three days before his admission, whilst in a state of intoxication, he was thrown down and struck his head on the pavement. He was taken up insensible; the wound was dressed, and he was sent home. In the evening he had a fit, which was styled epileptic, and on the following day he had another. On admission he appeared like a man not yet recovered from a state of concussion, and in the evening was very restless, so that he was thought to have delirium tremens. On the following day an epileptic fit occurred, after which he became almost maniacal. Until his death he kept having occasional fits, being in a lethargic state in the intervals. The post-mortem examination showed that there was no injury to the cranium, and that the dura mater was healthy; but when this was removed a layer of blood was seen covering the brain, especially on the right side. The clot was shreddy, firm, and of an ochry colour. At two spots on the base of the brain the coagulum was found adherent to bruised portions of the surface. No inflammatory products were present.

Tumours productive of mania.—In stating that the symptoms of brain disease are dependent upon the part implicated rather than upon the nature of the morbid change itself, I imply that it is not possible, from physiological considerations, to determine whether a tumour or an abscess exists. I have already shown that when either of these involves the surface convulsions may result, and that if the motor tract is implicated, paralysis necessarily follows. Thus, the symptoms of tumours vary with the position in which they grow.

The only anomalous cases which I find are those in which a tumour has been supposed to have been the cause of insanity, or, at least, in which this has been the only morbid condition discovered within the cranium. It is remarkable that in the few cases of the kind I have recorded, the tumour has always been of the cholesteatomatous kind, and this affords some ground for supposing that the disease may have been merely coincident with some other undiscovered change, more immediately connected with the altered mental state of the patient.

CASE 66. *Mania; tumour.*—A man, æt. 26, was admitted into Guy's Hospital with brain symptoms. He appeared partly demented, spoke thickly, and had tremor and general loss of power. Becoming maniacal and unmanageable, he was sent to Colney Hatch Asylum, where he was thought to be suffering from general paralysis. He died at the end of fifteen months. On post-mortem examination a tumour the size of a pigeon's egg was found at the base of the brain; it was imbedded in the pons, cerebellum, and middle lobe of the brain.

CASE 67.—A woman, æt. 35, an epileptic, violent at intervals, prone to mischief, and difficult of management, had a cholesteatomatous tumour, the size of a chestnut, at the base of the brain.

In a case of Dr. Thurnam's brought before the Pathological Society a cholesteatomatous tumour, twice the size of a walnut, was found between the cerebellum and the medulla, in a woman, æt. 60. She had been much altered in character, and was vicious, dirty, intemperate, &c. She hesitated in her speech, and was partially imbecile and slow of apprehension. On the day of her death there was constant convulsion.

Case 68.—A woman died in the hospital with a large tumour in the right hemisphere. It was not seen on the surface; but on slicing the brain it was found to occupy the medullary and to involve the under surface of the cineritious substance; it could be seen projecting into the ventricle. During life she had evidently been suffering under cerebral disorder, but had no definite symptoms. She was in a lethargic condition, and rarely spoke. She, however, talked to her friends, some of whom considered her to be rational, while others said they had for some time thought her to be mad. She was supposed to suffer pain in the head, but made no complaint. She had no paralysis nor convulsions, nor any other symptom indicating a definite local lesion.

In one of the journals was reported the case of a man who had a tumour the size of a small orange, growing from the dura mater into the brain on the left side between the anterior and middle lobe. His symptoms had been headache, drowsiness, irritability of temper, and difficulty in expressing his thoughts, with stammering and imbecility gradually growing upon him.

This case was considered to be of such a nature that the doctors consulted were those who especially practise in lunacy.

I must leave these cases as they stand, without, at present, attempting an explanation of their phenomena.

Disease of the medullary substance.—The general belief of physiologists is that the medullary substance consists of fibres connecting the surface of the brain with the central ganglia; and there is every reason to believe that this view of its structure is correct.

From the experiments made for us in the wards by disease not much can be learned, the symptoms of disease of the medullary substance being generally of an altogether negative character. No paralysis ensues; there is no disturbance of the intellectual functions, or, if present, it consists merely in a general obscuration of the faculties. There are no fits, and in many cases there is no pain. Thus, softening, abscess, or any other change in the medullary substance, although sufficient to produce a general disturbance, may have so few local characteristics that the case may be regarded as one of fever or uræmic poisoning. This I have more than once seen. Should the morbid change be so extensive as to cut through all the connecting fibres between the centre and the circumference, then, in all probability, a form of paralysis would ensue; but the affections generally met with in practice are too circumscribed to produce this effect.

The following cases will be sufficient to show how few are the symptoms of disease of the medullary substance.

CASE 69. *Abscess in the brain; obscure symptoms.*—A woman, æt. 31, came amongst the out-patients, complaining of pain in her head, which was covered with a wet rag. Her tongue was furred and her skin hot, and she appeared like a person at the onset of fever. The next week she came again in the same condition, complaining of her head, and she was then taken in. The case still looked like one of fever, the principal symptom being headache. This subsided, but came on again in paroxysms. She also had difficulty in moving her left leg, and pain in the psoas region. She had no other cerebral symptoms. After her death there were found four abscesses in the hemispheres. They were believed to be secondary to suppuration which existed in the pelvis.

CASE 70. *Abscess; absence of symptoms.*—Three years before his death a man received a severe injury, by which his chest was crushed; he spat blood after the accident; he was not known to have received any injury to the head. He had, however, been subject to fainting fits. Three days before his admission he was as well as usual, when he was seized with tingling and numbness of the left hand. This increased until he came into the hospital, when it was clear that there was much loss of power on the left side. Afterwards he became completely hemiplegic, coma came on, and he died. In the left hemisphere there was a large abscess consisting of a wall of tough lymph, enclosing viscid green pus. This had recently made its way into the ventricle and involved the thalamus; but the cyst was evidently of some weeks' duration.

I have already mentioned cases in which tumours in the medullary substance produced no marked symptoms. Our records contain several others of the same kind.

Diseases of the cerebellum.—With reference to this part of the brain, morbid anatomy has unfortunately as yet taught us but little; the diseases of this organ have not given rise to any symptoms so characteristic as to indicate their seat.

Physiologists infer from experiments on the lower animals that it is the function of the cerebellum to harmonise and co-ordinate the various movements; so that, when an animal is deprived of this organ, it staggers like a man when intoxicated. They believe that without a cerebellum we should still have the power of movement, but that we could not walk steadily, move our hands with regularity, nor eat with propriety. Seeing that all physiologists make positive statements with reference to this matter, it is certainly remarkable that this view is not supported, so far as I am aware, by a single clinical fact. We are, no doubt, acquainted with diseases attended with a tottering in the gait and a want of control over the movements of the body. Such is the disease known as Duchenne's paralysis, which (according to this author's definition) consists in a "progressive abolition of co-ordination of movement, and an apparent paralysis contrasting with the integrity of the muscular force." With this titubation (to adopt the new expression) we should suppose a disease of the cerebellum; but this is not the case. It is the spinal cord which is said to be the part affected in this malady.

It may be that in cases of disease of the cerebellum the patient is too ill to attempt to walk, so that his want of co-ordination escapes observation. But in none of the cases in my books have I observed anything more than a desire to lie quiet in bed, and an anxiety to be let alone, and these are common symptoms in other cerebral diseases. A boy was lately under my care in the hospital with an abscess in the cerebellum connected with disease of the temporal bone; this boy was so weak that he could scarcely stand. The same thing was observed in a girl affected with a similar disease. Hence, if it were simply said that a removal of this organ diminishes the muscular power, no facts gained in the wards of the hospital could be mentioned in contradiction. Dr. Gull had for many months under his care a child with a tumour in the cerebellum; this child was blind, but quite sensible. He was too weak to stand, and answered questions very slowly, the words being drawled out in such a deliberate manner that it seemed as if the end of the sentence would never be reached. In this case there was also large ventricular effusion.

My belief is that in these cases there has been either much loss of power or at least an indisposition to move; and I have never witnessed a case in which the power was present, proper control being alone wanting. In like manner, my cases in no way corroborate another opinion, that the cerebellum is the organ of sensibility. Disease of this part does not in any way affect the intellectual powers. Andral remarked this many years ago with reference to tumours.

Case 71. *Abscess in cerebellum; no other nervous symptom but want of power.*—A boy, æt. 12, was admitted under my care looking very ill and very thin. He was quite intelligent, and had never been otherwise; but he spoke sharply, as if he did not wish to be disturbed. He had been suffering for some weeks from intense pain at the back of his head; this had been called tic douloureux. It was observed that he was deaf on one side, and had a discharge from the ear. He was sick, and his breathing was irregular. He was so feeble that he could with difficulty sit up in bed. He gradually died. An abscess was found occupying the right lobe of the cerebellum.

CASE 72. *Abscess in cerebellum; no symptoms.*—A woman, æt. 26, stated that eight days before her admission she caught cold, and that this was followed by rigors, languor, nausea, &c. She was sent to the hospital, being supposed to have fever; but she had no eruption, and her abdomen was shrunken and her bowels confined. She was quite rational. She gradually got lower, having no marked symptoms until two days before death, when she became drowsy and finally comatose. In the cerebellum there was a large abscess, which was connected with disease of the temporal bone, and had burst into the ventricle.

CASE 73. *Tumour of cerebellum; general weakness.*—A woman, æt. 28, about nine months before her death began to feel some strange sensations in her head with buzzing in her ears, but she had no actual pain in her head. Afterwards her sight grew dim, until at last she became blind. On admission she was also affected with partial paralysis of the left side of her face, and there was a general weakness of the whole body, although she could get out of bed. Her intellect was unimpaired. A tumour, the size of a bantam's egg, was found in the left lobe of the cerebellum, indenting the pons on that side; the seventh nerve was stretched over it.

CASE 74. *Tumour in cerebellum; obscure symptoms.*—A woman was admitted into the hospital a month before her death, but was unable to give any satisfactory account of herself; she had been ill for some months, with pain in the head, loss of memory, indistinct vision, and fits. She was unable to rise from her bed. A large scrofulous tumour, the size of a billiard ball, was found in the right lobe of the cerebellum; it was adherent below to the dura mater. The pons and the medulla were slightly flattened. The ventricles were distended with fluid. There was also general tuberculosis.

CASE 75. *Scrofulous tumours of the cerebellum; want of power.*—A girl was in the hospital for phthisis. Three days before her death she took to her bed, saying her legs were too weak to support her. She had, however, no decided paralysis and no cerebral symptoms. On the day of her death she had convulsions. In the inferior surface of the left lobe of the cerebellum were found two masses of hard tuberculous deposit.

Dr. Broadbent has published two very interesting cases of apoplexy or effusion of blood in the cerebellum, and it will be seen how very little the symptoms were indicative of any special lesion.

One case was that of a girl, æt. 20, who had an effusion of blood in the left lobe of the cerebellum, and died after a few days. She lay on her side, unwilling to be moved or spoken to. She had pain in her head, and seemed as if she suffered from great weariness, and wished to be left alone. She at last died suddenly from rupture into the ventricle.

In the other case, that of a girl, æt. 16, there was an effusion of blood bulging into the fourth ventricle. This girl was found leaning against a wall, complaining of pain in her head. She was taken to St. Mary's Hospital, and was then insensible. She moaned, and could move all her limbs, but resisted all movements made by others, and gave evidence of pain. She died in two hours.

Besides the symptoms of brain disease of which I have hitherto been speaking, there are others of a more general kind. I have already alluded to the fact that disease at the base especially affects the action of the heart and the respiratory rhythm, and influences also the stomach and intestines. Vomiting, however, is a symptom common to many cerebral diseases, and I have known more than one case of chronic brain disease or general paralysis of the insane treated for a long time as "liver." I have notes of a case of tumour of the cerebellum, in which vomiting was for a considerable period the only symptom.

Pain.—I must speak guardedly as to the presence of pain as a symptom of brain disease. It is certain that in a very large number of diseases of the brain proper, pain is absent, and it is equally certain that in many instances in which pain has been a marked symptom the membranes or even the bones themselves have been involved. In order to determine this question, a very careful anatomical dissection of the diseased part would be necessary. Thus, in comparing the symptoms in two cases of tumour of the brain, one attended with pain, the other not, it might perhaps be found that in the one instance the membranes were, and that in the other they were not involved. It may be remarked that in many of the cases of tumours of the cerebellum which I have given above pain was a symptom, but in these instances the growth was adherent to the dura mater. In very many cases of softening of the brain no pain has been present, but in some instances severe headache is spoken of. Further inquiries as to this point are necessary, not only as regards the brain, but also with reference to other organs. I think it may, perhaps, be found that none of the parenchymatous organs have themselves any feeling, and that all painful sensations are to be referred to the parietes of the body. For example, it is probable that there is no pain either in pneumonia or in the pleurisy which accompanies it, the great pain attending a pleuritic attack being due entirely to the fact that the costal pleura is affected in this form of disease. Careful investigation may show, too, that the hollow organs have no ordinary sensation, but are capable only of transmitting painful impressions, indicative of there being something wrong in their interior.

As regards the brain (which is the organ with which we are now concerned), we generally recognise the importance of pain as indicative of disease of the bones, &c. Of this we have an example in cases of epilepsy, when arising from injury or syphilis, in which cases we should naturally expect to find local disease.

It will probably hereafter be shown that great pain necessarily implies that the peripheral branches of sensitive nerves are involved, and therefore that the seat of disease is towards the outside of the brain. Thus, disease of the great mass of the brain would not be attended by pain, and an affection of the central ganglia, or of the grey nuclei of the nerves, would merely give rise to a loss of sensibility or a state of altered sensibility in different parts of the body.

I have already alluded to the fact that in cases of cerebral disease the pupils generally undergo frequent alterations. I think it probable that the frequency with which such changes take place will prevent the condition of the pupils being of much value as regards diagnosis. Certain general facts are known to us all. Thus, the pupils are generally dilated in cases of ventricular effusion; they are contracted when there is effusion at the base, and they are unequal in many diseases. An inequality of the pupils denotes cerebral disturbance, but it does not give any very definite indications; it is seen in general paralysis, and in the so-called *ataxie locomotrice*, as well as in other disorders. It is also of importance to note whether the pupils are or are not sensible to the stimulus of light. The constant changes in the pupils during an acute disease of the brain are sufficient to show how slight may be the causes which affect their condition.

Instant death rarely due to disease of the brain.—I may here allude to the fact (which must every day be becoming more generally known) that disease of the brain does not cause instant death. In other words, apoplexy is very rarely, if ever, a suddenly fatal disease. As a rule, a fatal termination does not occur for several hours after such an attack; and even when the blood is effused into the pons Varolii and the neighbourhood of the fourth ventricle, two or three hours elapse before the death of the patient.

Amongst the reports of several thousand post-mortem examinations I can find only one case in which death was sudden. This was a case of meningeal apoplexy.

CASE 76.—A woman, æt. 67, had been to town on Easter Monday with her family. On returning from the theatre to go home she fell down near the London Bridge Railway Station, and was immediately brought to the hospital. She was then found to be dead. The brain was covered with blood, large coagula existed at the base, and the fourth ventricle was filled with blood. The arteries were much diseased.

In this case it was, no doubt, the implication of parts near the so-called vital point that caused the suddenly fatal issue.

Again, we have daily occasion to observe that persons suffering from the most dreadful wounds of the brain may survive for hours. In such cases it is the exception to find persons killed on the spot, as in the following instance:

CASE 77.—An omnibus conductor, whilst attempting to raise one of his horses which had fallen, received a kick on the head. This occurred at the railway station, and he was immediately carried to the hospital; but when brought in he was dead, although only a few minutes had elapsed. The brain was found covered with blood, and the ventricles likewise were filled. These parts not being at first affected in ordinary apoplexy, the effusion of blood is not immediately fatal.

I have lately seen the case of a man who sent a bullet quite through his brain, but who nevertheless lived a day or two afterwards.

With very rare exceptions, we ought to ascribe sudden death to disease of the heart. But that this opinion is not generally held, is proved by what we read every day in the newspapers, in which sudden death is constantly attributed to apoplexy. The coroner and jury, indeed, seem to go wilfully wrong in this matter; for in the inquest of a well-known person who was shown by a post-mortem examination to have died of rupture of the heart, the verdict returned was one of apoplexy, due to the want of circulation of blood in the brain in consequence of the failure of the heart.[1]

[1] A friend to whom I mentioned this confirmed me as to the popular opinion (which is, no doubt, shared by the less informed members of our profession) by reminding me that in a popular London play one of the characters is made to die suddenly of apoplexy. But this is, at any rate, as near the mark as the idea conveyed in the question once put to me, whether Lord Dundreary did not represent a case of general paralysis of the insane.

Affections of the spinal cord.—Had time and space permitted, I would have referred to some cases of disease of the spinal cord bearing upon the physiology of this organ; but as it is, I will merely express my belief that clinical facts, to a certain extent, corroborate the opinion of Brown-Séquard, that the sensory fibres pass along the middle of the cord. If the medulla is competely severed there is, of course, a complete loss of voluntary movement as well as of sensation. It has, however, long been remarked that a paraplegia affecting motion is much more common than a paraplegia affecting sensation. This is probably due to the fact that disease most frequently affects the circumference of the cord, commencing as a meningitis, or even arising from without in the bones. On the other hand, when sensation is lost the destruction of the cord is generally found to be much deeper and more extensive. We do not often have an opportunity of observing an independent change in the grey matter, but such an affection occurred in the hospital practice of Dr. Gull.

Case 78.—A man, æt. 26, was admitted with complete paraplegia of sensation and motion as high as the hips. He stated that, eight days before, he had fallen on his sacrum across a piece of iron. He was drunk at the time, and was not aware that he had hurt himself. On the following day he had numbness in his legs, but went to work as usual, and continued to do so for four days. He then found that he was unable to pass his water, and sought advice at an hospital for what he called a stricture. He was afterwards attended at home. The numbness of the legs increased until he had lost all feeling in them. He died a month after admission into Guy's. When the spinal cord was removed it presented nothing unusual on its external surface; but on cutting it across at the junction of the dorsal with the lumbar portion, a clot of blood the size of a pea was found in the grey substance; this had undergone a brownish discoloration, and appeared so mixed up with the grey matter that none of this could be seen. This remarkable condition extended down to the very end of the cord; measured upwards from the termination of the cord, the discoloured part was found to be nine inches in length.

Much has yet to be learned with respect to the functions of the cord and the influence of particular parts of it over the structures to which nerves from it pass. It has been stated that in "Duchenne's Ataxie" the posterior columns have been found to have undergone a grey degeneration; but I believe that this has been present likewise in cases in which no such form of disease had existed. In illustration of the connection between the spinal cord and the cerebral centres, I may mention the following case:

CASE 79.—About a year before his death a young man fell on his back; this injury was followed by symptoms said to be due to concussion of the spine. He resumed his work, but he subsequently became affected with paralytic symptoms. Four months before he died he lost entirely the use of his leg; subsequently his arm became paralysed, and his eyesight became affected; his intellect remained clear. After death it was discovered that the whole of the cord was diseased, the affection also extending from the lower part upwards through the pons to the corpora striata; and without going into further details, I may mention that, on section, the cord was found to have undergone great change, containing a translucent material, and presenting what has been styled a "grey degeneration of its substance." This condition existed chiefly on the surface, but in places it had reached the interior. It involved the superficial layer of the pons Varolii, but not its interior. The thalami and corpora striata showed on each side patches of the same translucent material, which, however, did not penetrate deeply.

The exact nature of the influence which is constantly being conveyed by the nerves from the cord has not been ascertained. That some influence is thus conveyed there can be no doubt. It is shown by the difference which exists between a paralysed arm and the arm of a person asleep; and a further proof of it is afforded by those cases in which the division of or injury to a nerve leads to wasting of the limb to which it is supplied. In the case of progressive muscular atrophy it is still *sub judice* whether the disease is due to a primary change in the muscular tissue or to some alteration in the spinal cord itself, leading, not to paralysis, but to wasting. We can quite conceive that, without the sensori-motor tract being interfered with, there may be an affection of a portion of the cord by which some special tonic influence is conveyed, and that such an affection may give rise to the symptoms observed in the progressive muscular atrophy. The pathology of infantile paralysis still remains to be unravelled; and another interesting subject upon which I cannot now enter is that of the influence of the nerves in the production of dropsy, a subject which has been brought under the notice of the profession by Dr. Laycock.

REMARKS ON SOME OF THE FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

By SAMUEL WILKS, M.D.

In the preceding pages my object has been to show that a more scientific classification of nervous diseases may be formed, by connecting their phenomena with morbid changes in the nervous centres. But, at the same time, I am anxious to prove to those who read these papers that I am not among those who expect to find a tangible change, discoverable in the post-mortem room, in every case in which nervous symptoms have been present during life. In reality, the fact that any one has devoted much time to pathological pursuits affords no ground whatever for the supposition that he, more than others, will be inclined to look for organic disease in all cases. It seems, however, that some imagine this to be the case; and, therefore, in order to show that I hold no such views, I propose now to make a few remarks upon certain diseases of the nervous system of a purely functional kind.

I need not enter upon the questions whether changes unappreciable by our senses may not really exist even in those diseases which are styled functional, and whether all phenomena occurring in the body must not necessarily be due to previous alterations in its elemental structures. It may, perhaps, be true that in certain viscera there can be no functional derangement without some alteration which might conceivably be detected by means of chemical or microscopical investigation. But there is no reason to regard the derangements of the nervous system in this light. The nerve centres do not work unceasingly, like the liver and kidneys, but are at one time in full force, at another in a state of comparative rest. The extreme limits of their action are daily seen in the ordinary phenomena of life and health. That a nerve centre should act violently, and that it should cease to act, are conditions which occur in health. Hence, we can hardly suppose that modifications of these changes, constituting morbid states of the nervous system, are necessarily evidences of organic disease.

If I can, by a voluntary effort, produce various irregular movements in my limbs, there seems no reason, when the same movements are altogether unnatural and involuntary, to look for any organic change in that centre whence these movements proceed. So many among the morbid conditions of the nervous system are of a kind completely parallel with ordinary phenomena observed in health, that no organic lesion can be admitted as the cause of these symptoms. This becomes still more evident if we consider that we generally mean destructive changes when we speak of organic lesions. By this term we, as a rule, intend to imply, not only an alteration which can be discerned by the eye, but also such an one as consists in a disintegration of structure. There is at present scarcely any organic change known which does not include more or less destruction with an accompanying loss of function. It seems to me that a mistake must be made by those who think that they have discovered similar changes in the spinal cord of those who have died of tetanus or chorea. When the nervous functions were exalted, we surely cannot suppose that alterations of this kind were present. In cases of depressed function, in which nerve-force is not seen in operation, such organic changes may indeed have occurred. Even in these instances, however, it is quite possible that the centres of power may be merely in a state of passivity. When the machinery is not working it may require all our acumen to discover whether it is simply at rest or is materially deranged.

We do, in fact, recognise an increased or morbid activity of the nervous centres in many conditions which come under the category of disease, without supposing that these centres have undergone a material change; and we also admit that they are passive, or even that there is a temporary suspension of the production of nerve-force in many cases of disease from which the patient perfectly recovers. For example, since the operation of the will is intimately associated with the cineritious substance of the brain, a depression of the function of this part may simply show itself in a failure of the necessary effort to stimulate the bodily organism. An inability to move the limbs may be nothing more than a protracted condition of the ordinary arrest of voluntary effort. On the other hand, a failure in the functions of parts unconnected with the will (such as the central ganglia) would probably imply a condition more nearly approaching to what must be styled an organic change. I may cite, in illustration, the case of paralysis arising from failure of the circulation, as after ligature of the carotid. Here the change in the centre is organic; yet it is not of the kind generally understood by this term, for in a few hours it may be perfectly recovered from.

If we now look to the ganglionic centres of the sympathetic system, we must suppose that their action also may either be exalted or depressed. Thus, the depraved action of the heart, stomach, or other organs, observed in certain diseases, may be due to failure of the nervous influence generally derived from these centres.

Thus, then, without discussing the question as to what conditions are necessary for the occurrence of a functional change, we must admit that the nerve centres (or those parts whence power proceeds) may be exalted or diminished in function without having undergone any of those visible alterations which we usually style organic; and we must also allow that these altered conditions may either be temporary and speedily recovered from, or followed by death in a direct or indirect manner; generally in the former mode when the function is exalted, in the latter when it is depressed.

These remarks with reference to the increased or diminished power of the nerve centres apply both to the cerebro-spinal and to the sympathetic system. In the former the exalted or the failing action is generally easily recognisable, whereas in the latter it is much less easily ascertained. For since the sympathetic or vaso-motor nerves rule over all the organs in the body, it is not always easy to recognise the influence of these nerves only. For our present purpose we need not ask whether this influence is of some occult kind, at present not understood, or is merely some force regulating the blood-vessels. In the case of the secreting organs it may be simply the latter; but in that of the heart it seems something more direct, for, according to physiologists, if the nervous ganglia be still attached to it, this organ will not only continue to beat after its removal from the body, but after it has been cut to pieces each segment containing such centres will contract. Our examinations in the post-mortem room have not yet been carried to the extent of investigating organic changes in these ganglia. But there can be little doubt that this is a wide field of inquiry. When the heart suddenly ceases to beat, and yet no disease is to be found within it, it is quite possible there may in reality be important alterations in the sympathetic ganglia. The same thing may be said of other organs, such as the stomach. We know that a dyspepsia may be due to a nervous cause; but it may hereafter be found that there is, in this condition, an actual alteration in the nerves, although the organ, to the naked eye, presents the appearance of health. In Addison's disease the semilunar ganglia, as well as the nerves proceeding from them, are involved in a morbid process; and associated with this we observe sickness at stomach and a remarkable depression of the system.

In women at the climacteric period we see the effect of this lowering of the nervous influence in the fluttering of the heart, and the sickness, sighings, headache, &c. Probably no other invalids really feel so ill as these patients; the whole bodily functions are disturbed, and consequently a depression is experienced far exceeding that which accompanies any real organic disease. Such patients describe their feelings with the utmost despondency; they experience successive changes of temperature, which they style flushes of heat; they complain of anorexia, of flatulence, and of irritation of the bowels, uterus, or urinary organs. In fact, there is not a single viscus which does not suffer disturbance, so that every disease in the nosology may, in turn, be supposed to be present. Why one organ should suffer more than another, or why a morbid sensation should be experienced in this part of the body rather than in that, is probably to be explained by the anatomical distribution of the nerves; but we certainly find that, probably owing to the large supply of the sympathetic nerve in the abdomen, greater depression is experienced in abdominal than in other forms of disease, and that in all low conditions of the nervous system morbid sensations are very often referred to this region of the body. Thus, we cannot but contrast the cheerful disposition of the phthisical patient, when on the brink of the grave, with the depression observed in one who has but a temporary disturbance of his stomach, liver, or bowels; and another indication of the same fact is the placing of the emotions in these parts, as expressed by the term "bowels of compassion."

Under the most varied conditions, both in men and women, when life is low, a number of morbid sensations arise. In a state of health man should be as happy and joyous as the lark flying in the heavens; he should have a keen sense of animal enjoyment, and he should feel nothing of the working of the machinery within him; but when his nervous system is depraved he becomes conscious of all these movements, he feels his heart beat, his head throb, and his back ache. A study of these nervous symptoms would probably show in what order they appear; I think that they commence, especially in women, with a pain in the left side, and that this is followed by pains on the top of the head and in the back, at the epigastrium, over the collar-bones, &c. I feel uncertain whether these pains are altogether subjective or due to some prior alteration of function in the parts whence the pain proceeds. This question, however, is one of very great importance in practice; for we often find that, by attempting to relieve symptoms, we gain no headway towards overcoming disease, whilst by altogether disregarding them, and having recourse to a general tonic plan of treatment, we can ensure a cure within a certain period. At the same time it cannot be denied that the application of remedies to the spot to which the morbid sensation is attributed is frequently attended with success. Thus, plasters to the side, sedatives to the stomach, &c., do give relief. Relieving the local symptoms in this way is not, however, incompatible with a treatment directed to the restoration of the nerve centres themselves.

In practice, we have almost every hour of the day to endeavour to discover whether morbid sensations in and disturbances of the viscera are due to an organic cause or to the mere failure of the regulating power of the nerves; that is, whether the disease is organic or functional. In such cases the diagnosis is doubly difficult in females, because symptoms resembling those of almost every form of disease may be produced by their more delicate nervous organization.

We can imagine, by way of analogy, that in the case of a steam-engine working irregularly we might for a time be at a loss to discover whether the derangement was owing to some material deficiency in the valves or joints, or whether it was due simply to an irregular supply of steam. Or, again, we can picture to ourselves a clock perfect in all its parts moving too slowly, from the simple fact of the weight having nearly run down.

In the cerebro-spinal system, again, an exaltation or a depression of function is constantly witnessed. Such conditions are observed in chorea, in hysteria, and in various passions of the mind. Müller speaks of the nervous principle in the medulla oblongata as being in a state of tension and always ready to act, and he says that the slightest change in its condition excites a discharge of nervous influence, as is manifested in laughing, sneezing, &c. Thus, every mental impulse to motion disturbs the balance of this tension and causes a discharge of nervous influence in a determinate direction. He also compares the nervous system to a musical organ, with its bellows charged and ready to force a stream of air in any direction, according to the particular key that may be touched. Using this illustration, we may imagine the air either to rush out with a scream, or to be hastily allowed to pass off by the larger tubes, or to be diffused melodiously through a series of musical pipes. In a similar way the superfluous nerve-force may display its operations in various ways, according to the sex, age, and temperament of the patient. For example, I have seen the same cause produce hysterics in a mother and chorea in her child, the one disease being almost peculiar to the adult period of life, the other to childhood. The same fright which excited so great an amount of nerve-force in the mother as to cause the explosion known as hysterics operated on the child in a slower manner, and gave rise to the less violent action known as chorea. The spinal system was excited to over-action by the cineritious substance above, which had been unduly stimulated by a mental shock, and remained temporarily impaired until the disease was cured. The explosion of nerve-force by an hysterical attack acts as a kind of safety-valve, protecting the internal machinery from danger; and although all are not alike impressionable, there is scarcely an individual who may not be in need of it when acted on by a sufficiently powerful stimulus. Even in the strong-minded Napoleon a fit is said to have been excited by passion. More commonly, however, relief to an over-excited nervous system is afforded by laughing or crying. Thus, as Byron observes, the power which women possess, as compared with men, of being able to pour their troubles into their pocket-handkerchiefs, is no doubt often very beneficial to them, so far as their health is concerned. A woman who is excited, if she do not go into hysterics or have a good cry, often allows the redundant nerve-force to escape through that unruly member the tongue, and thus an extreme volubility of utterance perhaps saves her from further unpleasantness. Of course, the talk which flows from her lips is altogether different from the result of an intellectual process; and thus it is still true now as it was in ancient times, that "anger is a short madness." In Switzerland, last summer, I met an Irish gentleman, who told me that he could make his wants known without a knowledge of the language, but that when he was irritated and wanted to swear he would sometimes give all he possessed to understand German. In other cases, again, the superfluous force escapes by the limbs; thus, an angry person slams the door, or destroys even her own property.[1] A man of better sense, when vexed, takes a walk, and thus gets rid of his extra nerve-force; or, if the irritation and its results are more chronic, sits down, takes up his pen, and by publishing "the whole correspondence" eases his mind.

[1] An example I find in the newspaper whilst writing this:

"A young man, a gardener, came before Mr. Paget, the magistrate, to ask his advice. He had been married five months to a woman who had turned out to be a great shrew, and who, acting under the advice of her mother, had broken up her home three times. On Tuesday afternoon he was engaged in a gardening operation, which he could not complete by dinner-time. There was a delay of an hour, and when he did come home he was soundly rated by his wife, who commenced breaking the plates and dishes, destroyed the furniture, and made a general wreck of the place; she then attacked him," &c.

Should this tension of the nerves not be lessened in any one of these various ways, it is very likely to react on the bodily functions, and may thus produce injuries as serious as would the pent-up steam in a boiler, even amounting to the destruction of the individual who is the subject of it. This is a popular piece of pathology, as I lately learnt from a woman who brought her son to the hospital affected with fever, and who said that his illness arose from a fright caused by a dog, but that probably it would never have appeared had the beast's master allowed the lad to go to the water-closet, as he desired. It is well known that pent-up grief brings its evils with it; but although every medical man must have seen instances of this kind, they are to be found recorded in tales of fiction and in books of poetry rather than in works treating of disease. Of all these functional derangements of the nervous system we shall find more illustrations in the writings of the poets than in those of medical authors. Thus, Tennyson, in his 'Princess'—

> "Home they brought her warrior dead.
> She nor swooned nor uttered cry;
> All her maidens watching said,
> 'She must weep, or she will die.'"

In the female sex, again, unavowed love may be the cause of a fatal chlorosis. We may, then, say, as Viola said to the Duke, of her whose history was "a blank:"

> ". . . . . . She never told her love,
> But let concealment, like a worm i' the bud,
> Feed on her damask cheek. She pined in thought,
> And with a green and yellow melancholy,
> She sat, like Patience on a monument
> Smiling at Grief."

That distress of mind may lead to fatal consequences is no poetical fiction. I have more than once witnessed it, and every medical man must have seen the same thing. The late Sir H. Marsh related the case of a young lady who accidentally poisoned her father by giving him laudanum instead of a black draught. This occurrence so preyed on her mind that she took to her bed, became anæmic, and before many months had elapsed died, without any apparent organic disease. Not very long ago I witnessed a melancholy case in which death seemed to be due simply to a similar cause. A young lady, of high intellectual attainments, had, in an unguarded moment, been seduced by a former lover, whom she accidentally met. Finding herself pregnant, she became the prey to remorse, not so much on her own account as because she was bringing disgrace on kind friends. She took to her bed, refused her food, and began to waste away. She daily grew weaker and weaker, and died of exhaustion before her shame had been proclaimed to the world. The issue of her case realised the story in the 'Lady of the Lake,' of the mother of Brian the Hermit, who was said to have conceived by some supernatural process, and died as she gave birth to her son:

> "All night in this sad glen, the maid
> Sate shrouded in her mantle shade;
> —She said, no shepherd sought her side,
> No hunter's hand her snood untied,
> Yet ne'er again to braid her hair
> The virgin snood did Alice wear;
> Gone was her maiden glee and sport,
> Her maiden girdle all too short,
> Nor sought she from that fatal night,
> Or holy church or blessed rite,
> But locked her secret in her breast,
> And died in travail, unconfessed."

Instances such as the one we find clothed in poetry in these lines are seen in everyday life, and must not be ignored by the physician.

The most remarkable case which I have witnessed of a person dying from grief was one which I lately had an opportunity of seeing with Mr. Brown, of Lewisham.

CASE 1.—*Mental shock; death in five weeks.*—Two young ladies, residing with their widowed mother, were most devotedly attached to one another. The younger died rather suddenly of disease of the heart. The elder was, for the moment, like one thunder-struck. At first she could not realise the calamity by which she was afflicted, but she soon saw the event in all its terrible reality. She never shed a tear. She declared that, her only object of affection being gone for ever, she would go seek her sister in another world. She then arranged the whole funeral ceremony for her sister, and chose the grave in a neighbouring cemetery. Almost immediately after returning home she began to suffer from palpitations, sickness, and pain over the region of the heart, as her sister had done. She would eat nothing, and declared that she had her sister's complaint, and should shortly follow her. There was no reason to suppose that any disease existed; in fact, the disturbance was clearly functional, and, as she herself declared, was produced simply by emotion. She was a well-grown, healthy girl, and I had no fear that her illness was due to anything more than temporary excitement. However, I failed to gain the co-operation of the friends to have her removed from the scene of her trouble; for they not only sympathised with the girl, but agreed that her case was the exact counterpart of that of her sister. I, on the contrary, regarded her symptoms simply as the result of good acting. However, in spite of all the influence that could be used, she would not be comforted; she refused her food, and rejected what was given to her; and, at last, much to my horror and surprise, she died in a kind of hysterical convulsion, exactly five weeks after her sister, and was laid in the same grave. Thus, by her own will, she attained the object of her fixed determination.

We have all heard of the celebrated comic actor who, consulting a physician for depression of spirits, was advised by the doctor (who was ignorant of the name of his patient) to go and see Grimaldi play; upon which the patient retorted, "Alas! I am that unhappy man!" In such a case it is not improbable that the very excitation of the brain necessary to produce the humorous result to be appreciated by others might, from not finding the ordinary channels of outlet, have produced a reaction affecting the nervous system of the actor himself. Had he laughed with his audience, much of the humour might have been wanting, but he would, perhaps, have suffered less.

A repressed nervous force or emotion must be regarded in altogether a different light from the mere absence of such force or emotion. In the former case a spirit is roused which cannot be allayed, and which, if not allowed to work in the proper direction, will seriously injure the individual in some other way. Though apparently not acting, it is silently working mischief within. Not so the dormant will, which has never been roused. In this instance there is a mere passivity of brain; there is no development of nerve-force, the will is in abeyance, and the disease is usually styled hysteria. This term is applied to many functional nervous affections. In cases which receive this name we often find the different organs and functions, including the uterine, in perfect order; the structure of the body is in a normal state, and so is the spinal nervous system which rules over it; but the brain is inactive, and there is a want of will to exercise the necessary controlling power. The patient, perhaps, feels an inability to move one of her limbs, although this is well nourished, and though the nervous supply to it is perfect; the machinery is good, but the force is wanting to drive it. The remedy must, of course, be such as will rouse the dormant will, and necessarily consists in moral treatment. For, adopting another course, and making the patient swallow mixture and pill at stated intervals, would be less likely to cure her than using the dances and charms of the medicine-man of the North American Indians. The latter means, indeed, might produce a mental shock, which could not be effected by the former. The physician who treats each of the innumerable symptoms of which such a patient complains is pandering to her complaint and strengthening its hold upon her. The only rational method by which the will can be roused is to gain influence over the patient, instead of succumbing to her. When this is done, the cure is only a question of time. Each case must be treated on its merits, and according to the temperament of the individual; sometimes by severe lecturing or by threats, sometimes by persuasion, sometimes by an appeal to the reason, sometimes by making her see that one is convinced that it is quite possible for her to recover should she choose. Such cases are almost always avoided by medical men, because of the trouble which it gives to adopt any measure beyond writing prescriptions or sending out physic. We have much better opportunities of treating these patients in hospitals, where they are removed from all home influences and placed under strangers, to whom they must submit. I am always ready to take such cases into our wards, having seen the immense good which admission into a hospital can do them, and from which the richer classes are debarred. I could enumerate many striking instances of the benefit thus conferred, but I will content myself with the mention of two such cases. The first case is one of which an account was published last year in the weekly journals, the second is that of a girl who has just left the hospital.

Case 2.—*Hysteria; patient bedridden, with loss of speech, for a year.*—Elizabeth P—, æt. 22, the daughter of retired trades-people in Bermondsey. Having gone to the house to visit the father, I was asked to see the daughter, who had a spinal complaint, and had been given up by the doctors. About two and a half years before, she had been apprenticed to a dressmaker, after which her health became impaired. She kept growing weaker, until she took to her bed, from which, when I saw her, she had not risen for more than a year. About six months before, her speech had begun to fail, and since that time she had never uttered a word. I found her lying in bed, in very good condition, and not wasted like a patient affected with organic disease. When spoken to, she answered merely by a nod, all her correspondence being carried on by means of a slate. She spent her time in executing elaborate embroidery, and her bedridden condition had excited much sympathy in the neighbours and friends. I was told that her spine was diseased, and that the slightest movement sent her into a fit. However, I had her moved, upon which she threw her head back, closed her eyes, and appeared unconscious. I found that she had a lateral curvature. Without giving further details, I may say at once that I saw it to be a case of hysteria, and promised to cure the girl if she would come to the hospital. This was refused at the time; but after a week or two her friends waited on me, and requested me to take charge of her. She came into my ward, accompanied by her slate and pencil, worsted work, and needles, and, being placed in bed, commenced the old plan of operations. I then, in the presence of my clerk, began to talk seriously to her, upon which she closed her eyes and went off in a pretended fit. I, however, continued my conversation, informed her that I was perfectly aware she could talk if she chose, and that if she did not daily improve I should denounce her to the students as an impostor. My clerk took a report of the case, in which it is stated that at first she was lifted out of bed by two nurses to have her bowels relieved, her body being extended stiffly and her legs perfectly rigid. Her slate was taken from her, and then she moved her lips, as if unable to speak. With a great deal of pains and by using threats of galvanism (which she did not like), we got her, at the end of a week, to say "yes" and "no" in a whisper. After this her voice returned, and next she began to move her legs. She was then taken out of bed, dressed, and placed in a chair, and was constantly made to move her legs. But when we tried to make her walk she dragged the legs as though they were powerless. She afterwards, however, became capable of standing, and then of walking. She left the hospital, and at the end of three months after leaving home had walked a journey of three miles. She has since entered a shop as book-keeper, and is now well.

Case 3.—*Hysteria; patient bedridden, with loss of speech, for four years.*—Eliz. B—, æt. 28, was living at Camberwell. I was requested to see her by Mr. Hindle. Her illness had commenced about four and a half years before, with sickness, pain in the chest, &c. While attempting to get out of bed she lost the use of her legs. She has kept her bed ever since, but her symptoms have undergone some changes during this time. Fifteen months ago she lost the use of her hands, and became unable to hold her water; but these symptoms abated. Ten months ago she was seized with pains all over her, and suddenly lost her voice. Thus she had been bedridden for more than four years, was quite unable to move her legs, and for ten months had not spoken. The catamenia were natural. She had seen many medical men; her head had been shaved, various applications to the spine had been used, and the whole pharmacopœia had been ransacked in vain. It did not seem very clear how physic was to rouse her moral nature, but it was quite evident that such treatment made all her ailments more real to her. Like the other patient, she also communicated all her wants by writing on a slate. She occupied herself in fancy needle-work. She was visited by the benevolent ladies of the district, and the clergyman's portrait hung at the side of her bed. Like the other girl, too, she was fond of religious books. I informed her mother that, if the girl was allowed to come to the hospital, I believed I could cure her, and, on April 7th, 1866, she was admitted. She was put into bed; she had no power over her legs, and moving her out of bed produced sickness, and a violent pain through the body, reaching to the tongue. I talked to her, telling her that an effort on her part was necessary for the cure, that she was to be galvanized, and that I should expect some improvement every day. She wrote that she did her best, and was anxious to get well, but that once, on an alarm of fire, she had felt that she could not move, but would be burnt in her bed. My clerk was very assiduous in the daily use of galvanism; and on the 24th she said, "Oh dear, yes." After this she talked slowly, and thus her speech returned. In the beginning of May she was taken out of bed and dressed; but she was quite incapable of moving her legs. In the middle of the month, however, she moved them slightly, and by the end of May she could stand, leaning on a chair. During the next month she gradually though slowly improved; and in the commencement of July she walked about, pushing a chair before her. Afterwards she gave this up, and in the middle of July, being able to walk out alone, she left the hospital cured. I believe that the galvanism acted on her mind, and was useful by accounting for her favorable progress, without the necessity of attributing it to her own volition.

These are merely types of very common cases in which the will is in abeyance, and has to be roused by appropriate means. Such means as those above mentioned can scarcely be adopted in the upper and middle classes of society; and this is why treatment fails. A melancholy example of this was long under my notice.

CASE 4.—*Hysteria* (?); *starvation and exhaustion.*—A young lady, a patient of Mr. Cornish, at New Cross, was seen by me about three years ago. She complained of pain in the abdomen, constipation, and various other symptoms which I considered to be functional. She did not eat much, and was sick. All remedies failed to relieve her, and she was taken to see several medical men, all of whom regarded the case as nervous. At the end of a year or more she began to keep her room, and said she could eat nothing. What little she did take made her sick, and her bowels were obstinately constipated. She then became thin, this being the effect of her abstinence. All her symptoms became aggravated, and at last she went several weeks without a motion. Mr. Cornish, by means of enemata, removed a large number of scybala. She lay on her back, would eat nothing, and merely nibbled a biscuit, and she drank wine by drops, being sick if she took more. She never afterwards had her bowels opened naturally; but some weeks later, hard masses were again removed from the rectum. The wonder now was that she could live so long on so scanty a diet. Putting together the constipation, sickness, and inability to eat, I concluded that there must be some cause of partial obstruction in the small intestine; and I renounced my earlier opinion that the case was nervous, or one of hysteria. More than two years after the occurrence of the first symptoms she died rather suddenly. Hearing of her death, I was most anxious to have an examination, which, after much trouble, I obtained. I found the body fatter than when I first saw her, and heard that she had lately eaten more food. I examined the interior of the body carefully, but failed to find a particle of disease. The intestine was healthy, and was not constricted at any part. In fact, to my surprise, I found absolutely nothing to account for death. I was therefore fain to return to my original opinion, that the disease was in the first instance imaginary, and that the girl had actually killed herself by her own wilfulness. I never heard that any moral cause had been in operation.

We have seen that the hemispheres of the brain may remain passive, and so fail to supply the nerve-force necessary to keep the body in activity; and it would seem that they may likewise be over active, and afford an unusual amount of this force—that is, that by a great mental effort an actual increase of power may be produced. Herein lies one of the most difficult problems which has ever been considered by the physiologist or the metaphysician; for if we believe that a change in the brain can cause a disturbance of the mental functions, it seems an anomaly to speak of the will as being productive of an increased physical force. The connection between mind and matter may be a mystery; but we, at any rate, know that in our mental activity we are conscious of exerting power, and therefore it seems as if the will and the action were respectively the antecedent and the consequent. It would be a parallel case if we not only found disease of the heart causing palpitation, but also observed palpitation setting up an altered state of the heart. I need not now discuss this question, for there is little doubt that what we call a mental effort is capable of producing a true nerve-force, which is instrumental in bodily acts. We have everyday evidence, in the remarkable differences in the power of withstanding disease possessed by different individuals, that the mind not only acts on the body, but does much more in developing nerve-force. Whilst one person succumbs, another conquers his malady by mere force of will. Nothing is more worthy of study than are the differences in the temperaments of patients in this respect. One man, after receiving a trifling hurt, declares he shall die, and his anticipation kills him. Another, of greater energy of purpose (as in an instance I have lately seen), declares that "he will get well, and cheat the doctors." These facts are so well known that the only difficulty is to select cases in illustration; but it will be sufficient to say that a mental effort can probably supply an actual nerve-force to the bodily organism. The thought of home with the mother and little ones has often sustained the soldier in battle, and has knit his arm to one more decisive blow. As our Poet Laureate says in the 'Princess:'

> "Thy voice is heard thro' rolling drums
> That beat to battle where he stands;
> Thy face across his fancy comes,
> And gives the battle to his hands;
> A moment, while the trumpets blow,
> He sees his brood about thy knee;
> The next, like fire, he meets his foe,
> And strikes him dead for thine and thee."

It is, however, important to distinguish between the capability of creating nervous force in this way to overcome an obstacle and that of resisting untoward circumstances by mere inertness. These two kinds of power indicate different temperaments. Thus, the man who from mere stoicism bears trouble or physical pain may be possessed of no great nervous power, whereas the most sensitive person may have immense moral courage. I think it is John Foster who relates the case of an officer who resigned his commission lest he should disgrace himself by cowardice; this man had no physical courage. I remember the case of a man admitted into the hospital with obscure head symptoms; there being some evidence of a blow, he was asked whether he had received any injury. At first he said that he had not, but he afterwards owned that he had had a slight knock on the head; he endeavoured to make light of the affair, but it was evidently more serious than he would admit, and at last he died of inflammation of the brain. It was found after death that he had an extensive fracture of the skull, and that it was caused by a violent blow with a poker. I may contrast with this the case of the late Sir R. Peel, who had nerve and courage enough to stand up in the House of Commons, and to discard his old opinions and convert his party into enemies; yet, so delicate was his nervous organisation that, when he fell from his horse, he never rallied from the shock, and in a day or two succumbed to an injury which some men would almost have disregarded. In this respect the influence of race appears to be very great; for there is no other cause to which we can attribute the remarkable fact related by the Indian surgeons, that under a supposed mesmeric influence the Hindoo will undergo the severest operation without evidencing pain. In some other nations the same thing has been observed. Mr. Palgrave, in his recent 'Narrative of a Year's Journey through Central and Eastern Arabia,' says—"What is really remarkable among them is a great obtuseness in the general nervous sensibility. On more than one occasion I had to employ the knife or caustic, and was surprised at the patient's cool endurance. While at Riad a young fellow presented himself with a bullet lodged deep in the fore-arm; it gave him some annoyance, and he insisted on having it cut out. The operation was, from my inexpertness, a difficult one; the muscular fascia had to be divided down to the bone. Meanwhile the Nejdean held out the limb steady and inflexible, as though it belonged to a third party, and never changed colour, except it were a flush of excited pleasure on his face when I finally drew out the ball through the incision and placed it in his hand. After a short interval of bandaging and repose he got up and walked home, carrying his leaden trophy along with him. Much similar I saw and heard. The Arabs are not a nervous or excitable race."

In the cases of which I have been speaking it is often difficult to determine how much is due to an effort of the will, how much to a mere obtuseness of the nervous system. When the nerves have received a violent shock it is probable that in some cases the will is paralysed, whilst in others the centres associated with the spinal system are most probably temporarily affected. If the will may be inefficient to move a machinery itself perfect, there is every reason to conclude that the automatic apparatus itself may for a time be similarly affected. It sometimes happens that in hysterical or highly susceptible women all nervous power seems for a time to have disappeared. In such cases there might be a doubt whether anything more than the volitional power is in abeyance, were it not that the total absence of sensation affords a proof that there has really been a shock to the centres themselves.

CASE 5.—*Nervous shock; anæsthesia.*—Some time ago I was asked to see a girl, æt. 15, in consultation with Mr. Fisher, of Deptford. She was excitable, and had lately been much impressed by some powerful sermons at her chapel.

One evening, coming home late from school, she said that she had been frightened by a man, and fell into a strange mental condition, on account of which the doctor was sent for. On the following day I also saw her. She was lying in bed, and seemed not to be aware of what was going on around her. In fact, she was almost in a maniacal condition. Her face and hands were constantly affected by choreal movements, which at times became almost tetanic. It was her menstrual period. She afterwards became calmer, and her condition became more like that of patients affected with chorea. When spoken to she appeared to understand, but she spoke with difficulty exactly as in that disease. The most remarkable feature in her case was the perfect anæsthesia which existed. I was able to thrust a needle deeply into the skin without her feeling it in the least. I tested this in many ways, and was convinced that sensation was altogether absent. She remained in much the same state for a fortnight; at which time she stated that she had been frightened by a man who had accosted her, and that she was then constantly seeing him in the corner of the room. At the end of three weeks I saw her again; she then walked with difficulty, but sensation had quite returned. It seemed that this girl, with her peculiar organisation, had received a shock which, for the time, produced a general paralysis of motion and sensation.

CASE 6.—*Nervous shock; anæsthesia, &c.*—A few months ago I was called to Surbiton, by Mr. Coleman, to see a young lady who was said to be in a trance. Like other members of her family, she was of a nervous temperament; but she had been well until two days before, when her father died quite unexpectedly from apoplexy. She then had an hysterical attack, in which she threw her arms about, clenched her hands, stared wildly, and talked incoherently. She afterwards became quiet, and was put to bed. She then remained perfectly motionless, and on the third day I saw her. She was lying in bed, like one in a tranquil sleep; her breathing was quiet, and her pulse feeble. Her eyelids were closed; but when they were opened the pupils were seen to be natural. We attempted to make her drink by pouring some fluid into her mouth; she retained it for a time, but presently made a convulsive effort to swallow, and threw her arms up. Her condition approached very nearly to that observed in catalepsy, for her arms would remain in whatever position they were placed. She had lost all sense of feeling; but it was not very clear whether this was a true nervous anæsthesia as in the preceding case, or merely the result of unconsciousness. I at once removed all the alarm felt by her friends, by attributing her condition to a nervous shock. In this opinion I was confirmed by being asked to see a married sister, who had come to the house, and whom I found lying on a sofa with spasmodic chokings in the throat. In a day or two my patient came out of her trance, and then I was hurriedly sent for again, on account of her having lock-jaw. I found her with her teeth firmly clenched, so that no food could be got into the mouth. This was overcome by a little management, and she quickly recovered. It was remarkable that the married sister declared that she did not remember me, and had no recollection of what had occurred on the evening on which I first saw her.

In illustration of the influence of peculiarity of temperament on the nervous susceptibility, I may mention the cases of two sisters, to see whom I was called out late one evening, and who were said to be in fits. One of them was married, and had just arrived from Australia. Both of them were in hysterics; I do not know whether this arose from the pleasure which they felt in again seeing one another, or from some other exciting cause. One sister had the ordinary hysterical attack, but the other had an occasional convulsion, in which her limbs became rigid; she clenched her teeth, and seemed for the time wholly unconscious.

It must not be supposed that such complaints are peculiar to the female sex; for we occasionally see young men affected with symptoms to which we can give no other name than hysteria. I have had in the hospital two such cases, in which even the hysterical choking has been present.

Amongst the functional nervous maladies which occur in the male sex there is none of more importance or more deserving of our attention than the form of hypochondriasis founded on an imaginary spermatorrhœa; for this disease often ends in melancholia, and is probably a more frequent cause of suicide than is generally supposed. Such cases are the most perplexing and troublesome which come before the medical man, especially as he is often unable to discover whether the depressed condition of the nervous system is the cause or an effect of the sexual disorder. The numerous charlatans who treat these affections, having only their own interest in view, of course insist that every imaginable complaint of the patient is dependent on spermatorrhœa; but I think that even more legitimate writers, such as Lallemand, have exaggerated the influence of this affection, in producing the numerous evils of which they suppose it to be the cause. It seems to me that this writer must too often have taken his tone from the deplorable accounts of the patients themselves, without reflecting that the story might very often be exactly reversed. If Lallemand's description is correct, every hypochondriacal patient who waits upon us, and gives us a minute account of the state of his sexual functions, is really imparting to us the cause of all his troubles; in fact, this author states that hypochondriasis generally has spermatorrhœa for its cause. I cannot but think that this is an error, and one of great importance. For if this view is correct, our attention ought to be directed mainly to the genital organs; whereas if it is incorrect, we cannot too earnestly endeavour to keep the patient's mind from the imaginary source of all his troubles. I believe that in the different nervous affections, including epilepsy, in which such a local cause of irritation does exist, its detection requires great acumen and perseverance on the part of the doctor, because the patient, of his own accord, says nothing about it. Whereas in the class of cases of which I am now speaking every symptom referable to these organs is noted down by him exactly as in the books to which I have alluded. Indeed, it is remarkable how alike are these descriptions; how the patients dwell upon their want of virility, their hanging testes, their thick urine, and the palpitation and general nervousness from which they suffer. When questioned, they often state that they do not have an emission more than once in two or three weeks; and in many instances I have seen the patient rapidly recover when assured that this is of no importance. Even should the occurrence have been more frequent, so that the patient asks for medicine to stop it, I have found that the administration of tonics, and the use of shower-baths, aided by a few moral directions, have been most successful in satisfying his mind. I know no class of cases in which a medical man does more essential service. In the first place, the patient should be made to deliver up any book which he has been reading on the subject; and this should be thrown into the fire in his presence, or should be destroyed in some other degrading manner; and then his confidence must be gained, and an ultimate cure must be promised in a few words of encouragement. If, on the other hand, the only object of the doctor be to fill his own pockets, these patients become a ready prey.

As I have already said, I believe that the symptoms described by Lallemand as dependent on spermatorrhœa are only such as may occur in any form of hypochondriasis. Hence the diagnosis is sometimes very difficult. Quite lately a young man came to me and informed me that he had spermatorrhœa, that he had taken much medicine in vain, and that he had at last been cauterized, but without effect. He told me that he had seminal emissions; but on careful inquiry I could not ascertain that this was really the case. He was convinced of it, because he felt languid in the morning, because his urine was thick, and because a nervousness would sometimes come over him when in society, so that he was obliged to leave the room. I told him that his disease was imaginary, but that if he wished it I would examine his urine. Having placed some of it under the microscope, I assured him that no spermatozoa were to be seen. He smiled, and said that that was no test, for the seminal ingredients or elements might pass away before the spermatozoid was formed. I charged him with having read Lallemand's work, which he said he had not done. But his description of his own condition tallied so closely with some of the professor's cases, that I confess to have had no answer for him. I could merely inform him that I considered him to have an imaginary complaint, and he still remained convinced that his vitality was daily escaping from him.

From the general character and temperament of the persons who come before us complaining of this disease, I feel convinced that in many instances it is of a purely nervous or hypochondriacal kind. To treat it as a reality would be to strengthen the patient's idea as to the nature of his case, and sometimes, I believe, to lead him to self-destruction. The hypochondriacal state of the patient is shown by the minute attention which he pays to himself, by his examining his urine, and by his looking at his tongue in the glass. The fact that a person watches the state of his tongue is in fact one of the best signs of hypochondriasis. These patients, fearful of forgetting some of the particulars, often write down all their feelings on paper, and give the description to the doctor to read. In hospital practice this is very common, especially as these persons do not like to expose their troubles to strangers. Such documents are so characteristic, and give so good an account of the cases to which they refer, that I have not hesitated to transcribe two or three of them. I have chosen for this purpose those written by hospital patients, because these persons are more likely than others to give expression to their real condition. It will be seen that the first of these descriptions was written by a man of some education; the others were given to me by illiterate patients.

CASE 7.—*Hypochondriasis.*—"I am now suffering from general debility, frequent seminal emissions, not the result of irregularities or abuse, dull pains in the back and loins, and inside the thighs. Occasionally sharp pains at the lower part of the belly, irritation about the bladder, the genital organs have become shrivelled to much less than their natural proportions, the testicles flabby and disorganised, the left being lower than the right. Lately (last week or so) a frequent desire to pass urine which is voided in small quantities without affording the usual relief; it has a strong smell like that from a bladder; the urine appears whilst voiding a straw colour, but left in the vessel, till about a quart, it presents a yellowish-brown. From the age of sixteen to twenty-one there was almost always a thick ropy appearance, and a sediment of a whitish look, frequently a corrode on the side and bottom of the vessel of a reddish colour. Occupation, a draper's assistant; diet plain, beef and mutton; beer, about a pint, half at dinner, half at supper. Bowels regular, slight degree of flatulency; breath rather unpleasant. Tongue furred in the morning, never rise refreshed from sleep, always with a feeling of lassitude, memory bad, and unfit for the least exertion; no desire for society or the least enterprise, perspiring nervousness about anything particular, low spirits, trembling at times, scurf on the head and face, sometimes also irritating pimples on the face. Many of the above descriptions will be unnecessary, but from my ignorance of their nature I thought it best to explain all.—W. S—."

CASE 8.—*Hypochondriasis.*—"Kind Sir, Please to give your kind consideration to my case or i am a loast man for my sufferings as Been for a long time and i have Been Brought so weake Before that i could not whalk without going By the Whal and my voice so that i could not scarseley spake and lost all veryil power for a many months and am so a gaine my testecles feel as if they where down to my kneese and like a peace of cord that whas untwisted and paine me very much and when i cough i feel something strine from me or when i exert my selfe the least i am very weake and soone out of Breath and tremble very much i hope something may Be done for me Beging your pardon for the sufferings of me and my family i am your humble pathant."

CASE 9. — *Hypochondriasis.* — John R—, native of Cardigan, 27, Stephen Ward.

"Dear Sir,—On account that females is in attendance (to my sorrow) I kept the following *secret* from you that I have committed the banefull practice of self pollution or Onanism over ten years at intervals, but not consious of its dangerous consequences. Untill I consulted Dr. B— of Liverpool about two months ago, who recognized me by my first appearance to be a practical masturbator asked if I suffered from loss of memory and etc. But ever since I discontinue such a self ruin habit which left me in my present debilited state unfit for no kind of situation nor Society even with my own class of people. A work treating on this subject by Dr. Marston Anatomical Museum London came to my hand which convinced me that I should let the medical gentleman I am under know of this sinfull habit before I could be restored to my strength and vigour. No ulcers on my body as yet. Very seldom seminal emission occur during sleep. When I make water the last few drops is whitish. I shall feel very thankfull if you let me go under a course that may bring me to health.

"Yours truly,

J. R."

CASE 10.—*Hypochondriasis.*

"DR. WILKS.

"Dear Sir,—I humbly beg of you to read these few lines i have rote conserning this desease wich is almost driving me to comit suiside for i belive it has ben brought on of my self wich causes me to be to much ashamed to tell you before any other Jentlemen but if you will look into my case and doo wat you can for me i will pay you as much as lase in my pour i humbly beg of you to ceepe it as secret as you can for i fancy that every one that looks at me thinks that i have ben doing sum thing rong if i was Jeard about it i do not no wat would become of me for i have ben a great siner in the site of god and man i beg to be excused if i have don rong by riting in this way for i doo not no wat to doo else for i ham in such a state of mind that I though to lay my case before you in this way I went to sea at 11 years of age and i was inticed to comit self polotion on my self * * * * (The patient in a very long document gives an account of his going from sea to land and *vice versa* in order to give rest to his mind) Wen in strange compney my hands would shake very much then after that i had stinging pains in my penis and my testicles youst to hang very lo and be sore and then i had a kinde of a feaver come over me wich made me feal as if my flesh was crawling and my tong was very wite and ruff i bought a book wich is called the silent frend wich gave the discriptions of my case very clear and recomended cordial Barm of Siracom to be tacon wich i took 5 bottles of wich gave me a little relif, then i shiped for london and i made up my mind to fall overboard for fear that it should be none wat was the mater with me but the almity was merciful and gave me courig to wether the pashig but wen we got round the horn into warme wether I youst to strip and bathe my testicles wich seemed to give me ease my penis and testicles is very small and at one time the head of my penis was as wite as chalk and now under the head of it as black like brused blod and stingin pains and my testicles hang down and get sore thear has always in the day like litle black spots and hares before my eyse al tho my site is very good as yet some times if i should want to sine my name i canot for my hand shakes so my hare is always coming out wen i come it very much and at the least excitement or harde work my hart beats very heavy and i ham all of a trimble i fear that i ham in a bad state that if som one dose not befrend me i shall dy a misrable deth i feal to be pining away this last 4 or 5 months you will please to excuse me for riting in this cinde of a maner for i ham no scolar and if i have don rong i hope you will for give me. I ham a outpatient in guy's hospital from last Wednsday but i could not tell you all that ailed me for i belyve if my case was publickly none i should make away with my self."

It would be a crime of the blackest dye to give to patients such as those who here record their own cases the abominable books which have been mentioned, instead of endeavouring to inspire these miserable creatures with comfort and hope.

A FEW WORDS

ON THE MEANS TO BE ADOPTED FOR

ESTABLISHING A COMMUNICATION

BETWEEN

THE BLADDER AND THE EXTERIOR OF THE BODY,

WHEN THE URETHRA HAS BECOME IMPERMEABLE.

THE LAST RESOURCE AVAILABLE IN CERTAIN CASES.

By EDWARD COCK.

In this short communication I have no intention of alluding to the treatment of ordinary stricture; nor shall I yield to the temptation of criticising the various methods and appliances which, within the last few years, have been either invented or resuscitated for the purpose of restoring a narrowed urethra to its normal condition. Some ingenious mechanical contrivances cut, some tear, some burst, some are supposed to dilate with marvellous rapidity. My objection to their use is founded on a forty years' experience, which has taught me that such cutting, tearing, bursting, or rapid dilatation is often exceedingly mischievous in its effects, and fails in establishing a permanent cure, and that the object in view may be accomplished by much milder, surer, and safer means.

The purport of this paper is not to show how a strictured urethra may be treated, but how the bladder may be got at when the canal has become positively and absolutely impermeable to instruments of every description.

Some years ago I sent to the Medico-Chirurgical Society a paper, which was read and published in the 'Transactions,' detailing my experience in the operation of puncturing the bladder through the rectum.

The object which I then had in view was to prove, by the test of experience in forty cases, that this operation is safe, easy of accomplishment, and without danger as to its consequences, and that in cases of retention which resist ordinary treatment it is greatly to be preferred to long-continued attempts at catheterism, which, whether successful or not, must be infinitely more injurious to the urinary organs than the simple and almost painless operation of tapping.

I considered that the benefit of the operation consists not merely in the immediate relief given to the patient, but also in the opportunity which it affords, by the retention of the canula in the bladder through an indefinite period, of diverting the flow of urine from its ordinary channel, and thus giving quiet, freedom from pain, and the natural means of restoration to the maimed, irritable, or diseased urethra.

From this point of view I conceived that the bladder might be tapped with advantage in cases of obstinate stricture, in which retention of urine did not actually exist.

I wished to rescue the operation from the obloquy which had been cast upon it by every surgical writer, as a desperate kill or cure remedy, only to be used when every other means had failed; and to restore it to its legitimate place in the category of surgical means and appliances.

It is gratifying to know that since the publication of my paper in the 'Transactions,' the bladder has been tapped by many surgeons, and that even some of the warmest opponents to the operation have since received it as a very available friend in need.

Still, however useful and available may be the means of relieving the bladder by tapping through the rectum, this operation does not fulfil every necessity that may arise; for, to ensure a successful result, certain conditions are required, which do not always exist. An empty, habitually contracted bladder, and a very large prostate, are almost fatal objections to the operation.

A class of cases not unfrequently come under our observation, where the bladder must be got at somehow to save the life of the patient, and where yet it cannot be reached, either by ordinary catheterism through the urethra, or by tapping above the pubes or through the rectum.

We tap the bladder because it is distended and we cannot pass a catheter. But there are other conditions of the bladder more chronic, yet not less fatal, than acute retention. Every surgeon must be familiar with cases where stricture has existed for a number of years; where the urethra has become permanently obstructed, or destroyed by the constant pressure of urine from behind, and by reiterated attempts, generally fruitless, to introduce an instrument; where extravasation into the perineum has again and again taken place, causing repeated abscesses and their consequences, the formation of urinary sinuses and fistulæ, until the normal textures of the perineum become obliterated, and are replaced by an indurated, gristly structure; where the bladder has become thickened, and contracted by the constant action of its muscular coat until little or no cavity is left; and where the urine is constantly distilling by drops, either through the urethra or through one or several fistulous openings, which dot the surface of the perineum, penetrate through the indurated scrotum, and even find their way to the nates below, and the region of the pubes above. If unrelieved, these cases invariably terminate fatally. The patient slowly sinks under his constant efforts to micturate, and the unceasing fever and constitutional irritation produced by the passage of the urine through abnormal channels, and the frequent formation of fresh abscesses. Finally, the ureters and pelves of the kidneys become dilated; renal disease is set up; and the patient at length dies with symptoms of blood-poisoning by urea.

To remedy this complication of evils—viz., an impermeable urethra; a disorganised perineum, of which all, or nearly all, the anatomical features have disappeared: fistulous sinuses communicating with the urethra, and distilling urine; recurrent abcesses from the irritation of the extravasated fluid; broken down health from diseased bladder, the forerunner of renal disorganisation;—I say, to give the patient a chance of life or restoration, it is necessary to establish a free communication between the bladder and the exterior.

The natural passage through the urethra is not available, as the channel has long been either rendered impermeable or altogether destroyed. The sound and catheter are therefore useless; while the contracted condition of the bladder forbids the operation of puncture behind the prostate.

The surgical means adopted in these miserable cases should have a twofold object in view—an immediate and a more distant result. The one object is the prompt establishment of a free communication with the bladder behind the obstruction. The second and more remote is the restoration of the urethra to its normal condition, so far as this may be possible.

The operation which is recommended and described in most books of surgery proposes to combine both these objects—the relief of the bladder, and the cure of the stricture. It consists simply in effecting a union between the two permeable portions of the urethra, by cutting down upon and dividing the intermediate strictured or obstructed portion. "The urethra is opened anterior to the stricture on a staff carried down to the spot. The stricture is then to be traced backwards, and divided until the canal behind it is reached and laid open. A flexible catheter is then to be introduced through the urethra into the bladder, a portion of the catheter representing the splice between the two permeable portions of the canal. The wound fills up and unites over the catheter, and the cure is then complete."

The theory is plausible and good, could it be carried out, but the practice is most difficult, tedious, and severe; often fatal, and always unsatisfactory as regards the ulterior result. Even if all the tissues of the perineum were in a healthy normal state it would be most difficult, often impossible, to lay open a stricture which lies at the bottom of the wound, and which has long been impermeable to the smallest instrument, and can neither be seen nor felt by the operator. I have frequently seen this operation performed, or rather attempted; and I doubt whether in any one instance the original intentions of the surgeon were carried out. In several instances death followed as a consequence of the operation, and a post-mortem examination revealed that, although all the structures of the perineum had been most unnecessarily incised and damaged above, below, before, behind, and round about the stricture, it was the stricture alone which had escaped untouched and without mutilation, while the catheter had been carried out of the canal above the stricture, and reinserted below. This very circumstance is fatal to the prospects of a permanent cure, for the splice, not consisting of restored urethral tissue lined by mucous membrane, but being a mere factitious canal, formed by granulations around the catheter, will begin to contract as soon as the use of the instrument is abandoned, and will, at no very distant period, become again impermeable. I need scarcely say how all the difficulties I have described must be multiplied when the perineum is swollen, indurated and perforated by sinuses, when every anatomical guide that could be appreciated by sight or touch has disappeared, and when it is impossible to ascertain how far backwards the obstructed and diseased portion of the urethra extends; and yet these are precisely the cases in which surgical interference is necessary.

For many years I have adopted another operation, which is at once easier, simpler, and much more successful; for the objects aimed at in the operation I am about to describe can be accomplished, while I doubt if those of the other have ever been fulfilled.

However complicated may be the derangement of the perineum, and however extensive the obstruction of the urethra, one portion of the canal behind the stricture is always healthy, and often dilated, and is accessible to the knife of the surgeon. I mean that portion of the urethra which emerges from the apex of the prostate—a part which is never the subject of stricture, and whose exact anatomical position may be brought under the recognition of the finger of the operator.

Thus, when we cannot introduce a catheter by the ordinary method, and even when we cannot tap the bladder through the rectum, it still remains to us to tap the urethra as it emerges from the prostate, and thus to effect the desired communication.

There are considerable advantages attending this operation. The bladder is reached without any unnecessary mutilation of the perineum. The communication is effected in nearly a straight line from the exterior to the cavity of the viscus; so that the canula, which is inserted and retained, can be removed whenever necessary, and can be easily replaced. The functions of the entire urethra are suspended, and may be kept in abeyance for an unlimited period. The urine no longer finds its way abnormally through the stricture and sinuses of the perineum. The tissues are no longer subjected to constant irritation from infiltration. The constitutional symptoms are relieved, and time and opportunity are given for the removal, by absorption, of those adventitious deposits which obstructed the urethra, indurated the perineum, and rendered the introduction of an instrument impossible. The pressure on the kidneys is removed, and, if expedient, the bladder may be readily washed out, until its lining membrane assumes a healthy character. The strictured and damaged portion of the urethra, being no longer subjected to the constant pressure of urine from behind, may probably so far recover itself as to allow of restoration by the ordinary means of dilatation; or, should the canal have become permanently obliterated, the patient still retains the means of emptying his bladder through the artificial opening without difficulty or distress, and at very moderate inconvenience to himself.

This operation, which (for the sake of distinction) I may call "tapping the urethra at the apex of the prostate, unassisted by a guide staff," is alluded to in some books on surgery, but does not appear to have found much practical favour. I have performed it many times, but have seldom seen it attempted by others. Indeed, I do not think that the mode of procedure recommended in surgical works and manuals is likely to lead to a successful result; for it is made to depend on a slow and protracted anatomical dissection, through structures where, almost invariably, all anatomy has been destroyed, or if present, cannot be brought under the eye of the operator.

My experience has suggested the following method of reaching the urethra; and by adopting it I have never failed to accomplish the object in view.

The only instruments required are, a *broad* double-edged knife, with a very sharp point; a large silver probe-pointed director, with a handle; and a canula, or a female catheter modified so that it can be retained in the bladder.

The patient is to be placed in the usual position for lithotomy; and it is of the utmost importance that the body and pelvis should be straight, so that the median line may be accurately preserved. The left forefinger of the operator is then introduced into the rectum, the bearings of the prostate are carefully examined and ascertained, and the tip of the finger is lodged at the apex of the gland. The knife is then plunged steadily but boldly into the median line of the perineum, and carried on in a direction towards the tip of the left forefinger, which lies in the rectum. At the same time, by an upward and downward movement, the vertical incision may be carried in the median line to any extent that is considered desirable. The lower extremity of the wound should come to within about half an inch of the anus.

The knife should never be withdrawn in its progress toward the apex of the prostate; but its onward course must be steadily maintained, until its point can be felt in close proximity to the tip of the left forefinger. When the operator has fully assured himself as to the relative positions of his finger, the apex of the prostate, and the point of his knife, the latter is to be advanced with a motion somewhat obliquely either to the right or the left, and it can hardly fail to pierce the urethra. If, in this step of the operation, the anterior extremity of the prostate should be somewhat incised, it is a matter of no consequence.

In this operation it is of the utmost importance that the knife be not removed from the wound, and that no deviation be made from its original direction, until the object is accomplished. If the knife be prematurely removed, it will probably when reinserted make a fresh incision, and complicate the desired result. It will be seen that the wound, when completed, represents a triangle; the base being the external vertical incision through the perineum, while the apex, and consequently the point of the knife, impinges on the apex of the prostate. This shape of the wound facilitates the next step of the operation.

The knife is now withdrawn, but the left forefinger is still retained in the rectum. The probe-pointed director is carried through the wound, and, guided by the left forefinger, enters the urethra and is passed into the bladder. The finger is now withdrawn from the rectum; the left hand grasps the director, and along the groove of this instrument the canula is slid until it enters the bladder.

The operation is now complete, and it only remains to secure the canula in its place with four pieces of tape, which are fastened to a girth round the loins. There will probably be no escape of urine until the stilette is removed from the catheter.

A direct communication with the bladder has now been obtained, and the relief to the patient will be immediate. Unless the kidneys have become irremediably disorganised, we may confidently anticipate a favorable result; and the restoration of the urinary organs will be more or less complete, in proportion as the obstructed portion of the urethra is more or less amenable to the ordinary judicious treatment of stricture. The canula may generally be retained in the bladder for a few days, and if the state of the urine renders ablution necessary, the viscus may be frequently washed out. The canula may then be removed, cleaned, and reintroduced. A flexible catheter is sometimes more desirable and congenial to the feelings of the patient than a metallic canula.

If the previous destruction has not been very great, and if the case progresses favorably, the swelling of the perineum and scrotum gradually subsides, the induration disappears, and the urinary sinuses become obliterated. The urethra may then be examined in the ordinary way, to test its permeability, and one may be agreeably surprised to find that the sound or catheter readily passes through the former stricture, until it strikes against the canula. An attempt may then be made to introduce a flexible catheter into the bladder, and its passage may, if necessary, be facilitated by passing a director through the perineum into the bladder; and guiding the catheter along its groove. The urethra once restored to its normal condition and calibre, the artificial opening through the perineum soon heals up, and, barring the liability of stricture to return if not attended to, the cure may be said to be complete.

We must not, however, always expect so favorable a result. I have operated in several cases where the obstruction of the canal was complete, and its impermeability permanent.

In such cases the patient is condemned to pass his water through the artificial opening in the perineum, unless a new passage should be bored to unite the upper and lower portions of the patent urethra—an operation which I have seldom or never known to be successful.

The necessity of micturating through the perineum may seem to be a considerable hardship, but, with a little arrangement, the inconvenience is not very great; and be it remembered, that the man's micturition is merely assimilated to that of the other sex.

To keep the artificial passage in a permeable state, it is generally necessary to pass a flexible bougie through the opening occasionally, and to retain it *in situ* for a few hours. The patient very soon learns to do this for himself.

A few years ago there were at least half a dozen men on whom I had operated, and who were happy and comfortable, passing their water through the perineum. I have now under my frequent observation two men, on one of whom I operated twenty-five years ago, on the other twenty years ago, and both of whom are thankful for their condition.

I have not found that this operation, with its result in a permanent factitious urethra, at all interferes with the sexual function, although it is of course a complete bar to procreation. One of the individuals alluded to above has, I am sorry to say, taken a somewhat unfair advantage of his immunity from ordinary liabilities, by promiscuous and vicarious indulgence of his passions, and is well known as a gay Lothario and modern Don Juan among the fairer sex.

ON A CASE

OF

VITILIGOIDEA PLANA ET TUBEROSA.

By F. W. PAVY, M.D., F.R.S.

VITILIGOIDEA is the name given by Drs. Addison and Gull to an affection of the skin first described by them in vol. vii, second series, of the Guy's Hospital Reports. The affection, they say, so far as their observations extend, "presents itself under two forms, namely, either as tubercles, varying from the size of a pin's head to that of a large pea, isolated or confluent; or, secondly, as yellowish patches of irregular outline, slightly elevated, and with but little hardness. Either of these forms may occur separately, or the two may be combined in the same individual." They add, that the only account which at all corresponds with the affection described by them is that given by Willan of Vitiligo; and, believing it to be probable that Willan would have included the cases recorded by them under Vitiligo, or an allied affection, they named them accordingly—Vitiligoidea, distinguishing the two varieties by the terms Vitiligoidea tuberosa and Vitiligoidea plana. Five cases are mentioned in their communication. In the first, the eruption consisted of shining tubercles of a lightish colour with here and there superficial capillary veins meandering over them. It extended across the nose, and slightly affected both cheeks. There was no jaundice in association with it. In the second case, after jaundice had existed for fourteen months, patches of a light opaque colour, and with the surface and edges slightly raised, began to show themselves around the eyes. A similar alteration of the skin also occurred upon the palms of the hands, and on the palmar aspect of the fingers. The third case resembled the first in the absence of jaundice, and in the eruption consisting of scattered tubercles. The fourth and fifth cases were each associated with jaundice. In one, the eruption consisted of patches and tubercles together; in the other, as in the second case, patches only existed.

During the past summer an exceedingly well-marked case of Vitiligoidea, which forms the subject of this communication, has been under my care in the clinical wards of Guy's Hospital. The disease is of the double variety. The following is the history of the case from the report of my clinical clerk, Mr. Morison:

Louisa L—, æt. 39, admitted into Guy's Hospital May 29th, 1866. She is a married woman, and has had a large family. She says that she always enjoyed good health up to about seven years ago, when she was in great trouble from sickness and a death amongst her children. She then had diphtheria; afterwards an abscess in her breast, boils over her body, and about this time a miscarriage. Three years ago she was confined of her eleventh child, and six weeks afterwards became affected with jaundice. The jaundice was associated with great stinging and itching of the skin, especially in the hands and feet. It lasted for about ten months, disappearing two months before her next and last confinement. A fortnight after this confinement one of her sons died rather suddenly, and the shock so upset her that two days afterwards she found herself jaundiced still more deeply than she had been before. Since then she has never been free from jaundice. Shortly after its reappearance she suffered again from stinging and itching of the skin, this being also tender to the touch. Thus, it gave her pain to sit down from tenderness of her seat, and she also experienced pain when she took hold of or held any hard substance in her hands. About this time small lumps began to appear on the backs of her fingers; similar ones have since come out on other parts. Cream-coloured patches have likewise shown themselves upon the hands, around the eyes, and elsewhere. Those around the eyes first became visible about two months before her admission. Both lumps and patches have remained without undergoing any change, except that they have gradually increased in size. Her skin generally is of a greenish-yellow colour. She has been getting thinner, but her appetite has been pretty good. The pulse is natural. A systolic murmur is audible over the apex of the heart. The lung sounds are normal. The urine is free from albumen, but is deeply coloured from the presence of the colouring matter of bile. A large swelling exists in the upper part of the abdomen on the right side, which is evidently due to enlargement of the liver. It is tender to the touch, and dull on percussion, the dulness extending for three inches below the ribs. No irregularity is discoverable upon the surface of this swelling.

The patches observable on the skin appear as if an opaque cream-coloured deposit pervaded its texture. The alteration is evidently situated in the cutis vera, the cuticular covering being to all appearance perfectly natural. A broad band of skin thus affected encircles each eye, giving to the face a very peculiar appearance. The deposit may also be observed to be irregularly dispersed over the sides of the face and neck. It is very conspicuous and forms extensive patches upon the backs of the hands. It occurs in spots upon the palms of the hands and the palmar aspect of the fingers, and in a like manner upon the heels. Lastly, it is scattered here and there over the body generally. Where it exists the surface is very slightly raised; at least this is the case with the patches around the eyes; but there is no loss of the natural suppleness and softness belonging to the skin. In the tips of the fingers it occurs in little discrete spots, and thus causes them to present a somewhat nutmeg appearance. On here passing the finger attentively over the surface a slightly nodular impression is to be perceived.

The tubercles vary in size, the largest being of not quite the size of a horsebean. They occur on the backs of the fingers, and particularly over the knuckles, on the ears, shoulders, elbows, and outer side of the arms, and on the nates, knees, and ankles. Some are simple tubercular elevations, of a whitish colour; others, the largest, are irregular, and composed of clustered nodules. Upon the ears little vessels are discernible meandering over them. Upon the knuckles they look something like gouty concretions.

In a second communication, in the eighth volume, second series, of the "Reports," Dr. Gull remarks, with reference to one of the cases that had been before described, that it was important to note that during the last few months the metacarpo-phalangeal articulations had become tuberose, having apparently undergone changes similar to that of the skin, although the integuments over them were unaffected. Precisely the same thing has happened with my own patient. In connection with the extensor tendons over the metacarpo-phalangeal articulations of the index and middle fingers of the right hand and of the middle finger of the left hand there exist firm, tubercular masses, which move with the tendon underneath the skin, this being perfectly healthy.

The broad, cream-coloured patches around the eyes form one of the most conspicuous features of the affection in my patient's case; and it is a noteworthy point that in each of the three cases associated with jaundice recorded by Drs. Addison and Gull, the disease should have similarly shown itself here. The affection certainly seems to have some connection with the existence of jaundice. In the case given by Drs. Addison and Gull, in which the plane and tubercular varieties were combined, a morbid sensibility of the skin is spoken of, the patient experiencing pain in using a knife to cut bread. In the other jaundiced cases, also, an increased sensibility of the skin was complained of. My own patient, at one time, was so tender upon the surface, that it hurt her to sit down, and when any hard substance touched her. It may further be noted, that all the cases hitherto referred to in this communication in which jaundice has been associated with the affection have belonged to the female sex. In another case in the hospital, under Dr. Barlow's care, in October, 1864, the plane variety which existed around the eyes and nowhere else was associated with jaundice, and the patient was likewise a member of the female sex.

I now desire to draw attention to the nature of this affection, a view having been expressed which is quite at variance with what has been observed in the cases that have occurred at Guy's. Thus, Professor Hebra, in the forthcoming English edition of his work on 'Cutaneous Diseases,' translated by Dr. Fagge, writes as follows concerning Vitiligoidea :

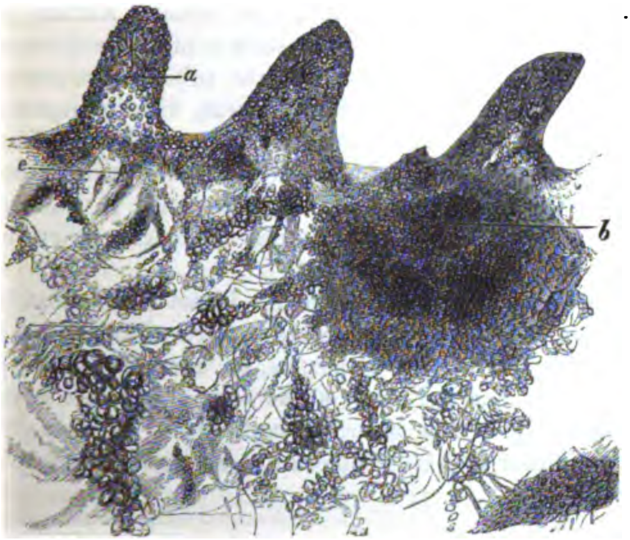
"This is another affection which arises from a morbid change in the sebaceous glands. It received the name of Vitiligoidea from the late Dr. Addison and Dr. Gull. . . . From the account of this complaint given by Addison and Gull, and also from the plates which accompany their papers, it is easy to perceive that the cutis itself could not have been the only structure affected in the cases which came under their observation. In fact, I am convinced that Vitiligoidea consists in a peculiar degeneration of the sebum, and the glands which secrete this substance; and that this disease is allied to other cutaneous affections which have long been well known, and particularly to that form of milium which I have already mentioned as occurring on the red part of the lips, on the nymphæ, and round the corona of the glans penis. . . . . . I must also point out that this affection may be detected in many persons on the upper and lower eyelids, and even on some other parts of the face. Its presence is discovered by stretching the skin forcibly, and making a tolerably deep incision into the whitish or yellowish patches, which then come into view. On applying pressure to the sides of the wound, there escape masses of degenerated sebum which had accumulated within the cutis. . . . . . As I have already stated, in each of the two cases of Vitiligoidea which have come under my observation I succeeded in squeezing out the morbid material from the cavities in which it lay, by applying pressure, after having made an incision through the cuticle. The fatty substance which I thus obtained was of firm consistence, and of a yellow, or at any rate a yellowish colour, and resembled the sebaceous secretion."

In Belcher's edition of Neligan's treatise on 'Diseases of the Skin,' I notice this affection spoken of as a form of stearrhœa:—"An account of what I conceive to be Stearrhœa flavescens" [a form of acne] "has been published by Drs. Addison and Gull, but denominated by them Vitiligoidea plana and tuberosa, from a supposed correspondence between the affection and the incorrect definition of Vitiligo which was given by Willan."

From the statements made by Hebra, it seems evident that a mistake in identification has occurred, and that the affection under consideration is not the one which formed the subject of his description. Drs. Addison and Gull say, in reference to one of their cases, that some of the tubercles "looked as if they were beginning to suppurate, and many were not unlike the ordinary molluscum; but when incised with a lancet, they were found to consist of firm tissue, which on pressure gave out no fluid save blood." In my own case the resemblance to molluscum is such that my first impression on looking at the tubercles was, that they belonged to this affection.

My patient's consent having been obtained, a large tubercle was removed from the back of the little finger, for the purpose of being submitted to minute examination. The following is a description of the appearances that were observed:

"The tubercle was of about the size of a horsebean, or rather less. It was distinctly nodulated on the surface. Under the scalpel it was tough, and of an almost cartilaginous firmness. When it was incised and squeezed, nothing but an opalescent juice exuded. On its under surface, the appearance of separate nodules was more conspicuous than on the surface



Microscopical appearance of a portion of a tubercle of Vitiligoidea, from a drawing by Dr. Moxon.

formed by the skin. They extended up to and involved the cutis, but the cuticle escaped implication. In one part the cutis was affected without there being any adherent nodule beneath, and it presented the appearance of being pervaded with a cream-coloured deposit throughout its texture. The nodules were opaque and cream-coloured, like the cutis above.

A portion of a nodule, being torn to pieces with needles for microscopic examination, was found to consist of a very dense fibrous structure. It was so tough, indeed, as to require considerable force to dissect it out. The drop of water in which it was torn up became opalescent from the juice that exuded, and under the microscope this was seen to be pervaded with what appeared to be fat-granules. The solid part consisted of fibrous tissue. There was no appearance of cells to be observed. The foregoing figure is from a sketch by Dr. Moxon, of a vertical section through the tubercle. The cuticle is missing. The papillæ (*a*) and texture (*c*, *b*, *e*) of the skin are seen to be filled with the same fat-looking granules referred to above as observable in the opalescent juice from the tubercle.

The minute examination, therefore, revealed nothing to lend support to the notion that this disease is of a sebaceous nature. On the contrary, it showed that the tubercles consist of nodules of exceedingly dense fibrous tissue, with interspersed fat-granules, and that the texture of the true skin implicated is pervaded with granules of a similar nature. Such a deposition of granules would seem to be the cause of the opaque, cream-coloured patches constituting the plane variety of the affection.

A further proof that Vitiligoidea is not a sebaceous disease is the fact that the deposit occurs in parts of the surface where there are no sebaceous glands, as, for example, on the palms of the hands and the soles of the feet. This fact is, indeed, mentioned by Hebra as being opposed to his view; but he does not admit that it is sufficient to absolutely negative it.

THE PLATE

Shows the appearance of the face and hands in Dr. Pavy's case of Vitiligoidea.

Fig 3.

CONTRIBUTIONS

TO THE

PHYSIOLOGY OF BINOCULAR VISION.

By JOSEPH TOWNE.

Section VII.

The observations contained in our former sections have, for the most part, had reference to the stereoscope and stereoscopic results; and we have been led to pursue this course of inquiry, from a conviction that a clear analysis of the phenomena of the stereoscope, would be a step towards a solution of the phenomena of natural vision.

In the present section it is proposed to apply, and illustrate, some of the facts we have accumulated; and, in a few additional and very simple experiments, to exhibit them in their relation to binocular vision.

The form and position of the eyes, and their relation to the bones of the face, determine that corresponding tracts of the retinæ shall receive their respective images from definite parts of the external field. It is, for example, obviously impossible that images should be referred to the temporal halves of the retinæ, from their own sides of the external field; or that the nasal halves of the retinæ, should receive their images from the opposite sides of the external field; and it is equally impossible, that the lower halves of the retinæ should receive their images from below, or that the upper halves of the retinæ should receive their images from above. Hence the retina has been described as consisting of four great tracts, an upper, a lower, an inner, and an outer. It has also been remarked, that the pictures falling upon the two right sides of the retinæ agree, and that the pictures falling upon the two left sides of the retinæ agree. Müller gives prominence to these points, as also to the identity of action existing between those tracts of the retinæ, which receive their respective images from the same parts of the external field.

It is observable, however, that more recent inquirers, have appeared to attach to these considerations, *practically* at least, but little importance; so that elaborate disquisitions, relating to the phenomena of binocular vision, may be found, which contain no reference to these fundamental facts, while experiments are sometimes adduced, that would seem to ignore their existence. Hence the term *tracts of the retinæ*, as frequently employed, would appear to mean little more, than that certain portions of the retinæ receive their images, from definite parts of the external field. If, however, the term tracts of the retinæ is to be understood in this limited sense, we might with equal propriety apply the same term to two cameras. But the conclusion at which we have arrived, differs materially from this view; and believing, as we do, that a full recognition of the office and operations of the different sections of the retinæ, is of primary consideration, in all questions relating to the physiology of binocular vision; it is our wish to give to this part of the subject the prominence it seems to require. Hence the first step in the following inquiries will consist, in illustrating the individual action of the different tracts of the retinæ. Our hope being, in thus observing the separate action of the component parts of the visual mechanism; that we may possibly obtain some more distinct ideas of the visual mechanism as a whole.

It is proposed, then, in illustration of this part of our subject, first to separate the nasal halves from the temporal halves of the retinæ, regarding each pair, the nasal and the temporal, as a distinct retinal field; subsequently, we shall treat in detail the different sections of the retinæ, and observe them in their individual as well as in their correlative actions; our aim being to show that, while the two eyes form in their combination one apparently single mechanism, yet that through all their combined operations, the distinctness of each individual section of each retina, is constantly preserved.

The experiments which it is proposed to adduce, will be considered from four distinct points of view: first, as they exhibit the great tracts of the retinæ in their relation to the external field; then in their relation to each other; subsequently, in their separate and distinct actions; and finally, in their combined and reciprocal operations, as they exist in the common field of vision.

We first direct our attention to the great tracts of the retinæ, considered in their relation to the external field. It has been sometimes said that the field of vision is limited, or bounded, by the bones of the face; we remark, however, that the visual field is limited in three directions only, by the bones of the face; namely, above by the brows, below by the cheek bones, and to the inner side (of each eye) by the bridge of the nose; while laterally, and to the outer side of each eye, no corresponding boundary exists; at this point the arch of the orbit meets the cheek bone, and leaves a depression or hollow, that admits the rays of light in a lateral direction, to the full extent to which the eye is capable of receiving them.

If a person stand midway between, but on a plane somewhat in advance of two lighted candles placed suppose twelve feet distant from each other, both the candles will at the same time come into view, while the eyes of the observer are directed immediately forwards. On closing the right eye the candle of the right side will be lost, and on closing the left eye the candle of the left side will be lost.

Two facts are made apparent by this observation: first, that the field is considerably extended laterally, reaching, indeed, somewhat to the rear of the observer, while the eyes are directed immediately forward; secondly, that a large proportion of the visual field belongs exclusively to the nasal half of the corresponding retina. This is evident from the fact that on either eye being closed, a considerable portion of the field on the corresponding side is lost, it being clearly impossible that objects occupying this lateral part of the external field, can be referred either to the temporal half of the corresponding retina, or to any part of the opposite retina. It is, therefore, evident that the nasal halves of the retinæ must, to some extent, be regarded as distinct organs.

Our next step consists in separating the nasal, from the temporal halves of the retinæ. For this purpose a sufficient field is obtained, by means of connected boards placed horizontally. Two parallel lines are then to be drawn through the central part of the field; the distance between the two lines agreeing with the distance between the centres of the two pupils; these are the visual lines. At the distant end a semicircular line is to be drawn, stretching from one side of the field to the other. Upon each visual line, and also in the direction of the semicircular line, are to be placed, at short intervals, a number of objects. We then draw a series of lines passing from the centre of each object to the outer side of the corresponding visual line, so as to converge nearly to a point, at a short distance from the eye; and we introduce, in the direction of the median line, a septum, extending from the eyes to the objects viewed. See fig. 1, plate I. It will be observed that by introducing a septum, we cut off all images from the *temporal* halves of the retinæ, while we offer no impediment to the *nasal* halves of the retinæ; the direction of the nasal halves of the retinæ being outwards, or away from each other. The arrangement being thus completed, we rest the distant end of the board upon the window-sill, and place the other end upon the bridge of the nose, while the eyes are directed immediately forwards, and to a remote object.

The visual result, is the superposition of the two objects placed upon the visual lines, the resultant image appearing in the direction of the median line; the septum is not apparent, nor is there any consciousness of visual obstruction. If the right eye be closed the right half of the field is lost, if the left eye be closed the left half of the field is lost, and it is only when we raise the head, and thus place the eyes above the septum, that we obtain a distinct idea of the real position of the objects viewed; or become aware that the objects placed on the two sides of the *external* field, are distant, the space between the visual lines; and that when viewed the two sides of the *visual* field approach each other and meet upon the median line; *thus the external field is represented in the visual field, less the space between the visual lines.*

If now we turn our attention to the temporal field, we need only observe, the directions from which the images are referred to the temporal sides of the retinæ, to ascertain the boundaries by which the temporal field is limited. Each retina may be considered as divided by a vertical line into symmetrical halves. All images falling to the inner side of this line, belong to the nasal field; they fall upon the nasal half of the retina, and are referred outwards, laterally, or to the corresponding side of the external field. All images falling to the outer side of this line, belong to the temporal field; they fall upon the temporal half of the retina, and are referred inwards, or to the opposite side of the external field. The inner boundary of the temporal field, therefore, is defined by the vertical divisions of the retinæ. But the outer boundary of the temporal field is determined by external causes; and we remark, *that the nose, from its form and position, acts as a septum,* placed intermediate between the two eyes. *And this septum it is that determines the outer boundary of the temporal field,* since no image can be referred to the temporal halves of the retinæ, from a point too lateral to allow of its transmission over, or crossing the nose, and in the direction of the pupil of the opposite eye. It follows, therefore, that the temporal field is bounded on the inner side by the vertical divisions of the retinæ, and to the outer sides by a line including so much of each retina as may receive images from the opposite side of the external field, and in the direction we have indicated.

For each of the experiments next to follow, a smooth flat board is required, about 18 inches in length, by 12 inches in width; these boards are to be covered with white paper. Having drawn in the median and visual lines, the point of intersection being 15 inches distant from the eyes, we place two objects, one on either side of the median line, and on a plane 1 inch nearer to the eyes than the point of convergence (that is, 14 inches distant from the eyes); and on a plane $12\frac{1}{2}$ inches from the eyes we have two other objects, each $1\frac{1}{2}$ inch distant from the median line. We also place two others 11 inches from the eyes, and distant from the median line $2\frac{1}{2}$ inches; two others, distant $9\frac{1}{2}$ inches from the eyes, and $3\frac{1}{2}$ inches from the median line; and, lastly, two more, at a distance of $8\frac{1}{2}$ inches from the eyes, and $4\frac{1}{2}$ inches from the median line.

It is to be observed that the distances named have reference to individual experiments, and are by no means arbitrary; the intention in the arrangement of these objects, being to secure a general balance for the two eyes, and to give uniformity to the figure, while the distance of the objects from the eyes is varied. The objects I have used for these experiments, are short pins, or rather pins that have been considerably shortened, so that, when inserted in the board, they stand about one eighth of an inch above the surface, while at the point of convergence I have placed a larger object, that is, a pin of greater length and having a larger head. The field being arranged as above described, the objects are to be connected with both retinæ by means of a line drawn from the centre of each object respectively, and passing to the *nasal* side of the corresponding retina. (Fig. 3, Pl. I.) These lines indicate the direction of the rays of light, in their transit from the object viewed, to the part of the retina affected; they have been called the *lines of direction*, but we feel it necessary to dispense with this term, which tends to mislead; for to conclude that these lines represent the visible direction of objects in the external field, is to assume that the laws which regulate the transmission of light to the retinæ, have their reflex in those laws which rule the transmission of the retinal impression to the brain, and determine the projection of the visual image into space. It will be evident, however, that these lines, when viewed under the conditions of the following experiments, appear under changed circumstances, and assume a new direction. We shall, therefore, of necessity, have two sets of lines to refer to,—the lines as they represent the direction of the light in its passage to the retinæ, and the lines as they appear when submitted to the eyes; in other words, the lines as they indicate the directions, in which the images are transmitted to the retinæ, and the lines as they indicate the direction in which the retinal images are projected into space. The former we shall term the lines of *transmission*, the latter the lines of *projection*.

The field being prepared, as above described, the proximate end of the board is to be placed upon the bridge of the nose, and the distant end is slightly raised, while the eyes are directed to the pin inserted at the point where the two visual lines meet. Thus viewed, the apparent result is a number of straight lines, issuing from the upper part of the nose. Of these lines one is seen in the direction of the median plane of the head, while the others diverge on either side. The median line springs from a point opposite the upper part of the nose, and passes to the pin inserted at the intersection of the optic axes; while the two lateral groups of lines are ranged one on either side of the median line, and each line contained in these groups passes to its corresponding object in the external field. If either eye be closed the corresponding half of the field is lost. (See result, fig. 6.)

In the next experiment, we retain the original figure, so far as relates to the arrangement of the objects to be viewed, but with this important difference. In the experiment just described, the several objects were connected *exclusively* with the *nasal* halves of the retinæ, those lines that would connect them with the temporal halves of the retinæ being omitted. In the present experiment, we omit those lines that would connect the objects in the external field with the nasal halves of the retinæ, and introduce those only which connect them with the *temporal* halves of the retinæ. The field, as now prepared, comprises a series of objects, each object being connected *exclusively* with the *temporal* half of one of the retinæ. (Fig. 4, Pl. I.) And viewed as before with both eyes, the result appears exactly the same as that obtained from the last experiment; the visual effect being now, as then, a series of lines, issuing from a point opposite the upper part of the nose, and passing each line respectively to the corresponding object in the external field. If the right eye be closed the left half of the field is lost, if the left eye be closed the right half of the field is lost; but when the field is viewed with both eyes, the apparent result is the same as in the last experiment. (See result, fig. 6.)

In these two experiments the objects through the entire field have been connected with non-corresponding sides of the retinæ.

In the experiment next to follow we connect the objects of one side of the field with the corresponding halves of the retinæ. If, for example, we take the right side of the field, we connect all the objects placed on the right side of the median line with the corresponding sides of the two retinæ. (Fig. 5, Pl. I.) When viewed, as before, with both eyes, the result is, that the visual lines appear superimposed in the direction of the median plane of the head; and that we see a series of divergent lines apparently issuing from the upper part of the nose and passing to the objects on the right side of the field, *these resultant lines being all grouped on the right side of the median line.* (See fig. 7, Pl. I.)

For Experiment 5 we again use the same objects, and view them under precisely the same conditions; excepting that for this experiment, we connect each object with corresponding parts of the two retinæ, by means of two lines drawn from the centre of each object in the field, and passing one line to the nasal side of the corresponding eye, and the other line to the temporal side of the opposite eye. The objects of the right side of the field are connected by means of red lines, and those of the left side of the field by means of black lines. (Fig. 1, Pl. II). When viewed as before, the visual result appears the same as in our former experiments—a number of straight lines are seen, apparently issuing from the upper part of the nose; one of these resultant lines passing in the direction of the median plane of the head, while the others are divergent lines, grouped on either side of the median line, and passing to the corresponding objects in the external field; *in short, each two of the lines of transmission, when submitted to view, coalesce and form one line intermediate between the two.* This, the line of projection, results, then, from a combination of the two corresponding lines of transmission, and thus through the entire field, the lines of projection have a direction exactly intermediate between those of the two corresponding lines of transmission; they apparently issue from the upper part of the nose, and pass each respectively to the corresponding object in the external field. It is to be observed further, that the red lines appear grouped on the right side of the median line, while the black lines appear grouped on the left side of the median line. (Fig. 2, Pl. II).

We have already stated that the experiments above described are to be considered from four distinct points of view; and first, as they exhibit the great tracts of the retinæ in their relation to the external field. In our first experiment it is shown that the two nasal halves of the retinæ, when separated from the temporal halves, and brought into simultaneous action, form a single, symmetrical, retinal field, and that this field covers the external field, *less the space between the visual lines*. (See fig. 1, Pl. I, and result.) It has also been shown that the temporal halves of the retinæ, when brought into separate action, form a single symmetrical field. This fact may be further illustrated by viewing the field with the optic axes parallel; the eyes being divided by a septum. Under these circumstances all objects placed within the visual lines fall upon the temporal halves of the retinæ, and, when viewed with the optic axes parallel, those objects placed to the right side of the median line, are referred to the left side of the external field, while those placed to the left side of the median line, are referred to the right side of the external field; the visual lines themselves meeting, and appearing as one line in the direction of the median plane of the head.

The temporal field, therefore, like the nasal field, represents the external field, *less the space between the visual lines*, and it does so whether viewed with the optic axes converged or with them parallel.

We may now consider these two distinct fields, first with reference to their individual completeness, and then with reference to their comparative extent. That the nasal halves of the two retinæ together, form a more extended and symmetrical eye than the eye of the right or the left orbit, is proved by Experiment 1, Pl. I. Since the single eye, that is, the eye of the single orbit, loses a portion of the opposite side of the external field, and therefore is both limited in range, and unsymmetrical; but the nasal halves of the two retinæ include the entire visual field, each half commencing from the median line, and extending laterally to a point slightly to the rear of the observer, *and these tracts of the retinæ together, form a perfectly symmetrical eye.* It is noteworthy, however, that the temporal field, though quite symmetrical and, so far as it extends, no less perfect than the nasal, is considerably more limited in extent. Hence it becomes evident that the nasal halves of the retinæ form, so to speak, a larger eye, while the temporal halves of the retinæ form a smaller eye. *It follows, therefore, that the space covered by the smaller eye must limit the extent of binocular coincidence.* And here several points of interest present themselves to our notice. In the first place we may enquire how much of the retinal picture falls upon the right eye alone, how much upon the left eye alone, and how much simultaneously upon the two eyes. And the answers to these several questions depend upon the comparative extent of the nasal and temporal fields. Now, with reference to the former, we need add nothing, it having been shown that *the nasal halves of the retinæ, include the entire visual field.* The limits of the temporal field have also been defined, but our previous statements have reference to the retinæ, and are of necessity somewhat vague, for, although the retinæ must be regarded as comprising the true field of vision, we do not, by tracing the limits of the retinal picture, obtain a distinct idea of comparative space in the external field. We shall, therefore, now offer a few additional observations respecting the boundaries of the temporal field, as they may be traced in their relation to the external field.

The temporal field, then, viewed with reference to external space, is bounded on the inner side by the median line, and its outer boundary may be determined in the following manner. Let two pencils be held in the vertical direction, one between the thumb and finger of each hand, and let the two hands be placed a short distance in advance of the person, *the eyes being directed immediately forwards.* Then, one eye (say the right eye) being closed, let the right hand be brought inwards towards the median line until the pencil held by this hand comes into view of the left eye; next (the right hand being retained in its position) let the left eye be closed, and a similar action be performed with the left hand until the pencil of this hand comes into view of the right eye. The two pencils, each held in the position which it occupied when first seen by the eye of the opposite side, will exactly define the lateral boundaries of the temporal field. The lines of vision will, of course, diverge as they are projected into space, but it is to be observed that they will retain the same relation to the retinæ.

From these observations we find, that binocular coincidence is limited to the central part of the field; or, to speak more definitively, to a space not exceeding the middle third of the field of vision. It will be observed that we do not attempt to define precisely the limits of lateral vision. This we avoid, partly because, from the conditions by which these limits are determined, they must, to some extent, be individual; and partly because the visual field fades off gradually towards the periphery, so that it might not be easy to define the exact point where vision ceases. We are not, however, at present, directing attention to the comparative distinctness, nor to the exact extent of vision in the lateral direction. *Our present object is to ascertain the extent to which, in binocular vision, corresponding images fall upon corresponding parts of the retinæ;* and in giving the boundaries of the temporal field in its relation to external space, we precisely determine this point. If, then, we say, the possible extent of binocular coincidence is limited to a space not exceeding the middle third of the field of vision, it follows that the remaining two thirds of the retinal picture fall exclusively upon the nasal halves of the retinæ, one third upon each side (the right third of the field falling on the right half, and the left third upon the left half); and we learn the important fact, that *to the extent of two thirds of the entire field, the impressions upon the two eyes are perfectly distinct.*

Having considered the great tracts of the retinæ, both in their relation to the external field, and also in their relation to each other, we are next to consider them in their distinct and separate action. In the experiments advanced with reference to this point, the objects viewed are connected with different sections of the retinæ, first with the two nasal, then with the two temporal halves; and, subsequently, the same objects are viewed under the same arrangement, except that each object is connected with corresponding halves of the retinæ. In reviewing this group of experiments, we observe a perfect uniformity in the results obtained, under conditions most varied and unusual, and we remark that this uniformity of result is associated, not only with corresponding parts of the retinæ, but that it is equally observable when non-corresponding parts of the retinæ are brought into simultaneous action. Hence we learn that the perfection of the visual mechanism, viewed simply with reference to identity of action, and reciprocation of its several parts, is consistent with uniformity of result, when identity and reciprocation are disturbed or annulled.

Experiments 2, 3, 4, in Pl. I, and Experiment 5 in Pl. II, show that the individual action of each section of the retinæ remains constantly the same, whether it be acting separately, or in connection with its corresponding section, or simultaneously with a non-corresponding section; whether for the time it be acting as a single or separate organ, or forming an integral part of the entire visual mechanism. In the first experiment of this series (fig. 3, Pl. I) we connect the objects *exclusively* with the *nasal* halves of the retinæ. In the second experiment of this series (fig. 4, Pl. I) we connect the objects *exclusively* with the *temporal* halves of the retinæ; and in Experiment 5 (fig. 1, Pl. II) we connect each object *throughout the entire field with corresponding sides of the two retinæ.* Under all these conditions, when the field is viewed simultaneously with both eyes, the result is apparently the same.

If the visual apparatus be regarded as a mechanism, designed with reference to identical images upon identical points of the retinæ, the most essential condition to the integrity of the visual function, would appear to be the existence of perfect co-operation, with undisturbed reciprocation through all its several parts. Hence it is remarkable to how great an extent this most delicate and complicated apparatus admits of division and sudivision, without very obvious disturbance to the integrity of its functions. The objects being arranged for the different experiments above described; and viewed simultaneously with both eyes, the visual result is in all apparently the same: namely, a number of straight lines issuing from a point opposite the upper part of the nose, and passing each line respectively to the corresponding object in the external field. For Experiment 5 (fig. 1, Pl. II) the objects are arranged with strict reference to the formation of identical images upon identical parts of the retinæ; and when the field is viewed simultaneously with both eyes, the result is consistent with this arrangement: namely, superposition of all the images. Still, we observe that, whether viewed with the right eye alone, or with the left eye alone, or with both eyes together, the result remains apparently unchanged; and it is to be remarked that this seeming anomaly accords with the experience of ordinary vision, for, whether we look with both eyes together, or with either eye alone, we perceive very little difference, excepting only that in closing one eye we lose a portion of the field on the corresponding side. *The reason is that the direction of every part of the retina is rigidly determined, and remains the same, whether it acts singly, or as an integral part of the entire visual mechanism.* Hence we find, that not only may corresponding sections of the retinæ be dissociated, but the one or the other may even be withdrawn, while the visual result remains apparently unchanged.

The foregoing observations lead to the conclusion that the vision of one eye may be lost without any obvious deficiency, excepting a curtailment of the field on the corresponding side. That both temporal halves of the retinæ may be lost, and the loss may not be observed;. that the two nasal halves may be lost, and still a good symmetrical eye remain, but limited to the middle third of the field. Be it remarked, however, that two corresponding halves of the retinæ cannot be lost without the loss being instantly recognised, since blindness over one half of the field is the inevitable result.

A glance at fig. 1, Pl. II, will make it apparent that there remains a point of much interest in connection with this experiment, to which attention has not yet been directed. The conditions of this experiment precisely agree with those of ordinary vision; the objects in the field are placed at various distances from the eyes, and are so arranged that, when the eyes are directed to the point of convergence, the images are referred to corresponding sides of the two retinæ. The figure to which we refer represents the direction of the rays of light in their passage from each object respectively, to corresponding parts of the two retinæ; when the field is viewed as above described, we lose all recognition of these lines, with reference both to locality, and to direction. Instead of two lines passing from corresponding parts of the two retinæ, and meeting at a point agreeing with the centre of each object in the external field, we perceive a number of single lines, apparently issuing from a point opposite the upper part of the nose, and diverging from this point in the direction of the several objects in the field.

The lines are drawn in two different colours. Those connecting the objects of the right side of the field are red; those connecting the objects of the left side of the field are black.

The red lines pass to the two left sides of the retinæ, the black lines pass to the two right sides of the retinæ; and these lines intersect in their passage to the opposite eyes. When viewed, they appear as two distinct groups; the red lines being grouped on the right, and the black lines being grouped on the left side of the median line. We remark that the eyes separate the red from the black lines, and form, so to speak, of the red lines a right, and of the black lines a left eye; and we observe that, though the visual direction of these lines does not agree with the right and the left eye anatomically considered, it yet agrees perfectly with the right and the left eye *physiologically* considered. This result connects itself with, and is confirmed by, Experiment 4 (fig. 5, Pl. I.) In this instance the objects on the right side of the field only are connected with corresponding sides of both retinæ; and the result is, that a group of single lines are seen apparently passing from each object respectively, on the right side of the field, and converging towards a point opposite the upper part of the nose. *These resultant lines being all grouped on the right side of the median plane of the head.*

Physiologists have arrived at the conclusion that, in binocular vision, there is an "interweaving of the impressions of the individual eyes." In other words, that the images of the two eyes are so intermingled in the common field of vision, as to render it impossible, in binocular vision, to distinguish the images of the different retinæ. Müller thus expresses himself upon this point:—"In the perceptions of the retinæ no distinction of right and left eye exists; they are identical."[1] The facts before us would seem to show that, in truth, there is a right and a left eye; and the mistake appears to have been that of regarding the eye of the right orbit as the right eye, and the eye of the left orbit as the left eye. If, however, we regard as the right eye that portion of the visual apparatus which commands the left half of the external field, and as the left eye that portion of the visual apparatus which commands the right half of the external field, we find that there is a right, and there is also a left eye. And this is no arbitrary view, since we prove by experiment that the eyes themselves will separate and group their respective images in

[1] See Müller's 'Physiology,' translated by Dr. Baly, vol. ii, p. 1203.

the manner we have described. It would also appear that the eye of the right side consists of the left halves of the two retinæ, and that the eye of the left side consists of the right halves of the two retinæ, and that these two eyes remain separate and distinct through all the phenomena of vision.

We have, in the course of our previous observations, laid some stress upon the uniformity of visual result, obtained under the various conditions of our several experiments. *And, if we inquire under what circumstances this uniformity of result is obtained, we shall trace it, in every instance, to one dominant fact, namely, that the space between the visual lines is lost in the visual field.* Thus, if we turn to the plates illustrative of the experiments we have been considering, and observe the results of these experiments as there figured, we cannot fail to notice that they are all associated with one uniform and constantly recurring phenomenon; namely, that the two visual lines meet upon the median line, while the images of either side of the field retain their true relation to the visual line of the corresponding eye. If, then, we regard the visual line as representing through its entire length the axis of the corresponding eye, the solution of all the results now under notice may be found in the closing words of our last section. Then, as now, we were referring to the projection of the retinal pictures into space, and in explanation of the phenomena to which we were then alluding, we stated, "That the axis of each retina refers its image to the median line, while the collateral images retain their true relation to their respective centres—that is, each image respectively, to the centre of the retina to which it belongs, and further that, whether the eyes be acting separately or in concert, their direction is the same."

Our first experiment consisted in separating the nasal and temporal fields, so that each field could be submitted to distinct observation. On viewing the nasal field thus isolated, the two halves of the field were observed to approach each other and meet upon the median line, *the external field being represented in the visual field, less the space between the visual lines;* in other words, the space between the visual lines appeared to be unrecognised in the visual perception. The same thing occurs whether the objects be viewed with the optic axes converged, or with them parallel, whether (as in our second experiment) the objects in the field be connected with the nasal halves of the retinæ exclusively, or (as in our third experiment) with the temporal halves of the retinæ exclusively, or again (as in our closing experiment) with corresponding halves of both retinæ. Under all these conditions, and in every observation we have made through the whole course of our inquiries, this one phenomenon has constantly prevailed,—*namely, that the space between the visual lines is lost in the visual field.*

So far as they act in concert, the two eyes receive their respective images from the same objects in the external field; and since the eyes are placed two and a half inches distant from each other, it follows that images transmitted from one point in the external field cannot arrive at both retinæ in the direction of a single line. The question then is, how do these two lines, having distinct directions, become adjusted in the common field of vision, so as to appear in one direction? *We have shown that through the entire field each pair of the lines of transmission, as the visual lines, are referred in a direction intermediate between the two.* This phenomenon may be well illustrated in the following manner by means of elastic threads. Place a strong pin upon the median line where the optic axes are to intersect, and two other pins, one opposite the centre of each pupil. Next pass an elastic thread round the pin inserted where the optic axes are to intersect, and let the ends of this thread be attached, one to the pin inserted opposite the pupil of the right eye, and the other to the pin opposite the pupil of the left eye. These two threads will represent the visual lines. Then fix a number of strong pins in the position of the objects in the external field, as in fig. 1, Pl. II. To each of these pins attach two elastic threads, of sufficient length to allow of their being used to connect each pin respectively with the two threads representing the visual lines, and in the same relation as shown in fig. 1, Pl. II. This is to be done through the entire field. The threads of each side of the field are to be fixed in their true relation to each other, and are to be firmly attached, each group respectively, to the pin inserted opposite the pupil of the corresponding eye. *The threads, when thus arranged, will exactly represent the lines of transmission.*

If now the pins corresponding with the centres of the two pupils be brought to the median line, the two threads representing the visual lines will be superimposed, and simultaneously with the superposition of these two threads the corresponding threads, that is, the two threads agreeing with the corresponding lines of transmission through the entire field, will be superimposed. *So that, by bringing the two threads which represent the visual lines to the median line, we secure a simultaneous adjustment of the entire field.*

We have thought it unnecessary to compare our present, with our former experiments, or to illustrate the phenomena we now describe by means of those which have formed the subject of previous communications. But it would not have been difficult to have shown that our past and our present statements perfectly agree, and we venture to think that they will also be found to coincide with all the phenomena of ordinary vision.

The facts now before us would seem to lead to the following conclusions:

That we do not see from the orbits, or in the direction of the two eyes.

That the eyes are not the true organs of vision, but must be regarded simply as two cameras, instruments for gathering in the retinal images, but having no action *external to themselves.*

That we see in the direction of the median plane of the head as from one central eye, and that this central eye consists of two symmetrical halves, the right half comprising the right halves of the two retinæ, and commanding the left side of the external field; the left half comprising the left halves of the two retinæ, and commanding the right side of the external field.

is, that the visual lines appear superimposed in the direction of the median plane of the head; and that we see a series of divergent lines apparently issuing from the upper part of the nose and passing to the objects on the right side of the field, *these resultant lines being all grouped on the right side of the median line.* (See fig. 7, Pl. I.)

For Experiment 5 we again use the same objects, and view them under precisely the same conditions; excepting that for this experiment, we connect each object with corresponding parts of the two retinæ, by means of two lines drawn from the centre of each object in the field, and passing one line to the nasal side of the corresponding eye, and the other line to the temporal side of the opposite eye. The objects of the right side of the field are connected by means of red lines, and those of the left side of the field by means of black lines. (Fig. 1, Pl. II). When viewed as before, the visual result appears the same as in our former experiments—a number of straight lines are seen, apparently issuing from the upper part of the nose; one of these resultant lines passing in the direction of the median plane of the head, while the others are divergent lines, grouped on either side of the median line, and passing to the corresponding objects in the external field; *in short, each two of the lines of transmission, when submitted to view, coalesce and form one line intermediate between the two.* This, the line of projection, results, then, from a combination of the two corresponding lines of transmission, and thus through the entire field, the lines of projection have a direction exactly intermediate between those of the two corresponding lines of transmission; they apparently issue from the upper part of the nose, and pass each respectively to the corresponding object in the external field. It is to be observed further, that the red lines appear grouped on the right side of the median line, while the black lines appear grouped on the left side of the median line. (Fig. 2, Pl. II).

We have already stated that the experiments above described are to be considered from four distinct points of view; and first, as they exhibit the great tracts of the retinæ in their relation to the external field. In our first experiment it is shown that the two nasal halves of the retinæ, when

Fig 1.

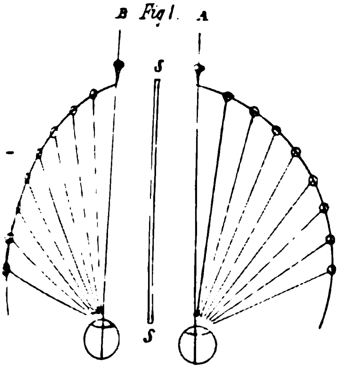
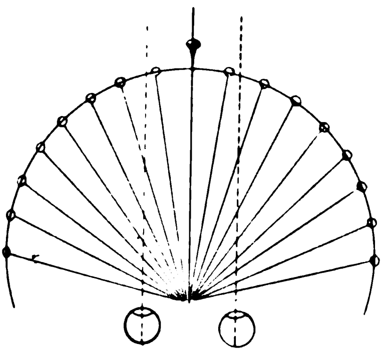


Fig 2



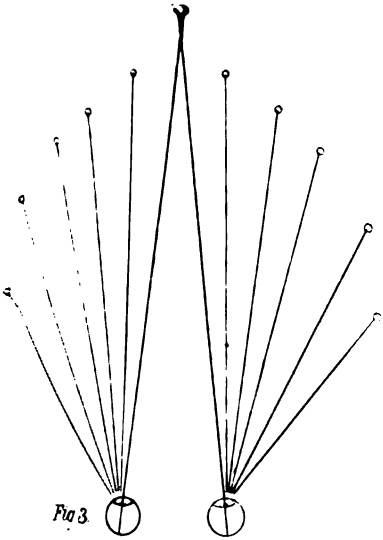


Fig 3.

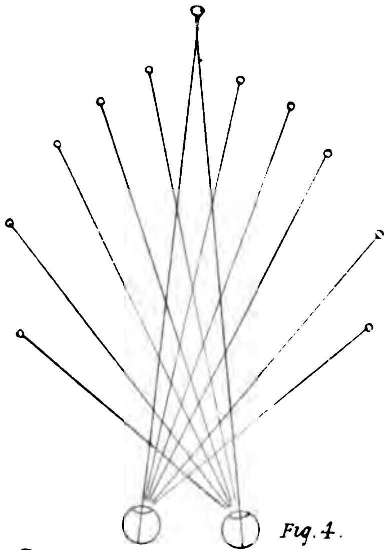


Fig. 4.

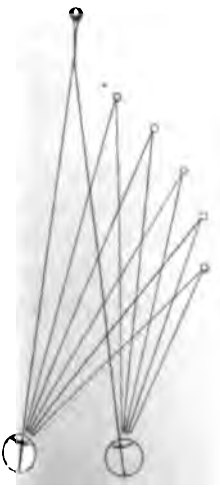


Fig 5

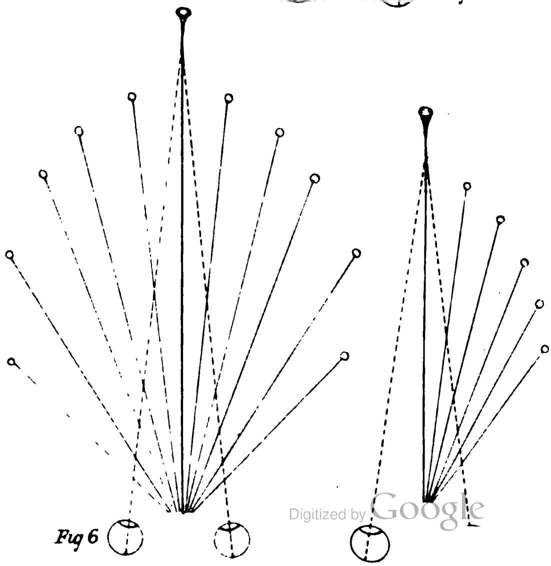


Fig 6

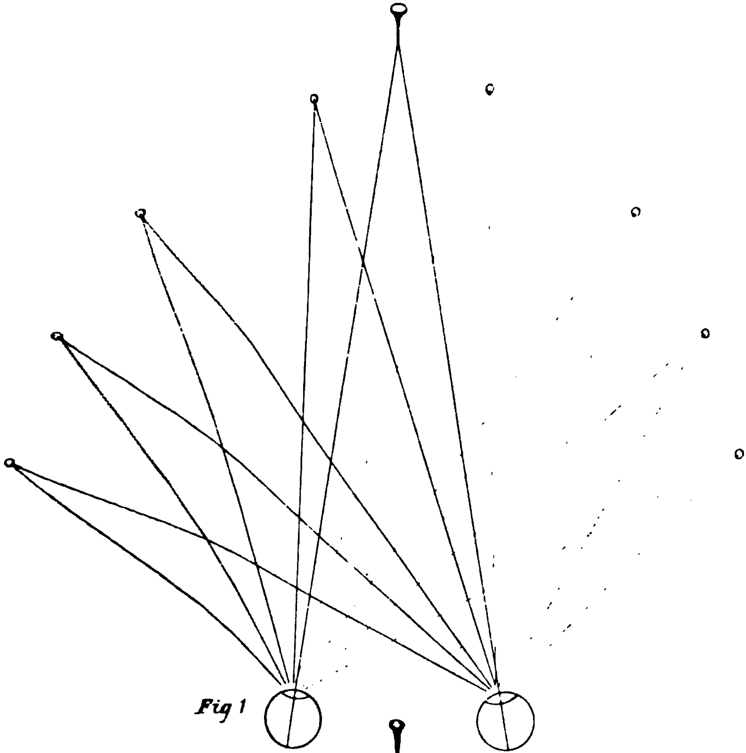


Fig 1

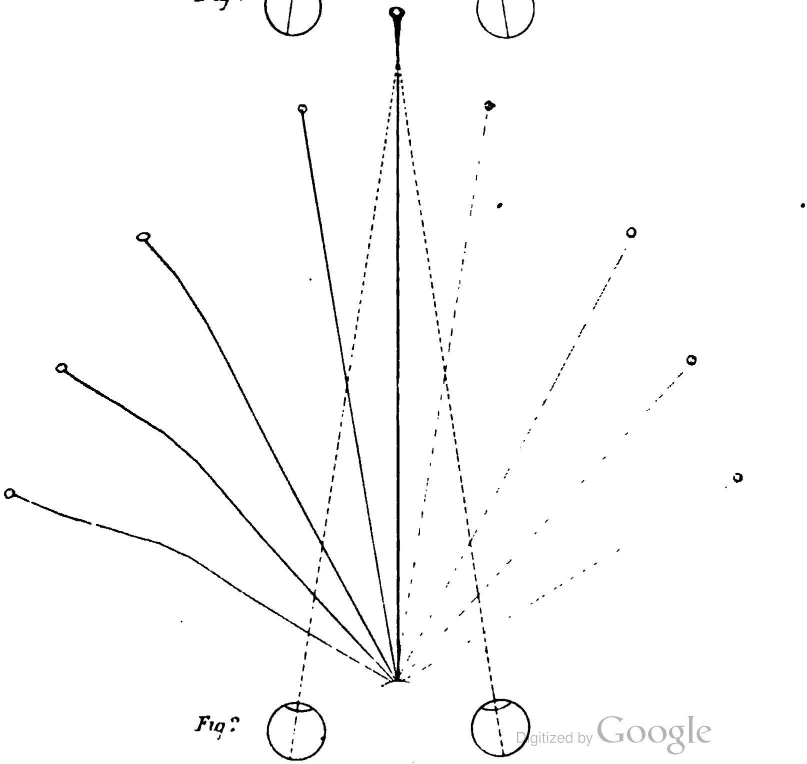


Fig 2

CLINICAL REMARKS

ON THE

TREATMENT OF DISEASES OF THE HEART.

By S. O. HABERSHON, M.D.

No class of diseases excite greater alarm than affections of the heart, and in none is greater relief afforded by right treatment; it is not, however, by any direct remedy or specific that alleviation can be effected, but rather by attention to certain plans or principles of treatment, which must serve as our guides in such cases.

New remedies appear, to keep pace with the deep-rooted and increasing scepticism as to the value of drugs: and, whilst some would merely watch disease, deploring that they cannot enlarge a contracted cardiac valve, or repair an imperfect one, others, by too great an activity and meddling, interfere with reparative action, and do more harm than good. Even amongst the numerous cases of *organic* disease of the heart brought before us, we scarcely know of any, in which right *principles* of treatment will not avail us to some extent; and when they cannot serve us in curing disease, they at least, mitigate its severity, relieve its distress, and in many comparatively hopeless cases, they even restore the patient to ease and activity.

These principles of treatment, in all cases of heart disease, are:

1st. To lessen the work of the heart.
2nd. To ensure regularity of action.
3rd. To lessen distension of its cavities.
4th. To prevent syncope.
5th. To strengthen the muscular fibre of the heart.
6th. To hinder the fibrillation of the blood in the heart and great vessels.
7th. To prevent secondary complications, and to relieve them when produced.

The *first* principle of treatment in disease of the heart is, as far as possible, to LESSEN ITS WORK; and this may to some extent be effected by *mechanical rest*, by *a recumbent position*, and by *the avoidance of sudden changes of temperature.*

Rest.—During exertion there is increased pressure on the vessels, especially upon the small capillary arteries; greater power is therefore required to propel the blood onwards; and, again, the contraction of the muscles, and the tense condition of the fascial coverings, in many parts of the body, increase the pressure upon the veins; and since the direction of the blood in the veins is uniform on account of the valves, the blood during muscular contraction is driven with greater force towards the heart, which becomes correspondingly distended, especially on its right side. Still further, in order to obtain a fixed basis on which the muscles of the arms may exert their energy, the chest and the shoulders are maintained in a stationary position, the breath is held by the firm contraction of the respiratory muscles; and as a consequence of impeded respiration the blood is hindered in its course from the right to the left side of the heart, and another source of distension and embarrassment is produced. These facts show, that if we would lessen the work of the heart, we must avoid taxing its energies by muscular action of any kind.

A very fertile source of organic disease of the heart is excessive muscular exertion. Up to a certain point the exercise of the strength of the body is beneficial, but beyond that point, it is a positive cause of disease. Athletic exercise conduces to the proper development of the whole muscular frame, but when the strength is overtaxed disease is very quickly induced. In this way those recreations which may strengthen and do good to the system, by excess become fraught with danger; by straining, as in rowing, and especially in boat-racing, the aortic valves become stretched, the aorta dilated, and at length the valves give way, allowing of regurgitation of blood into the ventricle of the heart; or the aorta becomes so enlarged that aneurism is the result.

In the treatment of heart disease, if acute or severe, the patient should remain in bed or upon the sofa, and if chronic all stairs should be avoided, for few things try the heart more severely than ascending steps; kneeling and stooping should be abstained from, for the raising of the body from these positions produces sudden increased cardiac action; straining of all kinds should be strenuously guarded against.

Recumbent position.—Whilst the body is placed in a horizontal position the heart works more easily, for the blood is propelled on the same plane; but it requires greater power when the column of blood is erect. This fact is often exemplified in cases of dilatation of the heart; the muscular strength may be quite sufficient for the horizontal position, but altogether inadequate for the erect one. As soon as the latter position is assumed violent palpitation with irregular action is induced, and actual syncope may be the result. In persons of tall stature the heart must be much more powerful than in shorter people, in order that it may properly complete its work; but many, who have attained to six feet in height and more, have a compressible pulse, and, unless care be used (especially during growth) in maintaining the strength, dilatation of the cavities of the heart quickly comes on. In a gentleman who was nearly 6 feet 4 inches in height extreme palpitation and irregularity were entirely relieved by this means—the recumbent position on the sofa. Months of distress had been passed in fruitless efforts to obtain relief, until, by retaining this position for many weeks, the ventricle of the heart at length regained increased power. The maintenance of a recumbent position is likewise of great importance when the heart is enfeebled from other causes, and the disregard of this rule has, in not a few instances, caused sudden syncope.

When the heart has become degenerated by fatty change, or when its muscular structure has been weakened by cardiac or other disease, as carditis or pericardial effusion, the increased exertion required from the heart may cause its action to cease altogether. The fact to which we have adverted is doubly important when sedative remedies, such as digitalis, have been given; whilst they quiet the action, they diminish the power of the heart, and the strength which is sufficient for a horizontal is unequal to an erect position; so that on the patient suddenly sitting up or getting out of bed the heart ceases to beat, and dangerous, if not fatal, syncope results. Some have spoken of this effect as of a cumulative character, that is to say, that the digitalis was gradually stored up in the system, and that it suddenly induced this excessive action; of this, however, there is no proof; and the severe result is rather to be regarded as the consequence of the heart's action being enfeebled by the administration of the drug to an extreme degree.

The avoidance of sudden changes of temperature.—Cold, acting upon the surface of the body, produces contraction of the peripheral capillary vessels. In this way the blood is driven from the surface into the deeper vessels and to the heart, thus increasing distension on the right side of the heart and causing a feeling of oppression. It is probable that greater force is required to propel the blood through the capillary arteries, for nutritive changes are lessened by sudden reduction of temperature on the surface, and, the elementary attraction by the tissues being less, the peripheral circulation is proportionately retarded. To some extent, also, the circulation of the blood through the air-cells of the lungs is temporarily impeded, and thus the distension on the right side of the heart is increased.

It is important to remember these facts in cardiac disease, and we have known patients with degeneration of the valves to have severe angina pectoris or dyspnœa induced by suddenly going from a warm room into the cold open air. Warmth of the chest and shoulders should also be scrupulously maintained in cardiac disease; the patient should be made to wear flannel, and in acute affections, such as pericarditis, the chest should be covered with cotton wool.

The *second* principle of treatment in heart disease is to ENSURE REGULARITY OF ACTION; and this may be effected by *avoiding mental excitement*, by *guarding against indigestion*, and by *never allowing constipation to continue.*

Mental excitement.—The vaso-motor nerve and its large cardiac ganglia are so intimately connected with the cerebrum, that any agitation of the mind at once tells upon the great centre of circulation. Any untoward news or exciting event is at once felt at the heart; even pleasurable excitement is quite sufficient to disturb its uniform action. The sudden entrance of a friend or relative may cause embarrassment or distress; and conversation, in itself pleasurable, may induce sympathetic excitement. In fact, everything which acts powerfully upon the emotions should, if possible, be avoided in cardiac disease. The more calm and quiet we can keep the senses of sight and hearing, the better will it be for the patient when affected with acute disease of the heart, as rheumatic carditis, pericarditis, &c.

Indigestion.—In many respects it is well that the appetite is impaired in acute cardiac disease; for after ordinary digestion, partly from distension of the stomach, from increased excitement of the vaso-motor nerve, and from a larger quantity of fluid being absorbed into the circulating medium, there is increased excitement of the heart. This excitement becomes still more marked when there is any disturbance of the heart itself from disease. In chronic affections of the heart with obstruction, the mucous membrane of the stomach becomes greatly congested, there is a large quantity of mucus excreted, and digestion is greatly interfered with; soon after a meal, especially of solid nitrogenous food, distension of the stomach takes place, and, as a consequence of this distension, the diaphragm is pressed upon, the heart's action is embarrassed, and a sense of great distress is experienced. Irregularity and palpitation are thus induced; and for several hours extreme dyspnœa is occasioned. It is probable that in some instances, in which a fatal result has taken place during sleep, the distension of the stomach, due to the slow digestion of a supper or late meal, has greatly conduced to the disastrous termination.

In acute disease of the heart a spare diet, without very much of solid material, should be taken; although stimulants are in some cases required. In chronic affections of the heart, with obstruction and distension of the right side, the diet must be carefully regulated. It ought to be simple in its character and easy of digestion, and the principal meals should be taken during the earlier part of the day.

Constipation.—When the bowels are confined there are two sources of increased embarrassment to a damaged heart; the one is distension of the colon, by which the diaphragm may be pressed upwards, and so the heart's action be interfered with; and the other, perhaps the more important, is, that constipation promotes congestion of the portal veins, affecting first the liver, and subsequently the right side of the heart. Many patients with organic cardiac disease have, in their own experience, found that as soon as the bowels become constipated their dyspnœa is increased, and so also the palpitation and irregularity of the heart. In selecting purgative medicines, it is well to employ those which act upon the small and large intestines, and to avoid those which irritate and distend the stomach. A free mercurial purge is often productive of great benefit, for it not only unloads the bowels, but it relieves congestion of the portal system, and acts upon the kidneys as well as the other abdominal glands. The magnesian purgatives efficiently relieve portal congestion, whilst they remove constipation; colocynth with henbane, and the gamboge pill with henbane, may be used with advantage, as also may the compound jalap powder; or aloes, alone or in combination, as the decoction of aloes with the old compound gentian mixture of the London Pharmacopœia.

Many of the milder laxatives, as vegetable diet, &c., are ineffective, for they distend the stomach, and produce flatulence without affording relief. Enemata are of greater service, as those of soap, colocynth, castor oil, &c. The milder the means that are employed the better, if they are efficient in their action, and, if possible, it is desirable to avoid repeated action which greatly fatigues and distresses the patient. The resin of podophyllum sometimes acts well; but it is uncertain in its effects, and in some cases I have often found it cause unnecessary griping pain.

The *third* principle of treatment is to LESSEN DISTENSION, especially of the right side of the heart; and this is effected *by purgatives,* by *diuretics,* and by *mechanically diminishing the quantity of fluid in circulation.* It is in chronic disease of the heart that this principle of treatment is especially to be carried out, but in acute affections it is also applicable. We have already referred to the injurious effects of constipation in increasing the distension of the right side of the heart and the cardiac embarrassment; but it is often desirable to act very freely on the bowels, so as to exert a powerful effect on the whole portal circulation; and this is, at the same time, still more effectually relieved if the kidneys are stimulated to more energetic functional activity. The urine is oftentimes scanty and high-coloured in chronic cardiac disease, and there are deposits of lithates with phosphates, and with the ammonio-magnesian phosphate. The long-continued engorgement of the renal vessels is also followed by an albuminous condition of the urine, whilst the actual quantity is reduced from 40 or 50 oz. to 10 or 15 oz. in the course of the twenty-four hours. The liver is in a similar state of engorgement, and the bowels become irregular; the complexion is sallow or semi-jaundiced, and there is a tendency to purpura. This great distension is an indication of the amount of obstruction in the heart itself; and the distension of the cavities of the heart is always greater than the secondary congestion. It is of great importance, then, to relieve this distension, and it is most effectually done by the means already indicated, namely, by diuretics and by purgatives. A very favorite combination of my late senior colleague Dr. Addison consisted of a grain of calomel, a grain of powdered digitalis, and a grain of powdered squill, given night and morning for several days. These remedies act powerfully upon the whole of the abdominal glands; free bilious evacuation takes place, a larger quantity of urine is excreted, and the venous distension is greatly lessened, and secondarily the right side of the heart is relieved of its excessive fulness. The pulsations become more free, the left side acts more energetically, and the pulse, previously small, irregular, and intermittent, may become fuller and more regular in its action. Gray powder, blue pill, or the black oxide, may be substituted for the calomel, the squill pill for the simple powder, and the foxglove lessened or omitted. With these agents saline diuretics and purgatives may be advantageously combined; as the acetate of potash with nitrate of potash and sulphate of magnesia, or with iodide of potassium, sweet spirit of nitre, infusion of broom, spirit of juniper, &c.

In some cases the action of the right side of the heart almost ceases from over-distension; and we have witnessed almost instantaneous relief obtained by mechanically withdrawing part of the distending fluid; that is, by opening a vein and removing a few ounces of blood. Great care is, however, required, or subsequent exhaustion may be the result. It is less hazardous to apply a cupping-glass between the shoulders, but the relief is less speedy. In acute pericarditis following scarlet fever, with albuminous urine, we have observed very speedy relief afforded by the application of cupping-glasses; the urgent dyspnœa has been lessened, and the pulse has become calmer and more regular. In chronic disease, accompanied with ascites and anasarca, mechanical relief is obtained by puncturing the thighs and allowing the serum to exude, or by superficial ulceration of the legs, and consequent exudation, or, again, by *paracentesis abdominis*.

The former exudations, however, often suffice for the discharge of serum from the peritoneum itself.

In making punctures in the lower extremities, it is well that they should be above the knee; and they are best made with a grooved needle, introduced obliquely without penetrating the deep fascia. If made in the foot or leg, erythematous or erysipelatous inflammation is very apt to follow, and, it may be, that ulceration or gangrene is the result.

The *fourth* principle to be regarded in the treatment of cardiac disease is the PREVENTION OF SYNCOPE. There is great tendency in several forms of heart disease to terminate in this manner; thus in acute pericarditis, especially with effusion into the pericardium, danger from this source is often very great. In fatty degeneration there may be the sudden cessation of muscular action and fatal syncope. In simple dilatation of the heart, or when regurgitative disease of the aortic valves is the cause of that dilatation, we have especially to bear in mind this tendency. The left ventricle becomes so distended that great power is required to empty the enlarged cavity; and if the muscular power fail, a state that might be designated paralysis of the heart results, the ventricle cannot contract, and sudden death follows. This dangerous state of muscular weakness is not, however, limited to the left side of the heart; the right side is likewise affected both with fatty degeneration and with over-distension. The former state is sometimes the cause of sudden death after intemperance and delirium tremens. The patient, having been composed by medicines, rests apparently in calm sleep; then, awaking, he suddenly sits up, and the enfeebled and degenerated heart, unable to bear the sudden afflux of blood, at once ceases to beat. How important, then, to bear this fatal tendency constantly in view! *Sudden muscular movements must be avoided; stimulants may be required,* as ammonia, brandy, &c., and *sedatives must be withheld or cautiously administered.*

As to muscular movements we have already referred to their importance in the treatment, both of acute and chronic heart disease. In some cases of acute pericarditis there is no pain, no distress, no dyspnœa, no febrile excitement, nor even quickened pulse; the patient is surprised that he may not get up and walk about, but such a proceeding would be fraught with danger to life.

Again, in many acute diseases, as rheumatic pericarditis and carditis, the action of the heart is so enfeebled, that it is desirable to administer brandy with caution; so also in chronic disease with dilatation, ardent spirits are frequently necessary; but the stimulant effect of ammonia is often of great value in these cases, by lessening the tendency to syncope; the sesquicarbonate of ammonia may be given freely; it apparently diminishes the irregular action and the distressing palpitation. It may be given with camphor water; and when mixed with syrup, 10 or 20 grs. of ammonia may be taken without irritating the mucous membrane, but, on the contrary, with great benefit. In a case of chronic valvular disease, with extreme tendency to palpitation and syncope, scruple doses of ammonia were followed by quieter action of the heart; the severe symptoms lessened, and life was prolonged for several years. The patient, himself a medical man, attributed his recovery from that alarming attack to the stimulant effect of the ammonia. I generally, however, give it in smaller doses.

In degeneration of the heart, whether from intemperance or otherwise, it is better to employ ammonia than to resort to the free use of ardent spirits, and in the former class of cases they are wisely abstained fròm altogether.

Sedatives.—Under this appellation, I refer especially to those remedies, which quiet the action of the heart by their effect on the vaso-motor nerve, and, therefore, not necessarily to narcotics; thė term is often incorrectly applied to remedies which directly produce sleep. Foxglove, antimony, tobacco, lobelia, tea, and in some patients chloroform, chloric ether, and morphia, thus act upon the cardiac plexus of nerves and reduce the power of the heart's action. They lessen irritability, but they only do it by diminishing power; hence the danger of their use where there is a tendency to syncope. Digitalis acts upon the kidney as a diuretic, and in this way lessens distension; but, in its sedative action, its use requires great caution. Tea is better avoided altogether; tobacco still further weakens the heart, and so also does lobelia. Chloroform, chloric ether, and, in a less degree, morphia, produce in some patients a sense of extreme faintness. The latter remedy can, however, sometimes be borne in very small doses with advantage, when larger ones are followed by distress.

The great object of all treatment in disease of the heart is to enable it to perform its required work efficiently; this can only be done when the muscular fibre of the heart is sufficiently strong, and the *fifth* principle of treatment is to STRENGTHEN THE MUSCLE. To attain this, suitable *nourishment* must be administered; if other conditions will allow, the *air should be bracing; chalybeate medicines* may be tried; and if the patient be exhausted by want of sleep, that symptom must, if possible, be relieved.

Nourishment.—The diet of a patient affected with cardiac disease is a subject of great importance, not only on account of the strength that may be afforded by nutritious food, but from the ill effects of dyspepsia, as we have before remarked. If the supply be insufficient, there is additional danger of dilatation of the ventricles.

Bracing air.—Few patients are more susceptible to atmospheric changes than patients affected with disease of the heart;

even in acute disease, patients soon feel oppressed, as if the free depuration and arterialization of the blood promoted the regular action of the heart. Still more is this the case in chronic disease, with distension of the right side of the heart, and with an almost persistent congestion of the lungs. These patients long for a free supply of cold air; even with urgent dyspnœa and great obstruction patients are benefited by change from an oppressive to a bracing atmosphere. We have known patients with organic disease of the valves apparently relieved even by horse exercise. This, however, requires an amount of muscular exertion which few persons with cardiac disease can bear. Hot and close rooms should always be avoided; the sleeping apartment should be airy; and, if possible, patients should reside upon a dry gravelly or chalky soil, rather than upon clay.

The want of sleep and the disturbance of the hours of the night by increased palpitation are common symptoms of heart disease; and it often happens that as soon as the patient falls asleep he awakes with sudden alarm. Nights are passed in sleepless weariness, until the patient becomes completely exhausted, and the general weakness and feebleness are increased; and unless rest be obtained, the irritability of the heart and all the symptoms are greatly aggravated.

Henbane, belladonna, morphia, or opium, may be used; but the latter should be combined with remedies, which prevent its confining the bowels and diminishing secretion.

Chalybeates.—In some instances of cardiac dilatation, with anæmia, the preparations of iron are of great service; and we have known irregularity very quickly subside after their administration. The great danger in many forms of organic cardiac disease is from dilatation, which is expressive of enfeebled power; and if the preparations of iron can be borne, they are very useful in increasing the muscular vigour. The mistake is often made of giving them in too large doses or on an empty stomach, thereby inducing excitement and increased palpitation. The milder preparations, such as the ammonio-citrate, the potassio-tartrate, or the phosphate, or a few minims only of the tincture of iron may therefore be given. The chalybeate waters are sometimes of benefit, for they combine, when the patient visits their localities, not only the good effects of an invigorating medicine, but the bracing results sought for by a change of air.

The next principle to be borne in mind in the treatment of disease of the heart is to PREVENT FIBRILLATION OF THE BLOOD. When inflammatory mischief involves the valves and the endocardium, the lining membrane becomes œdematous or thickened, and there is an associated deposit in most cases upon the valves themselves. These fibrinous deposits may be a minute fringe of granules or a more general thickening. They may lessen in size, and perhaps become removed, or they may remain and undergo various conditions of change, such as ulcerative action, contraction, atheromatous and ossific change, &c. Their presence produces irregularity of the surface; and, under certain conditions, fibrillation takes place upon previous deposit, and thereby greatly aggravates disease; and again, when the circulation of the blood is less active, and when great distension of the cavities of the heart has occurred, there is a great tendency for the blood to fibrillate and to form ante-mortem coagula, as is the case in an aneurismal sac. Some of the smaller fibrinous clots may be carried along into the current of the blood, and produce obstruction in the circulation of the glands or in the brain. In the latter case sudden symptoms of brain affection are produced. On the right side of the heart the blood is still more liable to coagulate when the circulation is retarded; and the fibrin then concretes in the auricle, or at the apex of the ventricle, or in the pulmonary artery. During chronic valvular disease sudden faintness may come on, and a death-like pallor, from this hindered circulation, and in not a few cases it is the immediate precursor of a fatal issue. The condition has sometimes been spoken of as "polypus of the heart;" and we may also add that in numerous acute diseases, as pneumonia, &c., this ante-mortem fibrillation and coagulation is the cause of the rapid failing of the pulse and of the pallor which immediately precede the closing scene of life. To hinder this fibrillation, then, is most important, but in the means we possess we are not able to verify hypotheses that have been made; the physiological action of ammonia in reference to fluidity of the blood-constituents is not established, and it is doubtful how carbonate of ammonia acts in relieving the symptoms of heart disease; but there is no doubt that in some cases of chronic valvular disease it is of great value. It acts primarily as a stimulant upon the cardiac ganglia; but this effect is a transient one, and the secondary effect is equally important. The extreme irregularity of the heart's action is mitigated, and the patient is able to recline more easily. I have seen great benefit accrue from this remedy when the dose has been gradually increased.

Other alkalies.—Potash, soda, and their salts, may hinder the fibrillation of the blood, but, if given continuously in considerable doses, they depress the action of the heart; thus the iodide and acetate of potash afford relief in a greater degree than is due simply to their diuretic effect, and we attribute part of the good result that follows their use to their action upon the blood. They may be advantageously combined with the carbonates of ammonia or, perhaps, with the hydrochlorate of ammonia.

In many conditions of cardiac disease there is a great tendency to syncope; and during these attacks the heart's action is enfeebled, and the current of the blood is slower; there is, then, everything likely to induce fibrillation of blood in the cavities and upon the valves of the heart; and thus, in a secondary manner, *brandy* prevents fibrillation. Alcohol, in its concentrated state, increases the tendency to coagulate; but, when given in a diluted form, the direct stimulant effect of brandy is of great advantage by preventing syncope. Rightly given, brandy is a most valuable medicine in cardiac diseases; and it may be the means of greatly prolonging life and relieving the distress of the sufferer.

The next and *seventh* consideration to be borne in mind in the treatment of cardiac disease is to PREVENT SECONDARY COMPLICATIONS, and to RELIEVE THEM WHEN PRODUCED. These complications are—1st, broncho-pneumonia and pleuritic effusion; 2nd, pulmonary apoplexy and other hæmorrhages; and, 3rd, visceral engorgement, as hepatic and renal congestions, with ascites and anasarca. These complications constitute the prominent features of heart disease in its usual closing stages; and, although we are unable to take away the physical cause of all the distress, a great deal may be effected in mitigating these secondary symptoms. The bronchitis may be relieved, the pleuritic effusion removed, the hæmorrhage checked, the visceral congestions diminished, and the ascites and anasarca entirely removed.

How, then, can all this be effected? Having, as far as possible, carried out the indications of treatment previously mentioned, we must seek to relieve the hepatic and renal congestions. In this way we shall lessen the engorgement of the right side of the heart, and all the symptoms which are the sequelæ of that condition. By freely acting on the bowels, the portal congestion is greatly diminished, and the liver is enabled to act in a normal manner. Thus, a free mercurial purgative is of great value, or the compound jalap powder or the elaterium powder may be employed. The kidneys may be excited to more vigorous action; and this is best effected by a combination of mercurial medicine with squill, and with digitalis, when the latter can be borne. It is most unwise and unnecessary to produce salivation. Diuretic medicines, as the nitrate, iodide, and acetate of potash, may also be given, combined with nitric ether, squill, juniper, or broom, as the occasion may require and admit.

Another very effective way of diminishing the anasarca is by puncturing the skin on the thighs. This allows serum gradually to exude from the cellular tissue, and so directly relieves the congested vessels. This is oftentimes effected by superficial ulceration of the legs; but cutaneous inflammation and ulceration, although beneficial to the cardiac symptoms, is a source of considerable pain, and often induces erythema or erysipelas, and even superficial gangrene. Warm fomentations should be employed to promote this serous effusion, and, if the ulceration is troublesome, nitric-acid lotion with *vinum opii* may be applied. In other instances punctures may be made with a grooved needle in the thigh, for, in this way, erysipelas is less likely to follow than if the calf of the leg or the foot be selected. The punctures should not exceed six or eight in each thigh. In several instances we have seen severe ulceration and inflammation followed by great relief to the patient, in consequence of serous transudation taking place.

To relieve hæmorrhage, dilute sulphuric acid with sulphate of magnesia and gallic acid may be tried, or the acetate of lead with opium, or, again, alum or the oil of turpentine; but this hæmorrhage, as the result of intense venous congestion, is mitigated in a greater degree by lessening the congestion than by direct astringents.

The pulmonary engorgement is sometimes greatly reduced by applying cupping-glasses between the shoulders or by the application of a blister to the chest.

By these means we have oftentimes witnessed patients who were apparently in a hopeless state, and suffering from extreme dyspnœa and exhaustion, restored to comparative comfort, so that they were enabled to take part in the ordinary duties of life, although the original physical defect in the heart continued. The same principles of treatment are applicable in acute as in chronic disease, whether acute pericarditis, rheumatic carditis, &c., for while we seek to remove and check the immediate results of the malady, as the effusions of fibrin and of serum, we must bear in mind the nature of the organ affected and the method in which a fatal issue takes place; and above all, we must treat, not the symptoms, nor the local disease, but the patient.

The following cases of heart disease were under our care during the past session in the clinical wards. They afford illustrations of the practical application of the principles we have laid down in the previous remarks, and they also suggest many points of interest in relation to diagnosis and pathology.

Contraction and adhesion of the valves of the pulmonary artery (congenital?).

(Reported by Mr. G. B. Stevens.)

Case 1.—John H—, æt. 19, a scale-maker, was admitted into Guy's Hospital, under Dr. Habershon's care, on May 2nd, 1866. He had generally been in a bad state of health, being always affected with shortness of breath; his mother stated that he had had a blue appearance from birth. At all times he had been very susceptible to cold, and especially during the winter months. Three years ago, during the winter, he had an attack of dropsy; but as summer approached he got better, and continued so during the warm weather. In the following winter, however, he was again not so well. On these occasions he passed little water, and an improved condition was always preceded by copious diuresis. During the last two years he had been subject to fainting, brought on by trifling causes of excitement.

As to family history, some of his relations had died from phthisis; his sister had had hæmoptysis, and his father bronchitis.

On admission his countenance was almost of an indigo colour, congested, and bloated. His chest was barrel-shaped, the ribs but slightly mobile, and the respiration for the most part diaphragmatic.

Percussion elicited a resonant sound under the clavicles in front, extending downwards rather more on the right than on the left side, and becoming completely dull above the nipple on the left side, and immediately below it on the right. Posteriorly, there was abnormal resonance beneath the scapulæ, as if from emphysema. The liver could be felt below the ribs, nearly to the umbilicus. There was great increase of the pericardial dulness. The respiration was puerile at the apices, and the voice increased in resonance. At the posterior part some crepitation could be heard at the base of the lungs.

The impulse of the heart was at the scrobiculus cordis, immediately below the ensiform cartilage. On auscultation there was heard most distinctly at the centre of the sternum a murmur which immediately followed the systole of the heart. This murmur could not be traced in front, in the direction of the aorta or the pulmonary artery, but it could be heard at the left apex *posteriorly*. The pulse was small, compressible, 80 in the minute, and irregularly intermittent. The abdomen was full and large, and the superficial vessels congested; there was some fluctuation. The lower extremities were slightly anasarcous, and the feet and legs intensely congested, as were also the hands and fingers. The urine was turbid and highly albuminous. The bowels being confined, he was ordered to take the saline rhubarb powder of the 'Guy's Pharmacopœia' every morning. By this treatment the bowels were freely acted upon and the dyspnœa was greatly relieved, and he was able to walk about comfortably. Some quinine was given. It was thought that he might leave the hospital; but on May 24th, after a bad night, sudden faintness came on, and he expired in about half an hour.

Post-mortem inspection.—Rigor mortis was well marked. Decomposition had commenced on the abdomen. *Brain.*—Cranial bones very full of blood. *Chest.*—Bronchial tubes reddened, and coated with a firm creamy layer of pus; the small tubes filled with muco-purulent secretion. Both lungs emphysematous; very moderate congestion of blood.

Heart.—The pericardium contained about fifty ounces of serum; the membrane was thickened, but there was no recent lymph. The heart was much enlarged, especially from the great distension and hypertrophy of the right auricle and ventricle. The right auricle was four times its natural size, and as thick as an ordinary right ventricle. The right ventricle also was greatly hypertrophied, and the carneæ columnæ of the tricuspid valve were enormously enlarged. *The pulmonary sigmoid valves had coalesced,* forming a *rounded cone, convex towards the artery,* at the apex of which cone was an aperture, only allowing the passage of a quill (see Plate). The pulmonary artery was very thin, and the vessel distended. The left ventricle was normal. The peritoneum contained a small quantity of liquid. The glands of the intestine were distinct.

The *Liver* weighed eighty-three ounces; its surface was granular and whitish. Large patches about the hepatic vein had lost their secreting structure, fibrous relics alone remaining; the whole gland was greatly congested. The *Spleen* was large, weighing ten ounces. Its capsule was thickened. There was an indentation at its upper part, as if from loss of substance, and there was much fibrous tissue at that part. The kidneys were dark and congested.

In connection with the case just detailed there are several points that demand our notice. 1st, the diagnosis; 2nd, the prognosis; 3rd, the indications for treatment; 4th, the pathology of the case.

1. The blueness of the countenance and the extreme congestion of the vessels were well marked in this case. The state was one of cyanosis, and this condition dated back to the

earliest years of life. The lips and face, the hands and feet, were almost of an indigo-blue colour, and the depth of the colour was the effect of more than mere congestion; it was evidently due to imperfect arterialization of the blood. Again, this at once pointed to some congenital defect about the heart, and an imperfect septum between the ventricles or an open foramen ovale, or an open ductus arteriosus was at once suggested to the mind. The physical signs were, however, not those which we have usually observed in these conditions. There was great cardiac dulness; in fact, the whole of the lower half of the left side was dull, and so was also the right hypochondriac region. The impulse of the heart was forcible, but the pulse was small. There was no bruit over the aorta, but a slight one was audible at the sternum, and the hypertrophy of the heart was at once referred to the right side, for we had no evidence of aortic obstruction. In ordinary instances of imperfect septum between the ventricles, and of open ductus arteriosus a loud bruit is heard, extending in the direction of the pulmonary artery towards the left shoulder; no bruit of this kind was present, although one was heard in the left supra-scapular region; and the nature of the malady, therefore, differed so far from ordinary congenital defect. The congestion of the liver, kidneys, and the whole systemic circulation, was attributed to the existence of obstructive disease of the heart; for although no regurgitative or diastolic bruit was audible over the aortic or pulmonary valves, a persistent post-systolic bruit was found over the centre of the sternum.

This bruit was heard immediately after the systole, and did not exactly accompany it like an ordinary systolic murmur; it was produced, we believe, by the obstruction at the pulmonary orifice, and was but faintly audible on account of the large amount of pericardial effusion. The dulness was due partly to this effusion, partly, to the enormous hypertrophy of the right side of the heart. The presence of so great a quantity of serum increased the embarrassment of the heart's action and the tendency to syncope; but the faintness to which he had been subject for several years was caused principally by the circumstance that only a small quantity of blood reached the left side of the heart, and consequently the substance of the heart and the brain were imperfectly supplied. The bruit at the pulmonary orifice would, we believe, have been more distinct had not a stratum of serum intervened, and prevented the free transmission of sound.

In the diagnosis we were led to believe, that the seat of the disease was on the right side of the heart, and that it had given rise to great hypertrophy. We also concluded that it dated from the earliest years of life, even if it were not congenital. There was no history of past attacks of rheumatism, either in the patient or in his parents.

As to the prognosis, the nature of the case forbade us to expect that we could do more than palliate the effects of the disease. For a time the treatment was so far successful, that the patient became relieved of his distressing symptoms, and the thought of his returning home was entertained.

The principles of treatment were those to which reference has been made, and we were surprised to observe how greatly the dyspnœa was lessened.

As to the pathology of the case, there can be no doubt that an inflammatory change at the pulmonary orifice had taken place, either during fœtal or infantile life. These valves thus became thickened and contracted, and adhered together, so that the opening was abridged, and measured only a quarter of an inch in diameter. We cannot affirm that the character of the change was rheumatic, but it was certainly not congenital in the sense of being an arrest of development. The obstruction at the entrance of the pulmonary artery was the cause of the other symptoms; for the blood could not reach the lungs so as to be properly arterialized; and the right ventricle and auricle became enormously distended and hypertrophied.

The same obstruction led to great enlargement and congestion of the liver, the blood being unable to pass from its structure. The kidney was in a similar condition, and this state was the cause of the albuminous condition of the urine. The superior and inferior cava being intensely distended, all the minute systemic capillaries were in a similar state of engorgement, and the extreme peripheral circulation presented the characteristic lividity to which we have before referred. Although the pericardium was thickened, the effusion was of a passive kind, and the result of the extreme distension of the ordinary veins.

The next instance may be taken as a sample of very many which are brought before us. It is a case of organic disease of the aortic valves, brought on by over-exertion and increased by intemperate habits. It was relieved by the principles of treatment which we have enunciated, but the relief persisted only so long as those principles were carried out.

Imperfect aortic valves; obstruction and regurgitation; relief by treatment.

(Reported by Mr. G. B. Stevens.)

Case 2.—William J—, æt. 42, an excavator, was admitted, under Dr. Habershon's care, June 7th, 1866; he had enjoyed good health till November, 1865. He had been accustomed to drink freely, but had never suffered from rheumatism. In November, 1865, after drinking to excess for three days, he was seized with dyspnœa on getting up in the morning; this did not, however, last long, and he went to his work as usual. In December he took a severe cold, and had a return of the dyspnœa, with cough; at the same time he suffered from a stabbing pain under the left nipple, and a sense of fulness at the epigastrium. His urine was high-coloured. At that time he was ill for five or six weeks; his legs were slightly swollen, but there was no affection of the joints. Afterwards he again tried to work, but could only go on for about a fortnight on account of the shortness of breath. Emetics were then given to him by some quack doctor, and produced great distress. He made repeated attempts to work, but, as before, was soon compelled to desist.

On admission, he appeared strong and comparatively well, but had an anxious expression of countenance. The body was fairly nourished, and when at rest he could breathe comfortably. There was no cough. The respiratory sounds were normal, but rather feeble. There was increased cardiac dulness. At the fourth left costal cartilage a loud double bruit could be heard. This murmur could be traced in the direction of the right sterno-clavicular articulation, and could also be heard in the right subscapular region posteriorly. The impulse of the heart was strong and nearer to the left axilla than normal. The pulse 84, possessing, though not in a very marked degree, the water-hammer character of regurgitation through the aortic valves. There was no enlargement of the liver, and no ascites or anasarca. The tongue clean; the urine normal. Magnesia mixture was given to act on the bowels, and extract of henbane was ordered to be taken night and morning. Afterwards the nitro-hydrochloric acid was prescribed.

There was a remarkable tendency in this patient, on the least exposure, to the occurrence of attacks of bronchitis, in which mucous râles were heard over all parts of the chest. The congestion of the lungs consequent on valvular obstruction on the left side of the heart, no doubt, favours bronchial irritation. But this was unusually marked in this case, and the great distension of the left auricle, by producing pressure upon the bronchi, as first suggested by the late Mr. Wilkinson King, may, perhaps, have had some causative relation with these attacks. The first attack was soon relieved by rest in bed, and by the administration of acetate of ammonia, nitric ether, and small doses of morphia. A second time this bronchitis came on, but quickly subsided. Quinine was tried, but it soon produced giddiness. The rest in the hospital and the sustaining diet he there received afforded much relief, and when he left he was stronger than on admission, but was still quite unfit for any active muscular exertion. There was then a slight systolic bruit below the nipple, and it was probable that the mitral valve had begun to yield from over-distension. The mitral defect probably modified the character of the pulse, for when both the mitral and the aortic valves are imperfect, the pulse has less of the water-hammer character.

The great exertion to which this man had been subjected in his laborious occupation, and the consequent stretching of the aorta, had, we believe, induced this disease, but it had doubtless been aggravated by his intemperate habits. A cure cannot be expected in these cases. The damaged valve can never be restored; but, with care, such patients may be kept in comparative comfort for many years.

In the next case we have an unusual complication of disease.

Aortic and mitral disease of the heart, with chronic phthisical pneumonia, in a man of intemperate habits; purpura; death.

(Reported by Mr. G. B. Stevens.)

Case 3.—John C—, æt. 44, was admitted June 13th, 1866. He was a window-cleaner, and was of intemperate habits. The account he gave of himself was that, till Easter, he had been pretty well, except that he suffered from winter cough; but that his ankles then began to swell at the close of the day. Four weeks before admission, he noticed some purple spots on his legs; but, after resting at home, he was able again to resume his work. The spots, however, returned, more severely; and on his admission, they covered the thighs and legs. In the beginning of June, he had noticed that the abdomen was larger than usual. For several years he had been troubled with hæmorrhoids, which had occasionally bled freely; and, of late, he had suffered from continued retching, especially in the morning.

On admission he was very ill, and appeared anæmic. The gums were very foul, and the mucous membrane of the mouth exsanguine. *Chest.*—On percussion the left side was found to be universally dull in front, and posteriorly the resonance was less than normal. There was no loss of tactile vibration. There was well-marked bronchophony over the whole of both lungs posteriorly, increasing in intensity from below upwards. This was also audible in front. There was mucous crepitation over the lower lobes of both lungs posteriorly, and also in front at the left apex. The pericardial dulness was increased, and a thrilling impulse could be felt in the region of the heart. A loud double bruit was heard to the right of the left nipple. This murmur could be traced in the course of the aorta, and it was also heard in the axilla, but not at the back. There was slight general anasarca, rather more marked in the legs and feet. The liver was much enlarged, and could be felt just above the umbilicus. The abdomen was enlarged, and fluctuation was perceptible at the lower part. The urine was high-coloured, and slightly albuminous. The pulse was 94, jerking or "water-hammer;" the expectoration consisted of a tenacious, green, muco-purulent secretion.

This poor patient was affected with incurable and advanced disease of both the heart and the lungs. He was exhausted and prostrate, broken down by intemperance as well as by disease. It was thought, that the best chance would be afforded by unloading, if possible, the engorged abdominal viscera, thus relieving the distension of the right ventricle and the congested lungs. A diuretic and alterative of squill and blue pill was given at night, and carbonate of ammonia with acetate of potash and nitric ether every four hours. The bowels acted freely, and the œdema became less. But on the 16th of June, contrary to directions, and most unwisely, he got up and walked to the water-closet. Syncope came on, and he shortly died.

Inspection.—The œsophagus was warty. The left pleura was at least half an inch in thickness, and the pleura was also adherent on the right side at the apex. The lower lobes of the right lung were red and dense (splenized); the upper lobe was small. Both upper and middle lobes were slightly emphysematous. The upper lobe of the left lung was dense and fibrous, and it contained small old cavities, its vessels were very thick, and there was scarcely any trace of normal structure; the whole lung was indurated, with scattered hard grey granulations.

The *heart* weighed 24 oz. There was great dilatation of the left auricle and ventricle, the cavity of the left ventricle being four times its natural size. It contained a large quantity of clot. The aortic semilunar valves were very bad, turned down, softened, thickened and adherent. The cordæ tendineæ of the mitral valve connected with the anterior portion of the left papilla were cut away by ulcerative action. There was a small quantity of fluid in the peritoneum. The liver and the kidneys were much congested, and the latter contained numerous cysts.

It might almost be said, that this patient came into the hospital to die; but the case deserves attention, not only on account of its great pathological interest, but as showing how important it is that patients with dilated heart and valvular disease should maintain the *recumbent position.* The effort of walking and the increased embarrassment of the heart caused by the erect position were, in this case, sufficient to produce sudden syncope. Free action of the bowels would otherwise very likely have afforded relief, and the life of this patient might have been preserved for a short time.

The aortic valves were very imperfect. In fact, they had almost ceased to act as valves; and the mitral valve, being unable to bear the great strain upon it, had become affected with acute disease, and had undergone a process of ulceration. The great dilatation of the left ventricle, without corresponding hypertrophy, observed in this case, is the most dangerous result of imperfect aortic valves, and one which frequently leads to a suddenly fatal termination.

The chronic pleuro-pneumonia, with fibroid degeneration of the lung, would alone have sufficed to destroy life; it greatly aggravated the distress and exhaustion of the patient. As to its cause, we have frequently found it in persons of intemperate habits. The immoderate use of alcoholic stimulants, therefore, had probably a most important effect in predisposing to and aggravating both the pulmonary and cardiac disease.

The next instance is typical of a class of cases unfortunately only too frequent.

Cardiac disease after rheumatism, affecting the mitral valve; relief under treatment.

CASE 4.—James M—, æt. 22, was admitted. under Dr. Habershon's care, May 2nd, 1866. He was a short spare man, married, and by occupation a clerk; he resided in Bermondsey. Although not robust, he enjoyed average health till three years previously, when he had rheumatic fever. Since that time he suffered from palpitation of the heart; his ankles had swollen, and he complained of pain in his head and shoulders, and also in his chest when lying down. The extremities easily became cold. His father had been subject to rheumatism.

On admission he was pale, and suffered from dyspnœa. There was increased pericardial dulness, and a loud systolic bruit was heard below the left nipple, and also in the axilla and at the back; the second sound was clear. There was occasional intermittence of the pulse, which was small and not much quickened. The respiration was free; the liver not enlarged; the abdomen not distended; the urine healthy; the bowels open.

The ammonio-citrate of iron with camphor mixture was given, and for his diet a chop with sherry wine. Cod-liver oil was afterwards prescribed in addition. This patient rapidly improved; the heart's action became quiet and regular, the dyspnœa was relieved, and the anæmia lessened. He left the hospital on June 4th, greatly benefited by the rest from active exertion, and by the course of treatment that had been adopted.

In this instance the rheumatism had been accompanied with inflammatory disease of the heart, and the mitral valve had become thickened and damaged; but on the patient's admission we did not find those symptoms of secondary congestion of the lungs, right side of the heart, liver, and kidneys, so often present when there is obstruction of the mitral orifice, and also at the close of valvular disease generally, as in the last case. When these secondary congestions have taken place no remedies afford greater benefit than stimulants to the abdominal glands, such as mild mercurials with squill and digitalis, or diuretic salines. But in this instance steel was indicated rather than these medicines, and was of great service in lessening the irritability and increasing the muscular power of the heart. To give steel in the *former* class of cases is to aggravate the distress; to give mercurials and salines in the *latter* is to favour dilatation, to promote anæmia, and to hasten the terrible train of symptoms consequent on an embarrassed and failing circulation.

DESCRIPTION OF PLATE.

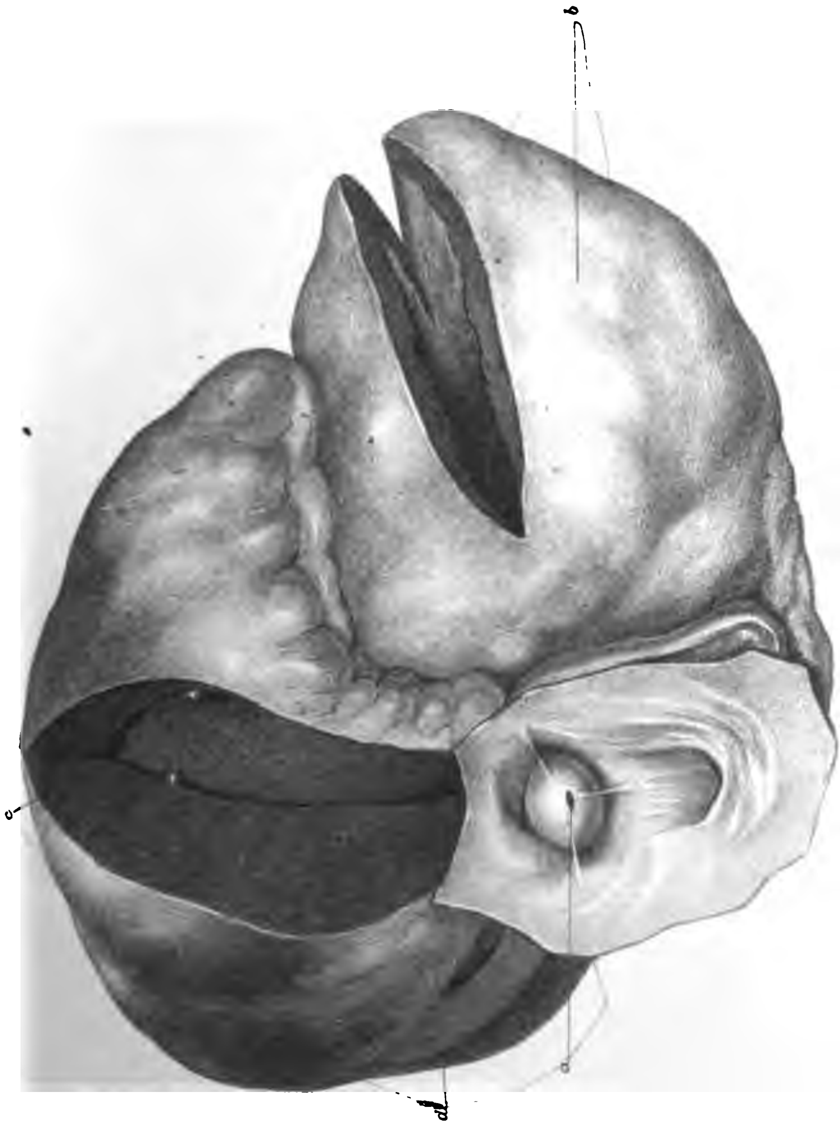
Dr. Habershon's case of Disease of the Pulmonary Valves.

a. Adherent pulmonary valves, seen from the arterial side.

b. Right auricle.

c. Right ventricle.

d. Left ventricle.



TWELVE CASES OF POISONING,

APPARENTLY FROM THE

USE OF COPPER FOR CULINARY PURPOSES.

DEATH FROM POISON OR DISEASE AFTER NINETEEN DAYS' ILLNESS.

By ALFRED S. TAYLOR, M.D., F.R.S.

A case, in which it was suspected that the death of an aged man had been caused by poison, was referred to me for examination by Mr. Secretary Walpole, on July 27th, 1866. The deceased, Mark Sims, was ninety years of age, and, although he had been ill some months before the attack which proved fatal, he had sufficiently recovered to eat his food with appetite, and, with the exception of the ordinary effects of old age, he appeared to be well. On Saturday, July 7th, he was seen by a medical gentleman, Mr. Covey, of Alresford, who found him suffering from sickness, purging, and severe pain in the abdomen. He died on July 25th, nearly three weeks after the attack, which came on apparently as a result of his having eaten some meat sent to him by a lady in whose service his granddaughter was living. An inquest was held by Mr. J. H. Todd, Coroner for Hants, who has kindly forwarded to me the depositions taken before him, in order that I may give a complete history of the case. Although the special object of inquiry was the cause of death of this aged man, the question involved indirectly a charge of poisoning against the cook in the family of Mr. Corrie, of Itchen Abbas. It seems that no fewer than twelve persons who had partaken of the food had suffered from similar symptoms, resembling those of irritant poisoning, although all ultimately recovered. It also included the medico-legal questions—whether the symptoms and appearances were consistent with natural disease, or the effects of an irritant poison; whether the food itself was wholesome, or whether any irritant substance had been intentionally or accidentally mixed with the food during its preparation.

The cases, taken as a whole, had a feature common to irritant poisoning, whether accidental or criminal, namely, that similar symptoms, consistent with the effects of an irritant, had shown themselves about the same time among many persons, including the deceased, who had also partaken of the food. This character, however, is common to all those cases in which the food itself, whether from decay or other causes, acts like an irritant.

The previous history of the deceased may be gathered from the account given by a neighbour, Ann Hoare, who looked after him. Deceased was in the habit of receiving a dinner daily from Mr. Corrie's house; he ate his food with appetite, and he was never ill after it until Friday, July 6th. About 6 o'clock on that morning this woman went in to light the fire; deceased had dressed himself, and was then coming down stairs; but complained of feeling poorly. She had seen him several times the day before, when he seemed to be as well as usual. She gave him some tea; he then began to be purged, and the purging increased during the day and was violent about 10 o'clock p.m., when he was put to bed. He was in great pain while the purging lasted, which was upwards of a week. He first *began to vomit* on Saturday morning, July 7th, but was *not sick long*. The vomited matter was thrown away. He seemed easier after the sickness; and after the purging had ceased he suffered from pains in his inside until his death. He constantly complained of a pain in his right side, and was insensible at times three or four days before he died. About 5 in the afternoon of Wednesday, July 25th, witness gave him a little tea, and he was immediately very sick, and taken with shivering. He vomited twice a sort of phlegm (mucus). He was sensible, and kept asking for more clothes to be put upon him. He was then getting very cold. He died that night at a quarter to 8 o'clock, having kept his bed from the time when he was first taken ill.

Mr. Covey, a medical man, stated that he had known the deceased for many years, but had not attended him for a long time until lately. He was a hearty man for his age. Saw the deceased in bed on Saturday, July 7th. He was suffering from severe pain in the bowels, with sickness and diarrhœa. Laudanum, camphor, Dover's powder and chlorodyne, were prescribed for him. He had every description of nourishment from Mr. Corrie's. The diarrhœa and vomiting ceased after some days, but the pain in his bowels continued until his death. An inspection of the body was made, and the following appearances were observed:—The general aspect was healthy, but the body was emaciated. The heart was healthy; the lungs were both adherent to the ribs. There was disease of the right lung, which was somewhat solidified and shrunk. The liver was healthy and the gall-bladder full. The stomach contained about half a pint of grumous fluid; the mucous coat was generally pale and healthy. There was a small patch of a dark congested appearance near the pylorus, but there were no ulcers. The duodenum presented a red inflammatory appearance throughout its whole length, waning off towards the jejunum, which was healthy. There was one large ulcerated patch in the middle of the duodenum. The ileum, in its upper part, was healthy, but at about fourteen inches from the ileo-cæcal valve the mucous lining was much inflamed, and was thickly marked by ulcers of varying sizes, some with ragged edges, and extending quite through the mucous coats. There was a space of about six inches in this region which was marked by effused blood. The colon contained a small quantity of fæcal matter, with two or three ulcerated patches similar to those in the ileum. All these morbid appearances were of recent date, and they would fully account for the pain suffered by deceased, and, in the witness's opinion, they were the cause of his death. The brain was not examined, nor the bladder. The medical witness attributed death entirely to inflammation and ulceration of the bowels, which he should decidedly say did not arise from natural causes, but must have been occasioned by some deleterious matter taken into the system. From the post-mortem appearances alone it was not possible to specify the particular kind of irritant matter which had been taken.

Mr. Buckell, a surgeon, of Winchester, had known the deceased many years and had attended him professionally several times, but for nothing serious until last January, when he suffered from severe inflammation of the lungs from natural causes. He appeared to have completely recovered from this attack before his last illness came on. He first saw deceased on this occasion on Sunday, July 15th; he was then in bed and appeared to be very ill, suffering from severe pain in his bowels, with diarrhœa and great exhaustion. He attended him occasionally until his death. The pain in his bowels continued until the last. He was present at the inspection of the body, and the appearances were as described. In witness's opinion those appearances were not sufficient in themselves to necessitate the supposition that they were occasioned by any poisonous matter taken by deceased, although at the same time they would be quite consistent with such a cause. He had no doubt that the inflammation and ulceration of the bowels were the cause of death, but it would require a chemical analysis to detect the presence of poison as the cause of the mischief.

It may be here observed that the stomach, liver, and diseased portions of the intestines, were removed from the body, placed in a jar, sealed and labelled, and on July 31st were delivered for analysis at the Chemical laboratory, Guy's Hospital, by an officer deputed by the coroner. The only facts with which I was made acquainted at this time were those contained in the evidence of the two medical gentlemen who examined the body. The parts received were—1, the stomach, with its cavity laid open, and the duodenum; 2, portions of the intestines, small and large; 3, the liver, with the gall-bladder fully distended with bile. When the examination was made on the following day (August 1st), putrefaction had advanced to a great extent. The stomach was cut open from end to end; there were no contents, and there was no appearance of food or of any foreign substances on the mucous surface. The lining membrane at the centre of the stomach and towards the intestinal opening was reddened and discoloured, probably as the result of cadaveric changes. A minute examination showed the absence of ulceration, perforation, or other disease.

No mineral or vegetable matter could be seen on any part of the organ, and there was no blood effused. The portions of the large and small intestines which were sent were much putrefied; the coats were of a dark red colour, running into the green and black colour of putrefaction. The lining membrane was softened, and in parts had been removed by ulceration. The coats were generally thinner than natural. The liver was dark coloured and soft; it was in a decomposed state. The gall-bladder contained an ounce of dark-coloured bile, which was removed for a separate analysis.

Without entering into the details of the various analyses connected with the viscera, it may be remarked that the liver, bile, and coats of the intestines, were tested by Reinsch's and other processes for arsenic, antimony, mercury, and their compounds, in their free and absorbed state, and that the salts of copper, lead, and other metals in the free state, were specially sought for in the coats of the stomach and the ulcerated portions of the intestines with the mucus adhering to them; but no metallic poison was detected in any of these parts. The process of Reinsch was applied to the liver, intestines, and stomach, for the detection of absorbed arsenic, antimony, and mercury, under circumstances which could not have failed to indicate the existence of minute traces of these metals had they been present; but the results were essentially and conclusively negative. The processes for detecting antimony by magnesium, by pure tin and hydrochloric acid, and voltaically by means of zinc and platinum, gave equally negative results; and the process for detecting copper by polished iron, as well as voltaically by zinc and magnesium, with platinum, also showed a complete absence of any of the salts of the metal in a free state. Finally, the application of pure magnesium showed the absence of any metallic compound in solution in the acid decoctions of the viscera. Absorbed and deposited copper in the liver was not sought for, inasmuch as traces of this metal may be found in the liver, kidneys, and other organs, if specially examined, in persons dying from causes wholly irrespective of poisoning. The discovery of mere traces of this metal in the absorbed form, therefore, had they been found, would have thrown no light on the cause of death.

The aqueous decoction of the coats of the stomach and intestines contained no metallic substance dissolved. Chloride of sodium and traces of alkaline sulphates were the only salts present, and these are the natural constituents of the mucous fluids found in these parts.

The only conclusion to be drawn from the chemical results was, therefore, that no irritant poison was present in the body of deceased at the time of his death; that the cause of death was inflammation and ulceration of the bowels; and that, in the absence of any other facts calculated to throw a light upon the case, the inflammation and ulceration were most probably due to natural causes.

Such was the substance of my report to the coroner, but the fact that deceased had a day or two before his illness eaten some veal which, it was believed, had caused severe illness among many members of Mr. Corrie's family, and that somewhat similar symptoms had presented themselves in his case, was well calculated to cast a doubt upon the conclusion that the inflamed and ulcerated state of the bowels had arisen from natural causes. On the other hand, the chemical evidence could hardly be said to negative the fact of poisoning. The deceased had lived after taking the supposed poisonous food nineteen days; there had been much purging and severe vomiting. No portion of the poisoned food could have possibly remained in the body for this long period, and, according to now well-ascertained and recorded facts, such poisons as arsenic and antimony may be wholly eliminated from the body within two or three weeks from the time at which they were taken. If this has been observed when the dose has been large, the elimination would, *à fortiori*, be more rapid when, as in the present case, if poison were taken at all, the quantity must have been small. The chemical fact that no trace of a metallic irritant poison was found in the body of deceased nineteen days after the alleged poisoned food was eaten, is not therefore inconsistent with the theory that some metallic irritant might have been taken with the food on the 6th of July, and have caused the inflammation and ulceration of the bowels which proved fatal on the 25th of July.

It appears that on Wednesday, July 4th, the servants, to the number of six, had their dinner as usual in the servants' hall, at two o'clock, the dinner consisting of baked veal, gravy, potatoes, and cabbage. It had been dressed as usual by the cook, Martha Tully, who had had notice from her mistress a few days previously, to leave her service. At variable periods, but *many hours* after the meal, various persons who partook of the food suffered from a similar train of symptoms, but all recovered excepting the deceased.

A magisterial inquiry took place on Monday, July 30th, the cook having been charged with wilfully administering a certain deleterious and poisonous drug, with intent to injure, aggrieve, and annoy. The prisoner denied the charge, but, according to one of the witnesses, she said, after those who had eaten the food had been taken ill, "If there is anything wrong about the veal, it must be from the copper stewpan the gravy was made in." The following evidence was given before the magistrates by those who ate the food.

Marian Dove, a kitchen-maid, said that the dinner consisted of hot baked veal, which had been partially cooked on Tuesday the 3rd but finished on Wednesday the 4th July, of cold bacon, vegetables, and some pudding. The prisoner cooked the meat, and witness dressed the vegetables. The food served as the early parlour dinner for the family; it was brought from the dining-room, and then served for the servants' dinner. There were, besides the prisoner, six servants, including the witness, who dined at the table in the hall. The prisoner, as usual, carved.

Elizabeth Hammond, a housemaid, was helped to the brown part (the outside) of the baked veal, as the meat was underdone, but she had *no gravy* with the meat. Witness herself had both meat and gravy; she ate all the gravy, but not all the meat. Dove then gave the following account of her symptoms. "About one o'clock next morning, Thursday, 5th of July (*i. e.* about *twelve hours* after the meal), I felt very unwell; my feet were very cold, and I shivered; my head was very bad. I did not feel sick then, but about four o'clock (*i. e.* three hours after the first feeling of illness) I felt very sick. I got up about six o'clock, leaving the prisoner, who slept in the same room, in bed. I attempted to do my work, but was very sick, and had diarrhœa." It further appeared from the evidence of this witness, that she took neither medicine nor food before she was seen by Mr. Covey, the medical attendant of the family, between eight and nine o'clock on the same morning. The prisoner came into the kitchen about 7 o'clock; she did not complain of sickness or diarrhœa, but subsequently complained of her head and in the latter part of the day, of feeling unwell. Dove continued to be more or less ill up to Sunday, the 8th, during which time she was attended by Mr. Covey.

The prisoner partook of the same dinner as the witness and the other servants, *i. e.* of the baked veal. The witness *saw* her eating some.

Susan Britten, a nursemaid, deposed that the prisoner gave her on the 4th of July two vegetable dishes—the one containing baked veal, the other vegetables. She took them upstairs for the nursery dinner. She and the nurse did not often have gravy with their meat, but on that day they had gravy. The witness observed nothing unusual about the baked veal; she and the nurse had their dinner about two o'clock, but they did not eat the whole of the veal; some was placed aside in a cupboard. They consumed the whole of the gravy. During the following night (two o'clock, a.m., on Thursday morning, the 5th of July), about *twelve hours* after the meal, this witness felt ill, very sick, and cold in the legs, feet and hands. She got up about seven o'clock, and was then sick (vomited) and had diarrhœa; these symptoms continuing until she was seen by Mr. Covey, about eight a.m. She had taken no food or medicine since the previous night. The sickness continued until Friday, the 6th of July, and the purging for a longer time. She was under medical treatment until Tuesday, the 10th of July.

Before taking the nursery dinner upstairs, witness had received from her mistress the child's dinner, which consisted of baked veal and gravy. The veal was cut up, mixed with potatoes and gravy, and given to the child—a girl about two years old. She saw her mistress cut up the veal. She believes the child ate the whole of it; after her dinner, at a quarter past one, the child went to sleep, and when she awoke she was ill; and at 12 o'clock the next day (Friday, the 6th of July) she was very ill. It appeared also from the evidence of this witness that when her mistress gave to her the child's dinner she also gave her in a separate dish, some baked veal, with potatoes, cabbage, and gravy, to take to her grandfather, the deceased Mark Sims, to whom witness was in the habit of taking a dinner daily. She took it to her grandfather's, and placed it in a cupboard of the house.

F. Amos, the nurse, dined with the preceding witness in the nursery on the 4th of July. She noticed the gravy because they seldom had it with meat. They ate all the gravy but not all the veal; some was put aside in the cupboard. About 12 o'clock on Wednesday night witness felt unwell, and about 1 o'clock (on Thursday morning) was very sick, and had diarrhœa. Her hands became dead (numbed) and her nails white, and she had a burning sensation in her throat. She was seen by the medical man about 8 o'clock on Thursday morning, and was still under his care. This witness further stated that on the 4th of July she gave the girl the dinner as it was received from her mistress. The veal and potatoes were cut up and given to her with a spoon. She was taken sick on the 5th (the day following); she seemed unwell and drowsy about 12 o'clock at noon, and was put to bed, and there continued until about 4 p.m., when she became very sick. The girl refused to take her breakfast on the following morning. The witness herself was affected similarly to other persons in the house, and was ill for about a week.

J. Shaw, butler, said that he took up the early parlour dinner on Wednesday, the 4th of July. It was hot baked veal with cold bacon, potatoes and cabbage. He observed that there was a great deal of gravy, and that it was dark in colour. He took the veal from the dining-room after dinner. He tasted the gravy in the passage from the dining-room to the kitchen; he took two dessert-spoonfuls. He afterwards dined with the other servants in the hall. The prisoner carved; she cut round the sides of the veal because the inside was underdone. He believed that he had a little gravy at his dinner. This witness was taken ill between 3 and 4 o'clock in the afternoon of Thursday, the 5th (*i.e.* from twenty-five to twenty-six hours after dinner), had great pain in his head, and shivering in his body, was sick in the evening, and suffered from diarrhœa about 7 o'clock. The sickness and purging continued two or three days, and on the first and second nights he was very ill.

H. Brooks, laundry-maid, deposed that she dined in the servants' hall on the 4th, and ate veal, bacon, cabbage, and potatoes; she had *no gravy;* she *was not ill at all.* With some few exceptions, all the family were ill. The exceptions were Mr. and Mrs. Corrie (her master and mistress), Miss Harriet Corrie, Elizabeth Hammond, and herself.

C. Selway, the page, dined with the other servants on the 4th, and ate veal, bacon, and potatoes, but had *no gravy.* There was a good deal of gravy, and it was dark coloured. Witness did not feel ill until Friday morning, the 6th (forty-two hours after the meal). He had headache, was cold, and had diarrhœa, but *no sickness.* He took his meals as usual from the time he dined on Thursday until Friday when he was taken ill. This witness also stated that on Thursday, the 5th, the day after the dinner, some remark was made in the servants' hall about the gravy. The prisoner then said, "She had boiled it too long in a copper saucepan, and forgot it."

Elizabeth Hammond, housemaid, dined with the servants on the 4th of July; had baked veal, bacon, cabbage, and potatoes. She ate the vegetables and veal, but was not sure whether she had any gravy. (One witness, Dove, distinctly states that she had no gravy.) She ate the *outside* of the veal, and noticed that some of it was underdone. *She was not ill afterwards.* A conversation took place in the hall on Thursday, the day following the dinner in reference to their having been taken ill one after another. The prisoner then said, "If there is anything wrong about the veal, it must be from the copper stew-pan the gravy was made in."

Mr. Covey, the surgeon, saw several members of the family on Thursday, July 5th, about 8 o'clock in the morning. All had been violently sick, and apparently from the same cause. The nurse was restless, complained of a feeling of sickness, pain in the region of the stomach, and coldness. Two of the sons of Mr. Corrie presented much the same symptoms; they were relieved by vomiting and purging. The nursemaid, Susan Britten, was similarly affected. Some of the vomited matter was tested by a chemist (Huggins) in witness's presence for arsenic, antimony, oxalic acid, and other poisons, but without result. At 2 o'clock on the same day he saw the nurse; she was almost pulseless, and could retain nothing on her stomach. The two sons were also very ill, the sickness having returned. They were all worse than when the witness saw them in the morning. Witness felt *no fear for the consequences* at that time, as they were all benefited by the treatment. When seen again, at 8 o'clock, the nurse and the other patients were, however, generally worse. Everything they took was rejected. He saw them all again the next morning at 6 o'clock (July 6th). On Sunday, the 8th, the nurse was much worse; he considered her case very serious for nine or ten days. The little girl was also very ill.

On Friday, the 6th of July, witness was called to see some children in the village of the name of Hoare, not connected with Mr. Corrie's family; they were vomiting and were purged excessively; he saw them after that on different days. The symptoms continued, but were not so severe as among the members of Mr. Corrie's family. He further said, in answer to questions put by the magistrates, that there must have been a great deal of verdigris in a copper saucepan to have caused so many persons to be ill. It must have been in a horribly filthy condition. He saw the prisoner at about 10 or 11 o'clock in the morning of July 6th. There was not much the matter with her. She complained of headache and severe thirst, with derangement of the stomach. She said she had not been able to take much nourishment. She did not complain of sickness or diarrhœa. She had no symptoms similar to those observed in the members of Mr. Corrie's family.

The witness Ann Hoare, who attended upon Mark Sims, gave the following account of the circumstances under which deceased had partaken of the food supplied from Mr. Corrie's table. The veal, as already stated, had been left in a cupboard at deceased's cottage by his grand-daughter, one of Mr. Corrie's servants. At the request of deceased, the woman Hoare had separated all the gravy as well as the vegetables from the plate in which the dinner had been sent, and placed the "dry" veal on another plate. It did not appear to have been disturbed since it had got cold. The vegetables and *gravy* which were left in the plate she gave to her two children about 11 o'clock on Thursday morning, and on that night they were both taken ill with purging and vomiting; they were seen by Mr. Corrie on the following day. The *dry* *veal* was placed on a clean plate, and the next day Mark Sims informed witness that he had eaten it for his supper on Thursday night (July 5th). It was thus clearly proved that some of the veal, deprived as far as possible of the gravy, had been eaten by the deceased; also that he first complained of feeling poorly about nine or ten hours afterwards, but he was not affected with any serious symptoms until after his breakfast on Friday morning.

The magisterial inquiry was brought to a close before the analysis in the case of Mark Sims had been completed. The case was dismissed, as the magistrates did not consider that there was sufficient proof to connect the prisoner with the act of wilful poisoning. It was stated by a solicitor who appeared to support the charge against her that some poison had been detected in the vomited matter as well as in the veal, and it has been since announced that "unmistakable traces of antimony were found in both by Dr. Letheby." This was not, however, proved before the magistrates, and they declined to adjourn the case for the evidence, considering that, even admitting the averment to be true, it failed to connect the prisoner with any criminal act of poisoning. It was argued on the evidence already given that the prisoner had deliberately put some poison (antimony) into the food served for the dinner, and that the accidents as described by the witnesses, could not possibly have arisen from the use of a dirty copper stewpan.

At the adjourned inquest in the case of Mark Sims, on the 16th August, the cook, Martha Tully, was summoned as a witness and examined by the coroner. It appeared that she had been in the service of Mr. Corrie only four months, and on the 2nd of July, just before this occurrence, she had received notice to quit. (This was supposed to supply a motive for the criminal act of administering poison in the food served for a large family.) She stated that a leg of veal was brought as usual by the butcher on Tuesday, July 3rd. There was nothing particular in its appearance, except that the knuckle was black, as if bruised. She pointed this out to her mistress, who thought nothing of it. The butcher's boy told her that the veal was killed on the Friday night preceding. By her mistress's order, she partly cooked the veal that night, cut the knuckle off for gravy, and put it into a copper stew-pan to boil it down. She directed the kitchen-maid to pour off the gravy, but this was not done, and the gravy remained in the copper stew-pan all night. She poured it off the next morning and drank about a table-spoonful, and observed that it had no particular taste or smell about it. The copper saucepan had a tin lining, which had been a good deal worn, but it was quite clean when the meat was put into it; the water was afterwards added by the kitchen-maid. There was no colouring in the gravy nor anything except salt.. After the gravy had been poured off on the following morning it was placed in a basin in the larder. On the next day (Wednesday, July 4th) she boiled up part of it for gravy to the fillet of veal, which was partly baked in the oven by the side of the fireplace on Tuesday evening, and again on the following day, when it was served up hot for the family dinner at 1 o'clock, with the gravy prepared from the knuckle. The veal was baked in an iron dish, lined with enamel. The meat was partly cooked on the Tuesday by order of Mrs. Corrie, for fear it should not keep until the next day. There was stuffing in the veal, made in the usual way. The suet used in the stuffing had been in the larder three or four days, but did not appear to be the worse for it. It was a new larder, and had just been painted. The witness then stated upon her oath that up to this time she had no knowledge or suspicion of anything wrong in the veal or gravy. After the family had dined, the veal was brought into the kitchen; she cut off the dinner for the two nurses, and helped it, as usual, with gravy as it came out of the dining-room. The remainder served for the servants' dinner in the hall about 2 o'clock; she then observed that the veal was underdone, and she mentioned it at the time. There were six who dined at the table, including herself, and they all had the same veal with some cold bacon, cooked the day before, with vegetables (greens and potatoes) boiled in the saucepan which had been used for this purpose for the dining-room. She herself ate a portion of the veal, with bacon and vegetables, as well as gravy. At night she fried up the remainder of the veal and bacon for the servants' supper, and four of them ate it, of whom she herself was one. The other two had boiled mutton. The two nurses had cold underdone veal for their supper. Next morning the kitchen-maid complained of illness, the symptoms being such as had been described. At 1 o'clock in the afternoon of that day (July 5th) witness was herself taken ill with headache and sickness, and cramps in the stomach. It was soon after this that she first became aware that several members of the family had been taken ill. She continued ill for three days. She was not aware of anything that could have made herself or the others ill, unless it was the veal. About nine days afterwards she was given into custody on a charge of having administered poison to Mr. Corrie's family. She was discharged by the magistrates on the 30th of July. Upon the oath she had taken, she was prepared to state that she was innocent of the charge that had been made against her.

On being further questioned she said that the gravy which was sent up with the veal was very brown (dark coloured), from the meat having been twice baked. That part of the gravy which was not used for dinner was put away in the larder. Having left the house, she did not know what became of it. She had no knowledge or suspicion of any person having put anything into the gravy unknown to her, but the larder was open to any one.

The report of the results of my analysis in the case of Sims was then read to the jury, and the coroner, on summing up the case, said—

"According to one theory, the cause of death might have arisen from the veal the deceased ate from Mr. Corrie's house on the 5th of July, because he was in apparently good health up to that time. It was stated in the medical evidence of Mr. Covey that Sims died of inflammation, and most extensive ulceration of the large and small intestines. It was suggested that the veal that was given to the deceased contained irritant poison, and the poison was probably copper. It was also suggested that the poison was intentionally put into the veal by Martha Tully, the cook. It was apparent from the evidence that Brooks and Hammond, two of the servants, ate a portion of this veal for dinner, and it might be that they ate as much or more than the deceased did, and yet they suffered no symptoms of illness after eating it. Hence the question arose, could there have been in the veal sufficient copper or other irritant poison to cause severe inflammation and extensive ulceration of the small and large intestines of the deceased? Now, it was contrary to all experience for any one to eat food in which he or she had knowingly placed poison, and the cook, Martha Tully, ate the veal as usual at her dinner. Tully's pretence of illness subsequently, when she knew that so many of her fellow-servants had been taken ill, might admit of explanation without rendering it necessary to suppose that she deliberately poisoned the veal. But the amount of morbid changes in the form of ulceration of the bowels was much greater than was known to have been produced by such very potent irritant poisons as arsenic, antimony, or mercury. Copper poison (according to Dr. Taylor's report) had never in large doses, as far as he knew, produced such extensive ulceration of the bowels as was found, and in cases where it was known to have destroyed life the stomach had never escaped its action. Now, while he (the coroner) used the theory of Dr. Taylor, the opinion of Dr. Letheby, as offered before the police magistrates, could not be entirely ignored, 'that in the vomit, antimony was present.' The jury, while weighing theories, must only do so in relation to the facts, and, there being no direct evidence either way, he (the coroner) should advise a verdict which would leave further inquiry open, if necessary."

After some deliberation the jury returned the following verdict:—"That the deceased, Mark Sims, died from inflammation and ulceration of the bowels; but whether such inflammation and ulceration was occasioned by natural causes or otherwise, no conclusive evidence appears."

The woman Tully then applied to the coroner for a "certificate of exoneration," but the learned gentleman told her that no one was accused before him. She had given her evidence as a witness, and left the Court with her character as untainted as she entered it.

Remarks.—It is impossible to separate the consideration of the cause of *death* in the case of Mark Sims from the cause of the *illness* among the members of Mr. Corrie's family. There is no doubt that Sims partook of a portion of the same food, namely, the veal, but the singular fact clearly brought out by the evidence, is that this was the very article which could not have been poisoned at the time it was served for dinner. In general, a slight observation will enable a medical witness to determine which of several articles of food served at a meal contained poison. Among a number of persons thus eating in common, it will be found that some have taken one article, others another, and thus it may be easy to connect the symptoms with one description of food to the exclusion of others. This is a natural process of reasoning, but it is often neglected, because the mind is influenced by a foregone conclusion. Several cases illustrative of this method of elimination have fallen under my notice. In October, 1839, a gentleman residing in the City of London had a dinner party, at which many of his friends were assembled, among others a medical man. In about half an hour after dinner some of the guests were seized with faintness, nausea, vomiting, and other symptoms indicative of irritant poisoning. The medical man who was present noticed that only those who had taken port wine suffered; the guests who had not taken port had not suffered from any symptoms. The bottle was brought to me for examination, and it was found that the wine contained arsenic dissolved in it in a large quantity. These persons recovered. In another case, the child of a gamekeeper died from the effects of arsenic. It was proved that the man had sat down to dinner as usual with four of his children, the eldest among them being a girl of about seven years of age, a sharp intelligent child. The man was charged with the murder of one of his children by administering arsenic, but the difficulty was to prove the administration, which, if it took place at all, must have taken place at this dinner. According to the evidence of the little girl they dined together, and each had the same food, namely, beef, with gravy, pudding, and vegetables; soon after dinner the deceased child alone suffered from vomiting and purging, and died within twenty-four hours. The body was buried, and exhumed after a month's interment, when the cause of death was apparent in the discovery of a quantity of arsenic in the stomach mixed with portions of food (husks of raisins, &c.), such as the other children had eaten at the dinner as well as the prisoner himself, but without suffering from any symptoms. It was elicited from the girl that the only difference made by her father in helping them, was that the deceased did not have "salt" from the salt-cellar on the table like the others, but her father got up and fetched it from an adjoining room, bringing a pinch of it between his finger and thumb, and placing it on deceased's plate. On a shelf in this inner room was a bottle of white arsenic, and there was no doubt, from all the circumstances, that he had placed white arsenic, which the girl had mistaken for salt, on this child's plate. This at once explained why the deceased alone suffered from the effects of poisoning.

In one case in which I was consulted a pile of plates had been placed as usual before the person who carved the joint for a large party of persons. All were helped to the meat and gravy. One only suffered from symptoms of poisoning with arsenic, and some white substance which was scraped off the plate was brought to me for examination. It proved to be white arsenic in powder. On this occasion no article of food was actually poisoned, but the arsenic, either by accident or design, must have been placed in the plate (the fourth of the series from the top), and when the meat and the gravy were put into this plate they became mixed with arsenic.

Applying these observations to the cases now under consideration, it may be remarked that, among the different articles of food served at this dinner, there is no reason to suspect that any noxious substance was mixed with the vegetables (potatoes and cabbage) or bacon. The effects must be attributed either to the veal or to the gravy, and of these two the gravy appears to have contained the noxious matter. This, indeed, is conclusively established by the fact that *those persons only* suffered who partook of the *gravy*, while those who had the *veal without the gravy* did *not* suffer. Dove, Britten, Amos, Shaw, and the two children of the woman Hoare (these children taking the *gravy without any veal*), all suffered, at about the same long interval after the meal, and from a similar train of symptoms, and all recovered. On the other hand, Brooks and Hammond, who ate the *veal* and took *no gravy*, did *not suffer* from *any symptoms* of illness. It appears, further, that Hammond had the *outside* of the baked veal, in which poison would be more likely to exist. It is clear that, as the veal was baked, if poisoned at all, it could have received poison only from the outside, and that those who ate this part would have suffered from the effects more than others who were helped to the inner portion.

Selway, the page, states that he ate the veal, but no gravy. He gives a confused account of his illness. He was not ill at all for forty-two hours after the dinner, and then he had no sickness or vomiting, but merely diarrhœa—whereas the primary symptom in all the other cases was a feeling of sickness, followed by vomiting, severe colicky pain in the abdomen, shivering, and purging. He took his meals, and went about his work as usual, from Wednesday, the 4th, at 2 o'clock, until Friday morning. There is nothing to show that the diarrhœa from which he then suffered was caused by the veal taken at dinner two days before. His illness appears to have differed from that of the others, and to have been of short duration. These facts seem to me to be quite inconsistent with the theory that the *veal* was poisoned, and that it caused symptoms of poisoning among those who partook of it; they are, however, consistent with the theory that the *gravy contained some poisonous* or *noxious substance,* and that this was really the cause of the symptoms.

The question now arises, how did the gravy acquire the noxious impregnation? The circumstances proved appear to favour the view that it contained the poison of copper in the form of chloride or subchloride of that metal. According to the evidence, the gravy was made as usual, with salt and water, in an old copper stew-pan. Tully, the cook, stated that this was clean when she used it for the purpose, but it was an old copper pan, lined with tin, from which the tin had been much worn off. The gravy was allowed to cool in this vessel, and to remain in it during the night. The fatty matter from the meat, as well as the salt, would thus be placed under favorable conditions to act chemically upon the exposed surface of copper.

Here, then, were the usual conditions required for impregnating the gravy of the food with copper. It is to be regretted that the residue of this gravy, of which a quantity was left in the house by the cook after the illness of her fellow-servants, was not submitted to analysis. The result would have at once proved or disproved the existence of copper, and would have confirmed or refuted what must now be regarded as a theory to account for the sudden illness of these persons.

It was noticed by several witnesses, as well as by the cook, that the gravy was dark in colour. The cook ascribed this to the meat having been twice baked with it. This may be the explanation; at the same time, the presence of a salt of copper (subchloride) tends to darken the colour of most organic liquids.

The butler, John Shaw, stated that he tasted the gravy; he took two dessert-spoonfuls before he had his dinner in the hall; but he made no remark about its having any peculiar taste, such as copper commonly imparts to food. It is possible that a small quantity of a copper salt, such as verdigris, might not be perceptible to the taste, and under the circumstances the taste might have been concealed by the strong flavour of the gravy itself. Notwithstanding the peculiar taste which copper imparts to organic substances, it is certain from cases, some of which will be mentioned hereafter, that food cooked in copper vessels has given rise to serious accidents, and that such food has not had a taste to give any warning that it contained a salt of copper. But the non-observance of any particular taste in the gravy by Shaw, Dove, Britten, Amos, and the two children of Ann Hoare, all of whom took the gravy, is not consistent with the view that a large quantity of a copper salt could have been present in it, and, therefore, with the theory that a copper salt had been intentionally and wilfully put into this article of food. It is not probable that a person intending injury to others would put only a minute quantity of copper poison into an article of food prepared for the dinner of many persons. The presence of a quantity so small as not to be at once perceptible to the taste may be taken to indicate accidental rather than criminal poisoning by copper.

That the poison in the gravy was copper is rendered highly probable by various considerations. The symptoms suffered by the persons who partook of it closely resemble those caused by the salts of copper when taken accidentally in articles of food, *i. e.* by the various kinds of verdigris. Thus, they came on *many hours* (about twelve hours) after the meal; they were indicated by a feeling of sickness, retching, heat in the throat or constriction, violent vomiting, severe (colicky) pains in the abdomen, with purging, headache, numbness, shivering, and a sense of coldness.

As on this occasion, persons thus affected recover in a few days, or so soon as the noxious matter has been discharged by vomiting and purging. Death has rarely been a consequence of this kind of culinary poisoning, and it is only likely to occur when a very large dose of copper poison, or a succession of small doses over a long period, has been taken. In these cases there was not a single death among those who partook of the poisoned gravy, although three of them were children who suffered severely from the effects. The symptoms observed in these patients, taking them as a whole, differ from those caused by arsenic, antimony, or mercury, in their mode of commencement, progress, and termination. When these poisons have been taken the symptoms appear *much sooner*, cause greater prostration of strength, and generally cause death at an early period when the vomiting and purging are severe.

Assuming, therefore, that the noxious substance was a salt of copper, contained, either dissolved or diffused, in the gravy served at the dinner, all the facts appear to admit of a reasonable explanation. The presence of copper, it is true, could have been easily demonstrated by an analysis of the matter vomited or of the gravy itself. Whether any copper was sought for and found in the vomited matters by Mr. Huggins or Dr. Letheby does not appear.

Mr. Covey states that two portions of the vomited matter were examined for arsenic, antimony, oxalic acid, and other poisons, by Mr. Huggins, a chemist, but without result. Dr. Letheby, it is stated, found traces of antimony in the veal and also in a portion of the vomited matter, but no evidence was given to this effect. It may be observed, however, that the symptoms among the persons who suffered were not like those of poisoning with antimony. Thus, the illness of Dove did not commence until twelve hours after the dinner. The symptoms then began with cold feet, shivering, and headache. There was no sense of heat or constriction in the throat, and no sickness for fifteen hours, while the sickness, when it occurred, was accompanied with purging.

Nausea, retching, and vomiting, are early symptoms in poisoning with antimony (tartar emetic), generally occurring within a quarter of an hour after the substance has been taken. Shaw, the butler, who took the gravy separately, had no symptoms for twenty-five hours; they then commenced with shivering, headache, &c., and not with retching and vomiting, such as antimony would produce. To ascribe the symptoms in these cases to the effects of tartar emetic, the only poison of antimony which could have been put into the gravy, would be wholly inconsistent with all that is known respecting the action of this substance on the body. Among metallic irritants, tartar emetic is remarkable for its rapid action on the stomach, and for this reason it is employed as an emetic in cases of poisoning. Shaw, the butler, took two dessert-spoonfuls of the gravy before he sat down to dinner. Had there been antimony (tartar emetic) in this gravy, it is not at all probable that he would have been able to eat his dinner, or that the ordinary emetic action of this substance would have been delayed for twenty-five hours, the man having taken his usual meals in the interim. Again, Hoare's children had some of the gravy at 11 o'clock on the Thursday. It was not until nine hours afterwards that they were seized with purging and vomiting. It is therefore impossible to suppose that this gravy contained antimony, or the usual effects would have been produced in one or both children in a few minutes, especially as children are very susceptible of the action of antimony. It is true that there are exceptional cases in which the symptoms are protracted, but the whole of these twelve cases are alike in this respect, and are inconsistent with ordinary antimonial poisoning.

With respect to the alleged presence of antimony in the veal, it is clear, from the fact that several persons partook of the veal without suffering any inconvenience, that the mineral, if present, was not in sufficient quantity to produce any ill effects. This was observed in respect to those who ate the outside, on which the antimony, if present, would probably have existed in the largest quantity. The detection of antimony, therefore, in traces, even if it had been proved in evidence, would not have explained the facts.

It is necessary here to remark that *traces* of metallic poisons may be occasionally detected in articles of food, but it does not necessarily follow that they have been criminally introduced into such food. In March, 1857, the magistrates of Hull forwarded to me for chemical examination two legs of mutton. I found distinct evidence of the presence of antimony in the flesh taken from the centre of each leg, and reported that it was unfit for human food. A number of sheep had been landed from the Continent, and, as some were suffering from pneumonia, they had been dosed with antimony. The sheep from which the two legs were taken, were killed about twelve hours after the administration of the antimony, and cut up for sale. Had this mutton been served at table traces of antimony would have been detected in it, and a cook who had prepared it for dinner might have found himself involved in a charge of poisoning. Physiology and pathology are great aids to chemistry in these investigations. The symptoms should in all cases be consistent with the usual mode of action of the substance detected in traces, or evidence of this kind may mislead a medical witness. If there have been no symptoms in accordance with the nature of the substance found in food, the detection of traces is of no importance.

The cases here recorded bear a strong resemblance to some which have been reported by Orfila, ('Toxicologie,' 5ème éd., tome i, p. 784). In one group twenty-one persons complained of severe colicky pains, with febrile symptoms. In all of them the first symptoms consisted of severe headache, with great weakness in the legs and over the whole body, dull pains in the thighs, with cramps in the calves of the legs. Those who were first attacked suffered from severe pain in the stomach, accompanied with a feeling of sinking and uneasiness as well as trembling of the limbs. In some these symptoms did not show themselves until the day following the noxious meal which was supposed to have caused them. It appears that these persons had eaten fish which had been cooked in a copper vessel. The cook, after pouring off some of the water in which the fish had been boiled, had ignorantly added some vinegar to replace it, and had allowed this mixture to remain in the copper vessel some hours. This at once explained the cause of illness among so many persons. All recovered, but one to whom a dose of tartar emetic had been given remained ill for two months. In another set of cases one man and his wife ate for their dinner some meat which had been stewed in an earthen vessel with a copper lid. At 2 o'clock on the following morning the man was suddenly seized with severe colicky pains, followed by vomiting. The symptoms did not appear in the woman until some hours later. Another man and his wife who had eaten part of the same veal were also attacked on the following morning at 7 o'clock with pain in the stomach, nausea, and frequent vomiting. The woman suffered from severe pains in the head. The pulse was small and irregular, and there was great weakness. They all recovered. It appears that on this occasion the copper lid had been pressed down upon the stewed veal, the vessel being quite full, and in this state it was allowed to cool. The food had thus acquired a noxious impregnation. I have elsewhere reported a series of cases which occurred to Professor Barzellotti, of Pisa, on which occasion the professor himself narrowly escaped partaking of the poisonous food. At a monastery near Sienna the monks were seized some time after their dinner with violent symptoms of irritant poisoning. They suffered chiefly from severe pain in the abdomen, nausea, difficulty in passing urine, spasms of the muscles, and trembling of the limbs. Those who were attacked with vomiting and purging were speedily relieved; but others who had no evacuations suffered from giddiness, headache, intense thirst, and an unpleasant taste in the mouth. Remedies were employed, and all eventually recovered. The cause of the accident was soon discovered. The monks were in the habit of keeping their salt fish, when any was left from the meal, in the tinned copper vessel, in which it was again boiled for a second day's dinner; it was found that the copper vessel was old and the tin worn off. The boiled fish was examined, and it was found to be covered with a greenish-coloured jelly, and the sides of the vessel where the fish was in contact with it, were of a greenish colour, evidently from corrosion. The cause of the symptoms was no longer doubtful—subchloride of copper had been formed by the action of the salt in the fish upon the metal while the contents were in the act of cooling ('On Poisons,' p. 463). These incidents show that a number of persons may be poisoned with copper without any suspicion being excited by the colour or taste of the poisoned food.

Dr. Moore traced many cases of supposed dysentery in India to the Hindoo custom of cooking fish, rice, and butter, with salt, on plates of copper. A few hours after taking the food the patients complained of violent pains and cramps in the stomach and bowels, and there was constant vomiting of a yellowish-green-coloured bile. When this was not ejected from the stomach the dry retching was most distressing. There were griping pains in the abdomen, with tenesmus, and a burning sensation at the sphincter ani. The attack usually commenced with acute fever, heat of skin, headache, urgent thirst, loss of appetite, prostration of strength, furred and clammy tongue, with a rapid, small, and wiry pulse. In some of the more severe cases there was great depression of the vital powers, the pulse was rapid and weak, the skin cold, and the arms and legs benumbed. In most instances the symptoms subsided in eight or ten days; in others a long time elapsed before the mucous discharges and tenesmus ceased.

These cases, taken as a whole, appear to me to lend support to the theory that the symptoms of poisoning suffered by the members of Mr. Corrie's family were owing to copper. It would have been more satisfactory had an analysis been made of the gravy, as the noxious effects are clearly traceable to this; but in the absence of this chemical evidence there is apparently sufficient to connect the symptoms with the action of copper, while at the same time they are not reconcilable in any one instance with the ordinary effects of arsenic, antimony, or mercury.

A case in some respects similar, but in which chemical evidence equally failed to detect the poison, occurred to Dr. Christison, and is reported by him in his valuable work on Poisons ('Treatise on Poisons,' 4th edit., p. 87). After advising medical men to be particular on these occasions, in investigating everything connected with the cooking, serving, and eating of suspected articles of food, he says, "By doing so, not only will the chemical analysis be facilitated, but likewise facts in it will be accounted for which might otherwise prove embarrassing, and even lead to the drawing of false conclusions from the result of the analysis. In 1827 a family in Portobello were poisoned by the maid servant, and it was believed that, for the sake of a trick, she had, while carrying to the oven the beef subsequently used at dinner, maliciously mixed with it tartar emetic or some other poison. One half of the beef having been preserved, and two persons of the family having been very severely affected, Dr. Turner and I, to whom the case was remitted, made little doubt that we should discover the poison by chemical analysis; but we did not. Being subsequently employed by the sheriff to inquire into the particulars, I found that the poison had been mixed with the gravy, which had been consumed almost to the last drop—that the gravy had been poured over the beef—that the upper half of the beef had been eaten—and that the remainder, which we analysed, had been transferred upon a different plate from that on which it was served for dinner. These particulars accounted sufficiently for the poison not having been discovered."

It is unnecessary here to consider the question whether the poisoning was a criminal act or the result of accident. The person charged with administering poison to Mr. Corrie's family, with intent to annoy and aggrieve, is proved by the evidence to have eaten the same food in common with her fellow-servants. She took her dinner as usual in the servants' hall. She was seen to eat the veal, and she stated in her evidence on oath that she took some of the gravy, namely, a table-spoonful for tasting before it was served up, and she also had a portion with the veal at her dinner. She was ill some hours afterwards, but, according to Mr. Covey, she was not so severely affected as her fellow-servants. These circumstances are strongly presumptive of the poisoning having been the result of an accident. It is not in accordance with experience that a person who has deliberately mixed poison with food, should sit down and partake of that food in common with those whom he has the intention to injure. As under such circumstances there can be no certainty respecting the amount of poison which would fall to the poisoner's share, it is obvious that, under some pretence or other, he would, if guilty, avoid eating the food. It would be straining an hypothesis to an improbable extent to affirm that the poisoned food might be eaten with a view of arresting suspicion. There is so great a dread of poisons among ordinary criminals, and generally so exaggerated an estimate of their potency, that a man is not likely to incur the risk of destroying his own life by eating the poisoned substance which he has prepared for another. I have met with only one instance to the contrary, which is reported to have occurred among the Thugs in India. One of these robbers mixed datura seeds in powder with some ground meal, and made it into cakes. In order to lull the suspicions of those whom he intended to rob, he sat down with them, and ate a portion of the poisoned food. They were all rendered insensible, but the poisoner died and his intended victims recovered:

> "Nec lex justior ulla
> Quam necis artifices arte perire suâ."

I am not aware of any instance in this country in which a sane criminal has thus exposed his own life to serious risk for the sake of inducing others to eat poisoned food.

The bearing of these cases upon that of Mark Sims will now be apparent. What caused his symptoms, and what caused his death nineteen days after the first attack, and twelve days after the prominent symptoms, purging and vomiting, had ceased? It is clear from the evidence that the deceased had a portion of the veal served at the dinner. The gravy was removed from it, and it was put on a clean plate in a dry state. The children who had this gravy suffered from vomiting and purging nine hours after they had eaten it. It is also clear from the evidence that Mark Sims ate the veal for his supper on Thursday night, and that he suffered no symptom of illness until after his breakfast on Friday morning, and therefore not until eleven or twelve hours had elapsed from the time at which he had eaten it. Did the veal, then, cause the symptoms? The answer to this is given in the evidence of Brooks and Hammond. They, as well as several members of Mr. Corrie's family, ate it, and suffered no symptoms. If the deceased suffered as a result of taking part of the same food, it can only be inferred that he must have taken with it a small portion of the gravy which had proved sufficient to render the children ill; and as his portion of the veal had been for a much longer time in contact with the gravy, it may have become more impregnated with copper poison. This is the only reasonable explanation of the facts which suggests itself. If the veal was really poisoned, Brooks and Hammond, and others who took it without gravy, could not have escaped.

The nature and course of the symptoms are inconsistent with the supposition that deceased had taken a noxious dose of antimony (tartar emetic) in the veal. Referring to the evidence, it will be seen that a long interval, *i. e.* about eleven or twelve hours, elapsed before any symptoms like those of poisoning appeared. The first symptom was purging (not vomiting), accompanied with severe pain in the bowels. The deceased only began to vomit on the second day, and the vomiting did not last long. The purging ceased in a week, but the pain continued, a fact sufficiently accounted for by the ulcerated state of his bowels found after death.

Assuming that the bowels were already in an inflamed and ulcerated state, it is easy to understand that the very small quantity of copper poison which the deceased could possibly have taken in the veal might have been sufficient to irritate the lining membrane, and to cause the first symptoms, namely, purging and pain. Either this must be admitted, or, in conflict with all the evidence in the case, it must be assumed that food which did not contain noxious matter to affect in any way other persons who ate it, contained sufficient to cause inflammation and most extensive ulceration of the bowels in the case of the deceased. This assumption is more improbable than that above suggested to account for the illness of the deceased.

The post-mortem appearances are also very different from those which have ever been recorded as a result of chronic poisoning by copper. Inflammation of the stomach and bowels have been observed, but not ulceration, except in one case described by Orfila, in which ulceration of the rectum was found in the body of a child who had died from the effects of a large dose of verdigris ('Toxicologie,' i, 787); but in all cases of this kind the stomach also has presented marks of irritation and inflammation, and sometimes the œsophagus as well. In Mark Sims' case the post-mortem changes were confined to the bowels: the stomach was healthy. These were of an unusually extensive kind for any supposed case of irritant poisoning, and it is only remarkable that a man of ninety years of age could have survived so long under such a condition of the bowels. It is said that until about the time of this occurrence he had appeared in his usual health. He had not, however, been seen or examined by any medical man, and it is possible that there may have been latent disease which had not attracted the notice of the neighbours.

The conclusions which the facts of these cases appear to me to justify are—

1. That the members of Mr. Corrie's family suffered from the effects of copper poisoning, the poison being in the gravy, and not in the veal or other articles of food.
2. That the mode in which the gravy was prepared, namely, by stewing the meat with common salt in a badly tinned copper stew-pan, and allowing the liquid to remain in the vessel throughout the night, involves conditions necessary for impregnating a liquid with a salt of copper.
3. That the symptoms and appearances in the case of Mark Sims are consistent with pre-existing disease; that, taken as a whole, they are not consistent with poisoning by arsenic, antimony, mercury, copper, or any metallic irritant poison.
4. That although the deceased is proved not to have taken that portion of the food which was impregnated with copper, there may have been sufficient in the food to excite increased irritation, assuming that the bowels were already in an inflamed state. If this assumption be not admitted, then there is an absence of any adequate cause to account for the inflammation and extensive ulceration of the bowels, while the stomach remained unaffected.

5. That Mark Sims died from inflammation and ulceration of the bowels, most probably dependent on natural causes.

In the absence of any chemical evidence of the presence of copper in the body, the jury returned what is called an open verdict, but at the same time it is clear, from the scientific and other facts in the case, that no portion of the small quantity of copper (if any) taken by the deceased in his food would be likely to remain in the stomach and bowels for a period of nineteen days. The continued purging for a week would effectually carry it out of the intestines.

ON

PAPILLARY TUMOURS OF THE GUM.

By S. JAMES A. SALTER, M.B., F.R.S.

During the summer of 1862 a very remarkable and extremely rare form of disease came under the care of Sir William Fergusson at King's College Hospital. It consisted of an exuberant papillary growth, which was developed along the alveolar border on the right side of the lower jaw in a very old man.

In the month of March of the present year a patient was sent up to Guy's Hospital from Dorsetshire to the care of Mr. Cock. In the upper jaw of this patient a growth had developed itself having the same ultimate anatomical structure as the tumour in the previous case.

There were some important differences in the histories of these two cases; but they were differences rather of degree than of kind, as will be hereafter shown.

In Sir William Fergusson's case the early history and the ultimate result are unfortunately both imperfectly known; and the only record of it which I have been able to obtain consists in a brief and crudely reported *clinique* delivered by Sir William when he operated on the patient; this was published in the 'Lancet.' As, however, the pathological anatomy of the disease is so singular and exceptional, I have thought it well to quote this report *in extenso*, more especially as it is not long.

"Familiar as he, Mr. Fergusson, was with diseases of the jaw, and he had seen as many examples as most surgeons, here was an instance, Mr. Fergusson remarked, of disease he had never seen before or read of. About the middle of June the patient had called upon him with a letter from a friend. He noticed something wrong with his jaw, and, on looking into his mouth, he asked if he had a bit of potato in it. To his astonishment, he found it was a growth upon the jaw. Some surgeons would call it fibrous; but it was a form of disease which he had never met with previously in this or any other part of the body. It looked like vegetable matter or greatly elongated papillæ. He could not undertake to give it a name. It was something like malignant disease; and a question arose as to what ought to be done.

"The friend who wrote to him (Mr. Fergusson) had operated several times. It would, perhaps, have been better to have removed the whole jaw, but he did not like to submit so old a patient to such an operation, for his age was eighty years. He selected a milder method, that of cutting the disease out, instead of making a large wound in the cheek. He removed on this occasion (June 21st) probably the greater part of it, together with its base. It certainly was not unlike medullary disease. He thought he had succeeded in taking away the whole of it. If there should be any left, he expected to remove or destroy it by means of chloride of zinc. We shall see, Mr. Fergusson observed in conclusion, when the granulations spring up, the process the disease may take; and we must look upon the present case more as an instance of the curiosities of pathology than of surgery.

"It may be remarked that the disease was confined to the right side of the lower jaw, and looked like meat that had been macerated for a long time, and had become bleached of a pinkish-white colour.

"On the 12th of July it became necessary to repeat the operation; for although but three weeks had elapsed since the last occasion of removal, the tumour had grown very rapidly, and in general characters resembled its predecessor. It was cut away chiefly by means of curved forceps, and portions of it were scraped from the bone. With regard to the last, very little of it was left, and Mr. Fergusson mentioned that it was necessary to proceed with caution in such an old patient.

"After remaining about another fortnight in the hospital, the patient left for the country. Up to this time there has been no further recurrence. The cicatrix was, however, touched with chloride of zinc." ('Lancet,' Sept. 6th, 1862, p. 255.)

On applying at King's College Hospital some months afterwards, I was unable to obtain any clinical note, or, indeed, any record of this curious case. The gentleman, however, who had been Mr. Fergusson's dresser while the patient had been in the house, gave me this important information—that the disease of the jaw was unaccompanied by any enlargement of lymphatic glands in its neighbourhood; he also told me that the tumour did not appear to be connected with the bony substance of the jaw. The patient was eighty years of age, and he died shortly after leaving the hospital, apparently from old age and natural decay.

Through the kindness of Mr. Partridge, who secured a portion of the growth when removed, I had an opportunity of examining its structure. It was a curious white mass, consisting of coarse detached fibres, pointed and free at one extremity and attached at the other; in fact, it was a mass of papillæ, many of them nearly an inch long, and similar in shape to the "filiform" papillæ of the tongue; their surface was shreddy and broken; among these elongated processes were a few rounded eminences, like "fungiform" papillæ, and these had a smooth unbroken surface.

Fig. 1.

Portion of papillary tumour (nat. size) from Sir William Fergusson's patient.

In microscopical structure I found the mass to consist almost entirely of epithelium, principally squamous and flattened; but in other parts the cells were aggregated together in groups, reminding one of the "bird's-nest" arrangement of epithelial cancer. The long filiform papillæ were striated or fluted in their long direction, and readily broke up into smaller hair-like processes; the centres of these processes consisted of very compact cells, not altogether characteristic of epithelium, but the more superficial were distinctly epithelial. The cells comported themselves as epithelium under the action of chemical reagents.

I did not succeed in making out the deeper structures of the papillæ, neither a limitary membrane nor a vascular loop. The base of the mass which was removed consists of fibrous tissue and a small piece of bone.

The particulars of the second case of papillary tumour of the gum are as follows:

George Marsh, æt. 57, a butcher and small farmer, resided at Wool, near Wareham, in Dorsetshire. He was under the medical care of Mr. W. S. Granger.

In April, 1865, the first upper bicuspid tooth of the right side was observed to be loose, and was removed; it was a sound tooth; the gum around it was supposed to be healthy.

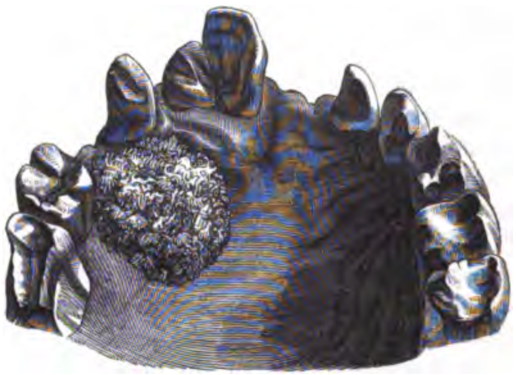
Early in the following August the patient first noticed a slight roughness and swelling on the side of the palate, extending from the inner alveolar border of the gum, where the bicuspid had been removed, towards the vault of the palate; the area thus affected was then about the size of a finger-nail. Smoking made the place smart, but beyond that it was not painful.

The patient now applied to his medical attendant, who burnt the surface with lunar caustic repeatedly. Still the growth advanced; it increased in area, and the roughness became greater by the development of distinct papillæ; these were only partially repressed by the caustic from time to time.

There had never been any discharge, or pain, or general swelling connected with the growth.

At the time of the patient's arrival at the hospital (March 20th, 1866) the growth was the size of a split chestnut, the attached base being rather smaller than the extreme circumference of the tumour; it rested on the side of the hard palate, extending from the edge of the alveolar border to near the summit of the vault. The position, form, and general aspect of the growth are displayed in the accompanying illustration. One very striking peculiarity cannot, however, be thus exhibited; it is the contrast of the colour of the tumour and that of the surrounding gum; the tumour was creamy white, and the surrounding gum the usual dark purple-red; the contrast was extreme, and the limit quite definite. The growth had much the appearance of a large, flattened, tegumentary wart, with unusually long papillæ.

FIG. 2.



Drawing from inverted plaster cast of upper jaw of George Marsh, showing papillary tumour *in situ*.

The illustration is drawn from a plaster-of-Paris cast of the upper jaw, which I took previous to the operation. The tumour looks a little smaller than it really was, from the pressure of the wax in taking the model.

On the 23rd of March Mr. Cock extirpated the growth. It was firmly attached to the periosteum of the hard palate, and in its removal the periosteum was stripped from the bone, which was left bare and quite healthy. There was much hæmorrhage, and a good deal of difficulty in stopping the bleeding from one artery. As it became necessary to use the cautery, Mr. Cock took the opportunity of destroying by that means the entire circumference of the wound. The patient returned to the country and was again under the care of Mr. Granger. On the 6th of July following (rather more than three months after the operation) that gentleman kindly reported to me the then condition of his patient.

The wound had healed with a healthy cicatrix; there were no glandular enlargements, and there was no return of the growth. Two small pieces of bone had exfoliated, most probably from the removal of the periosteum and the application of the cautery, as the bone was quite healthy at the time of the operation.

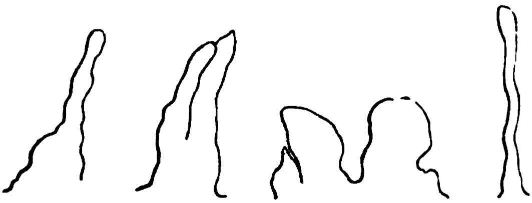
In microscopical structure the tumour consists of a hard mass of fibrous tissue, surmounted by papillæ, and the latter are mainly composed of dense coherent epithelium.

The accompanying figures represent a section of the growth the natural size, and outlines of some of the papillæ enlarged six diameters.

Fig. 3.

Section of the tumour (nat. size).

Fig. 4.



Outlines of some of the papillæ, enlarged six diameters.

The histories of these two cases are very different. The histology of the morbid growths is, as far as I can discover, identical. But whether the two are to be considered as essentially the same elements of disease I am not prepared to say.

In Sir William Fergusson's case the disease did not come to its legitimate issue, as the patient died from another cause, and the question of *malignancy* could not be fairly tested. At the same time, I do not think it was malignant; there was no ulceration, there was no lymphatic-gland enlargement, though the disease had existed a long time. The fact that some of the groups of epithelium were rather bird's-nest-like is not a *proof* of epithelial cancer. I have often seen similar appearances from perfectly healthy gum, especially after maceration, where a cluster of epithelium, coherent together, has "shelled" from the end of a papilla. As regards the recurrence of the disease in this case, it is a question if it was ever thoroughly extirpated.

The coarse anatomy of the growths in the two cases presented some differences, but they were rather those of degree than of kind. In the first instance the structure was soft and lax, in the second it was dense and rigid, and the papillæ in one were five or six times as long as in the other; but all this may have depended on the degree of nutritional activity and rapidity of growth.

ON

AMPUTATION OF THE CERVIX UTERI,

AND OTHER

METHODS OF LOCAL TREATMENT, IN CASES OF MALIGNANT DISEASE OF THE UTERUS AND VAGINA.

By J. BRAXTON HICKS, M.D., F.R.S.

I HAVE been induced to bring forward this subject because I think the advantages to be derived from local treatment have not generally received so much attention as they deserve; for, firstly, too much having been expected from amputation and potential cauteries, the disappointment which has followed has rather tended to cause a distrust of the value of these and other plans; secondly, the known difficulty of eradicating malignant disease in general has tended to discourage the use of everything except antiseptics and opiates; and lastly, because the tendency to hæmorrhage after examination which almost always exists has had an influence in causing many to refrain as much as possible from interfering with the parts. To the repulsive character of the disease I do not attribute any influence on practice, for we may claim for the medical man the will to do everything for the benefit of his patient, however unpleasant it may be to his feelings, and the use of disinfectants will readily remove, for the time required, all excessive unpleasantness. I need not now enlarge on the distressing character of the disease as regards the patient, because all are doubtless but too well acquainted with the severe suffering with which it is commonly attended, as well as the extreme weakness produced by the serous and sanious discharges, and their repulsive nature. But it may be well to consider here what are the principal objects to be attained in conducting the treatment of this disease.

We endeavour—1st. To eradicate or retard the disease.

2. To check the excessive watery and bloody discharges which drain the patient.

3. To destroy their offensive nature.

4. To stop the violent bleeding.

5. To remove the chloranæmia.

6. To remove local pressure caused by some of the growths.

7. To mitigate the pain.

These are the points we desire to attain, and to a certain extent we are able to compass them all with the exception of the first, namely, the complete eradication of the disease. And if we, not too sanguine as to the total extinction of it, are content to do all that we can to arrest its progress and remove its worst features, then I believe we are able to do much towards prolonging the life and promoting the comfort of our patient. And here I may remark that the following observations are based upon the results of cases treated in Guy's Hospital, a short report of which will be found at the end of these remarks, the details of which it is scarcely needful to add.

With regard to the extinction of the disease, my experience has not been very favorable. I have seen only two cases in which such a result may be considered to have been even approximately attained. In one of these the patient lived three years and a quarter after the removal of a very large epithelioma of the cervix; in the other case, the last time I heard of her, the patient was alive four years after the operation. In these cases the diseased parts were very completely removed, and in each instance before the cervix was very much involved. Generally, however, these patients do not apply until the disease is fully established, and consequently not early enough to secure the full benefits likely to be derived from removal. Malignant disease in the uterus, as in other parts, assumes a variety of forms; and as our plan of treatment much depends upon the particular form of the disease, it will apparently be the most practical method of dealing with the subject if we describe the forms separately; and after each, the mode of treatment which appears most suitable. But there is one form in which in particular we can hope for but little advantage from treatment, especially in the early stages. This is the diffuse infiltrating form, which, invading more particularly the connective tissue at the onset, surrounds the vagina, rectum, &c., embracing the canals, but not projecting into them until a later stage. This does not break up so early as other forms, and the bleedings therefore do not occur before a very advanced period has been reached, and generally not until the disease has produced by pressure serious interference with the functions of the pelvic organs.

In proceeding to describe the forms most amenable to treatment, it must be premised that they run much one into another, and frequently require joint modes of management. I shall purposely avoid entering into the minute appearances, having already done so in a former paper, and being now anxious to bring the diseases before the reader as they will present themselves to him in practice.

The first form is that enlargement of the vaginal portion of the uterus which, whether springing from the whole circumference or only from portions of the os and cervix, assumes more or less the form of an inverted mushroom—that is, it presents a large expansion of the lower portion with a smaller neck. It sometimes is attached to only one half of the os, but by the time it comes under treatment the disease has generally invaded the whole ring of the os and a part of the vaginal cervix.

Of this form there are numerous varieties, which differ both as to size and solidity, as well as in the relative amount of the superficial and deep structures involved.

In some the whole is of such a flimsy structure that it is crushed beneath the least pressure, and approaches the encephaloid form. Others have the character of ordinary epithelioma (as of the lips), with lobules of irregular elevation; others, being composed of the altered and enlarged villi of the part, approximate to the appearance of the cauliflower, from whence is derived the name of "*cauliflower excrescence.*"

Very frequently, while the superficies is soft and friable, the base is solid and excessively dense.

Of course, where the whole breadth of the base nearly equals that of the superficial portion, we may assume that the malignant disease has extended to the deeper tissues of the cervix, if not into the body of the uterus itself. This renders the effects of treatment, so far as regards the extinction of the disease, less distinct.

It is in diseases of this class that we find a very large quantity of watery discharge, more or less coloured with blood, but mostly without those severe floodings which proceed from the excavating ulcerations of other forms. Generally the discharge is offensive, but not always so; much, however, depends on the stage of the disease. But be this as it may, the drain thus established upon the system soon reduces the powers, and chloranæmic cachexia is recognised at a first glance.

With regard to the amount of pressure produced by the enlargement of the cervix uteri, everything depends upon its size. Often this is considerable, and if the disease is situated much on the left side it is very apt to cause annoyance to the rectum.

What is the best mode of treating cases of this class?

First, what is the chance of extinction?—for it is in this class in the early stages that we see the best opportunity for a radical cure, if such a result be ever attainable. As already mentioned, I fear the result of the cases, where removal has been accomplished, is not such as to give us much encouragement; still, in the cases mentioned above, where the removal was early and as complete as possible, we have some slight ground for hope that, in endeavouring by removal to retard the disease, we may occasionally be rewarded by more complete success.

But in the removal of the diseased parts I believe we have a valuable help in our treatment, for we certainly do, for a time, remove all the secondary effects of the disease and retard it so much that it has to begin again; consequently, if it ever return, some time must elapse before it shows itself, as before, in the vagina; in the meanwhile the system improves, the powers rally, and for a time the patient resumes, in many instances, a state of apparent health.

In some twenty-eight cases of which I have records every one of the patients made a marked improvement at once; this lasted for many months, in some instances for years, before they returned to their former state, the improvement being such as to lead the friends to the conclusion that the complaint was not malignant. The amount of improvement, and its duration, are almost in direct ratio to the earliness of the period at which the case is treated.

After the removal the discharges entirely cease in the less advanced conditions for a time; sometimes the disease never reappears in the vagina. This is a point to which I am anxious to call attention, because although, as before mentioned, the patient may ultimately die of malignant disease after removal, yet it must not be inferred that she has derived no important benefit. In a considerable number of the cases in which the vagina was not implicated the disease either did not return at all in the cervix uteri nor in the vagina, or not until just before death. The advantage of this, it will readily be perceived, is great; there is no offensive odour, no exhausting drain nor hæmorrhage; life is consequently prolonged, and the comfort of the patient much increased.

If we admit that these results accrue from removal, we may next inquire what is the best method of operating.

So far as my own experience goes, I cannot say I have seen any satisfactory effects from the use of the more powerful caustics, particularly when the disease extends deeply. The disease frequently grows more rapidly than we can with safety apply them; they are tedious and painful in their action (I mean comparatively), and require great care in their application; besides, it is impossible accurately to define the extent to which they shall act. I have therefore discarded them for the purpose of the complete removal of the cervix.

But in the use of mechanical means we have a much more prompt, decisive, and efficient plan, and by the employment of chloroform one of the great objections to its adoption, namely, the pain, is taken away. There are four modes by which it can be accomplished.

The first is to draw down the uterus by vulsellum forceps or such like instruments, and cut the cervix off with the knife or scissors.

The second is to employ the écraseur after drawing the cervix down.

The third is to leave the uterus *in situ*, and remove the cervix by a chain écraseur.

The fourth is to leave the uterus *in situ*, and remove the os by a curved chain, or by the wire-rope écraseur, or by a curved écraseur with a single stout wire.

There is to my mind much objection to dragging down the uterus in this disease, because, on the one hand, there may be infiltrations of it above, which might be injured by the traction; and also because the forceps are apt to give way through the soft growths, and thus the uterus may retract during the operation, though this has been counteracted by passing a wire right through the cervix, whereby to hold it. But the principal objection is, that when the uterus is pulled down so low the anterior and posterior pouches of peritoneum and even the bladder may also be so far pulled down as to be exposed to risk. This has occurred, and I have seen it. It is surprising how exceedingly low down the posterior pouch is situated in some cases. The only safe way is to push the uterus up as high as possible, and then gauge the length of the portion projecting into the vagina; no greater length can be safely amputated.

The objection to both knife and scissors, though of less force as regards the latter if blunt, is that profuse hæmorrhage may come on any time within a few days, as well as at the time of operation, which, besides being annoying and embarrassing to the operator, increases the already depressed condition of the patient. This objection does not apply to the employment of the écraseur, which should, if possible, be applied to the organ in its normal position. This is difficult to accomplish by the ordinary chain, which, having no flexibility in the lateral direction, cannot be applied round the cervix so as to give a transverse section. The endeavour has been made to overcome this disadvantage by curving the end of the instrument. This modification, although it is in some degree useful, is not convenient of application in consequence of its general rigidity; nevertheless, it is, perhaps, the most powerful instrument of the kind, particularly that form of it which has the click-clack movement.

The most adaptable écraseur I have used is the one I have already described elsewhere (vol. vii, p. 252). The only point to which it is especially necessary to look is to have a very strong rope for this work; one with seventy wires will, I believe, be strong enough for any cervix. A loose noose can readily be passed round the neck and tightened by the hand in any position required. The wire rope will bend at any angle to the shaft of the instrument, which should be passed up in front of the cervix.

I have never seen any bleeding from the use of this or of the chain écraseur except in one case, in which the operation was done a few weeks after labour; however, in this case the hæmorrhage was not at all out of control.

Now some have regarded this operation with a certain degree of dread. That fatal cases may arise is possible, but that they are rare I think is the fact.

In all the cases in which I myself have operated, or at which I have been present, amounting to more than twenty-eight, I have never seen any fatal result nor any untoward symptom whatever.

In some the line of division was through healthy, in some through affected tissues, but with the same result.

I am therefore inclined to think that the amputation of the cervix is a less dangerous operation than some have supposed, at any rate, in connection with our treatment of this disease.

To show how little irritation ensues from amputation, I may mention that I removed a very large cauliflower excrescence from a woman pregnant four months without inducing uterine action or any symptom whatever, and without hæmorrhage.

There is, perhaps, one slight disadvantage in the use of the écraseur, which is that the compression of the tissues does not always subside, and that the canal of the cervix remains somewhat occluded, causing dysmenorrhœa. This can readily be removed by dilatation after recovery, but it is only occasionally that it occurs, and it can only be important in those who have not passed the menstrual epoch.

It will be readily perceived that where the disease has extended to the mucous membrane of the vagina we cannot remove this portion, nor can we expect so long a retardation of the disease as in the former kind of case. All that we can look for is the beneficial effect produced by the removal of a large growth pouring out large quantities of serum and blood, and perhaps also the prevention of the return of the disease in this exuberant form, and consequently a diminution of the secretion in future. But in such cases it would be best to add to the treatment by removal the use of styptics, to be hereafter described.

To sum up in a few words, I would recommend in all those cases in which the mass is large, and more or less of a mushroom shape, that the cervix should be removed, if possible, *in situ*, and by an écraseur adapted for that purpose. The results are—marked improvement in the patient's health; removal of the fetid and bloody discharges, and of their effects on the system; a retardation of the disease for a considerable time; and this without much danger as far as the cases to be reported show.

The next form of malignant disease to be noticed is something like the first, but in it the mushroom form is absent, and in its place a large mass of encephaloid disease, having a broad base, fills up the vagina more or less completely; the growth is easily broken down, and bleeds upon the slightest touch.

Now, with regard to this form, it may here be remarked that, although a large bleeding may occur after examination, yet we may wholly break up the mass by the fingers without much more loss; not that this plan should be adopted if by any means we can pass the rope round the mass, after which the severance of it is generally a matter of great ease.

But this encephaloid form, although so easily removed, is very apt to grow again, returning the most quickly of any kind. Still, for a time there is, even in advanced stages of the complaint, marked improvement in the general health, and the blanched emaciated patient sometimes regains colour and fattens, for the discharges in this kind are very profuse, and their removal for a time is attended with great temporary benefit.

After the removal by mechanical means I always like to employ to the base a powerful styptic, which adds much to the efficacy of the other treatment. This application should be repeated in about a week, and occasionally afterwards, as required.

The next variety to be described is one in which there is simply a thin layer of epithelioma on the surface only of the os uteri. In fact, the disease is at a very early stage, so much so that it seldom is seen so soon.

I may detain the reader, perhaps, a moment to point out a feature which will materially assist in the diagnosis of this disease from non-malignant diseases of the os uteri, but which I have not seen noticed in any work on the subject. It is this—if we pass the finger to the line of junction of the os uteri with the cervix, we shall in this condition find that it turns over so as to form at this point a ledge, in such a manner that it is somewhat difficult to pass the finger round it thoroughly, thus giving the first stage towards the mushroom-shape before alluded to.

In the accompanying outline will be seen represented the peculiarity I have endeavoured to describe.

It can only be formed by the extension of the superficies of the os, while the cervix remains of the same size or nearly so; and there is probably no other condition of this part which does increase in that manner. Hence, when we find it, we may pronounce the disease to be epithelioma. At least I have never seen an instance to the contrary.

Now, the treatment in this case is the same as recommended in the more advanced states. If the cervix be long enough to permit it, and as the subjacent tissue is scarcely affected, it may be at least as good a way as any to remove it by the scissors in the manner of Dr. Marion Sims, because there is less difficulty in drawing the parts down.

But in case the cervix should be too short to be removed in this manner, we may employ styptics or caustics. For myself I should prefer the use of styptics, especially the Tr. Ferri

Muriatis, or more particularly the anhydrous sulphate of zinc in powder. For inasmuch as in such cases we cannot expect utterly to eradicate the disease by *caustics*, unless pressed to such an extent as would be attended with danger to the adjacent structures, we shall gain as much advantage by the stronger *styptics*, without the risk of injury. The anhydrous sulphate of zinc has a powerful effect on the abnormal tissue, and but little or none on the sound. We can therefore use it liberally without fear of injury. It seldom causes much pain, and should it do so this is readily relieved by injection of warm water. The elective power, as it were, of this salt gives it a great advantage over the more powerful caustics, which, acting on all tissues nearly alike, require such care as to render it practically impossible to attack all the diseased structures.

At the same time, in the use of styptics we find a point at which they fail; for inasmuch as they do not attack the sound, but act upon the diseased parts readily, there is a line where the diseased influence is at work which is not much affected by their application. Hence, although the superficial portion may be checked, and even cicatrize over, yet the disease may extend in the parts near the sound tissue. Thus, we find both in the use of powerful caustics and of styptics a difficulty in utterly removing the diseased parts.

To a very considerable extent we must also make the same remark as to the mechanical means for removal, the advantages of the latter being that they afford the more sure and rapid mode of treatment in the case of large masses, and the more complete in the incipient stage of the disease.

The anhydrous sulphate of zinc is a very efficient styptic, and has been much commended by Sir James Simpson. It is readily made by placing the ordinary sulphate of zinc in a porcelain basin over a spirit lamp. The salt first melts in its water of crystallization, after which it gradually dries; when completely dried, it can be powdered. On the sessile, early forms of disease it acts with great benefit, and in two or three cases I have seen it immediately and completely check the discharge from the surface, which never returned; and although the disease extended to the deeper tissues, yet the patients were saved the disgust which accompanies the offensive discharges.

In those cases in which the disease has gone on to the more advanced condition, or in which it has run on to the vaginal wall, so that amputation cannot be performed, the use of the styptics has been attended with a subsidence of the discharge in a greater or less degree, causing great relief to the patient. Although it is rare that the duration of this advantage extends over a month, yet upon the repetition of the application a similar result has taken place; so that in a degree more or less complete we have it in our power to do much in these otherwise hopeless cases towards promoting the personal comfort of our patients.

The next form of malignant disease is that more advanced condition in which, with the outgrowth of one part, we have excavation of another, the fœtor of the discharges being excessive. Of course, in these cases no measures for removal can be thought of, but much can be done for relief of the symptoms.

I think I may say that, were it possible to apply either the sulphate of zinc or some other powerful styptic to every portion of the diseased surface, we should remove for two or three weeks the major part of the discharges and their odour. But, of course, in many cases there is great difficulty in doing so. In the more irregular-shaped forms the application will be best managed by the use of a fluid, such as the solution of perchloride of iron, or the Tr. Ferri Muriatis slightly diluted. Carbolic acid might also be employed, but of its use as a powerful styptic I have myself had no experience. A very concentrated solution of alum with tannic acid, almost to the consistence of paste, will act well. The anhydrous sulphate of zinc mixed with glycerine to a paste-like condition is also a good form. When we use styptics it is best to employ a quantity considerably larger than is required to simply cover the part affected, because it will disseminate itself more, and will counteract the diluting effects of the bleeding so difficult to avoid. When a dry powder is employed, it is best to use a speculum, as large as possible, being very careful not to produce bleeding. The powder is to be carried to the upper part by a spatula or spoon, and then still further pushed on by a dossil of lint to the diseased surface itself and well distributed over it. It is as well to use two or three drachms. Where the employment of a speculum is difficult, I employ a glass tube fitted with a piston, which can push the powder beyond the end of the tube.

In addition to this we can employ disinfectants twice a day, but they seldom act beneficially by themselves in removing the odour, except for a few hours.

Again, it sometimes happens that the os and cervix uteri have projecting from their surfaces outgrowths, of a vascular malignant kind, but of polypus shape. These are easily removed by torsion, by the small wire rope, by galvano-caustic, &c., and therefore their removal should be accomplished as early as possible if amputation of the cervix is not practicable; they give rise to a large amount of bleeding whenever subjected to abrasion or the congestion of menstruation. When they occur very high up in the cervix it is well to remove them with the curette, as recommended by Professor Simpson. After the use of the curette it is safe to employ some form of styptic, to stop any bleeding, and to prevent recurrence, if possible. It is seldom, however, that they occur on surfaces otherwise quite healthy; there is generally some epithelioma in the mucous layer from the elements of which they are formed.

The conditions I have described embrace, in outline, the principal variations in which malignant disease occurs in the os and cervix uteri and vagina. There are some forms which are polypoid, arising from the upper parts of the interior of the cervix and body of uterus, but these have been purposely omitted, inasmuch as they are removed in the same way as polypi in this situation, or upon the same principles as here indicated. As before mentioned, the various forms mingle frequently, requiring a mixture of plans of treatment; but I think the divisions just noticed present the disease in the most convenient aspect in relation to treatment. With regard to the employment of local anodynes, we may do much good by opiate suppositories per vaginam or per rectum; indeed, they often act much more efficiently than the same drugs administered by mouth. I cannot say that I have seen much reliable relief from other local remedies; however, these remarks are not intended to extend to this part of the subject, but rather to state the results of the treatment I have adopted in as practical a mode as possible, and to call attention to some points which seem to have been somewhat passed by.

After amputation of the cervix care has been taken to keep the patient in bed for a week, a vaginal injection being used twice a day, with some disinfectant added to it, and an opiate suppository. This point is of much importance in the operations just described, as, indeed, in all other operations upon the uterus and vagina.

Abstract of Cases of Amputation of Cervix Uteri.

Seven cases have been already published in these 'Reports' (see Vol. VII of the present series).

Case 8.—H. T—, æt. 28, admitted June 12th, 1865. Cauliflower excrescence of os uteri; cervical structures also affected; amputation by écraseur; bleeding rather free; did well.

Case 9.—M. A. F—, æt. 34, admitted September 6th, 1856. Hæmorrhage incessant for five months; cauliflower excrescence of the os uteri, about two and a half inches in diameter, filling up and somewhat distending the vagina. The uterus was enlarged, though it was not conjectured that the patient was pregnant, the continual hæmorrhage affording no clue to it, but it was feared that the uterus itself was diseased; however, the cervix was removed without any bleeding by the wire-rope écraseur, and recovery took place without a bad symptom. About a week after, the patient began to feel movements in the uterus, which gave her a suspicion of pregnancy. This proved to be the case, and it was then clear that she was about half-way gone. She went on to full time, and was delivered of a live child (August, 1866), assisted gently by the forceps, by Dr. Ashurst, of Farningham, to whom I am indebted for the report. She is still living, though there is fear that the disease is repeating itself. Had the cervical disease been allowed to continue to full term, I am certain that delivery could not have taken place *per vias naturales*.

A fortnight after the operation no sign existed in the vagina of the former complaint, nor at the time of labour was there anything more than some slight thickening.

Case 10.—E. F—, æt. 23, admitted September 11th, 1865. She had had during one month severe flooding; a large mass of epithelioma was found, involving the cervix uteri, and partly ulcerating. The mass, of the size of a small orange, was removed by a wire-rope écraseur. She did well, and went out without any bad symptom having arisen. Through the kindness of Mr. Gooding, of Cheltenham, I am informed that she died on July 11th, from return of the disease in the glands within the abdomen. To the day of her death there was no discharge nor any other vaginal trouble, and she menstruated regularly till May.

Case 11.—J. M—, æt. 41, admitted August 11th, 1865. Severe flooding at times for six months past. The cervix and os involved in malignant disease, the uterus being fixed in normal position. The vaginal cervix was removed with wire écraseur; no bleeding after the operation. Slight rigors with pyrexia for two days, after which she went on well. Anhydrous sulphate of zinc, in powder, was twice applied to the stump before she went out. She very much improved in health, the chloranæmic condition went off, and she was free from hæmorrhages when last heard of, some time after the operation.

Case 12.—M. A. K—, admitted November 24th, 1863. Epithelioma of cervix, projecting into and distending vagina. The form of the disease was rather sessile, so that on attempting removal by the wire-rope écraseur this could only be done in part, as the growth had somewhat run on to the vagina; some hæmorrhage followed, which was checked easily by styptics. After the removal and application she was much improved, suffering less pain.

Case 13.—J. H—, æt. 45, admitted December 20th, 1865. Hæmorrhage for six months, reducing the patient to an anæmic and œdematous state. All was removed that could be by the wire-rope écraseur, without hæmorrhage. Slight irritative fever after for a few days. Appetite improved, and she went out in a month very much better, without any bleeding or offensive discharge.

Case 14.—The patient had been delivered at full term seven weeks before with forceps. She had had profuse hæmorrhage and watery discharge. There was a large epitheliomatous os uteri, three inches in diameter. Cervix removed by wire-rope écraseur. A smart bleeding ensued an hour after, which was restrained by the application of the actual cautery to one artery. The short time which had elapsed after labour probably was the cause of the bleeding, the uterine veins being still in a dilated condition. She recovered health rapidly, and went out apparently quite well, without discharge of any kind.

Case 15.—A. M—. Bleeding and watery discharge profuse. Anæmic appearance. Cervix filling vagina; removed by wire-rope écraseur without bleeding. Much improvement in every way for a very considerable time; ultimately the disease returned, and the patient died in about twelve months.

Case 16.—Malignant cervix and os uteri, three inches in diameter, nodulated. Caustics and styptics tried without result. Chain écraseur employed, and cervix removed as high as possible. The patient did well for some time, and then complained of dysmenorrhœa. There was some occlusion of the cervix from the compression by the chain; this was removed by a sea-tangle tent. For upwards of three years after the operation was performed, not any vaginal discharge appeared. There was for a year and a half a mass in the inguinal region, probably glandular enlargement; this produced ascites, and œdema of legs. The patient survived three years and a quarter, but had some hæmorrhage towards the last.

Case 17.—A woman about 30 had suffered from hæmorrhages for some time; cauliflower excrescence existed of considerable size. It was removed by wire-rope écraseur. She did very well for two or three years. I heard four years after the operation that she was still alive and tolerably well.

Cases 18—21.—Private cases, under the care of my colleague Dr. Oldham; all of them recovered the operation.

Case 22.—Amputation of cervix by the scissors, for elongation; no hæmorrhage; did well.

I might add six hospital cases, under the care of Dr. Oldham, which have done well after amputation of the cervix for malignant disease.

A CASE

OF

INTERMITTENT HÆMATINURIA,

WITH REMARKS.

By WILLIAM W. GULL, M.D.

THIS disease is characterised by the passing, at intervals, of urine of a dark colour, looking as if it contained blood; no blood-corpuscles, however, are present. The urine is, in fact, of a red Burgundy-wine colour, and is often turbid, forming a dark-coloured sediment on cooling. It coagulates by heat and nitric acid. The specific gravity is generally higher than that of normal urine. On examining it under the microscope we notice granules scattered or aggregated into masses, granular casts of the tubules, a few degenerated epithelium-cells, and crystals of oxalate of lime. No blood-corpuscles, or, if any, merely a stray or a doubtful one, is to be seen. The granules, when carefully examined, are found to consist chiefly of very small prismatic crystals of hæmatin; and even such granules as are not so distinctly crystalline are, on changing the focus, seen to have a somewhat similar crystalline appearance. When the crystals and granular matter are thrown slightly out of focus their colour changes from the claret hue of hæmatin to a pale green.

One of the most striking points in the clinical history of this affection is its intermittent character, the urine suddenly and within an hour or two changing from a deep blood colour to a pale straw colour.

The following case afforded many opportunities of observing these changes. It was usually the early morning urine which was abnormal. The urine after one or two o'clock in the afternoon usually presented the normal characters. The intermittent character of the affection and the aspect of the urine have led Dr. Harley to name this disease intermittent hæmaturia. I prefer, however, to call it intermittent hæmatinuria. The following case differs from those described by Vogel, in which the urine contained hæmato-globuline, in that the patient was not suffering from any exhaustive fever, and the hæmatin was visible in the urine in a crystalline form.

For the following particulars of this case I am indebted to my clinical clerks, Mr. Algernon Ewen and Mr. Henry Denne.

James G—, æt. 33, a labourer, was admitted January 31st, 1866, into the Clinical Ward, Guy's Hospital, under the care of Dr. Gull. He is a married man. He states that he has always enjoyed tolerably good health. His habits have been regular. He is an anæmic-looking man. The history of his present ailment is as follows:—Five months ago he noticed, when passing his water, that it very much resembled blood in colour. At that time he was employed in leather-dyeing, and in consequence he was very much exposed to "wet"—constantly more or less wet through—"the water being frequently sluiced over me." He continued to pass the bloody looking urine for two days, after which his urine assumed its normal appearance. He did not discontinue his work while the urine presented this peculiar character. Three weeks before admission he again observed a similar condition of his urine. This time he was compelled to leave off work, on account of a severe pain in his loins, which prevented him standing. The supposed bloody condition of his urine has continued ever since. If he exerts himself, as in lifting a weight, or walking much, he is sure to find his urine more highly coloured, and more like pure blood.

On admission. He looks anæmic, but is apparently a well-nourished man, but he thinks he has wasted lately. His skin is moist. Temperature 97·5 Some of the cervical glands on the left side are enlarged and hard. His tongue is moist and clean. Pulse 60, sharp and small. Respirations 24. Heart perfectly natural, as regards rhythm and impulse and sounds. Lungs apparently healthy. He complains of a shooting pain, which extends from the umbilicus into the loins, and is increased by coughing or drawing a deep breath. There is no pain in the course of the ureters, and there is no vomiting. The bowels have not been opened for two days. There is no œdema about the legs. The urine is of a dark mahogany colour, slightly acid, of sp. gr. 1014. On standing it deposits a light cloudy sediment. There is no change on the addition of ammonia. When treated with sulphate of copper and liquor potassæ it throws down a blue precipitate tinged with black. A heavy gelatinous-looking precipitate is obtained on boiling, which is not dissolved, but rather increased in quantity, by the addition of nitric acid. There is no evidence of bile, by either the nitric-acid test or that with sulphuric acid and sugar. Under the microscope a very few granular epithelial cells and some granular urates are seen, but no blood-corpuscles.

February 2nd.—The urine has a sp. gr. of 1024, is of an amber colour, and deposits a thick, whitish, flocculent precipitate on cooling. On boiling it a gelatinous precipitate is formed, which disappears on the addition of nitric acid.

Sumat Acidi Nitrici diluti ♏xv,
Infusi Cascarillæ ℥iss, bis die;

Pil. Rhei comp. gr. v, omni nocte.

3rd.—He got up this morning. After walking about the ward for a time he was seized with rigors, and with pains in his loins, extending into his abdomen. He passed some very dark-coloured urine, about three quarters of an ounce. This is opaque, almost of an indigo colour, and clouded with albumen. There is not a quantity of it sufficient to enable the specific gravity to be taken. The microscope shows granular casts, urates, and granular-looking cells, but no blood-corpuscles.

4th.—Urine highly albuminous, sp. gr. 1033.

5th.—He has passed 34 oz. of urine in twenty-four hours;

that passed during the night is of a mahogany colour, sp. gr. 1024; that which was passed this morning is of a normal amber colour, sp. gr. 1010.

On examining the dark-coloured portion under the microscope we observe no blood-corpuscles and no casts, but numerous crystals of hæmatin, having a brilliant red colour, a few crystals of oxalate of lime, and much granular matter. A gelatinous precipitate appears on boiling, and becomes much denser on the addition of nitric acid. The amber-coloured urine also gives a gelatinous precipitate by heat, which disappears entirely on the addition of nitric acid.

6th.—He has passed, during the night, about 30 oz. of amber-coloured urine, sp. gr. 1015. With heat and nitric acid it yields the same reaction as the amber-coloured urine of yesterday. This morning he has passed about half an ounce of bloody looking urine, sp. gr. 1030. The microscope shows no blood-corpuscles, but crystals of oxalate of lime, granular epithelial cells, and several crystals of hæmatin.

7th.—He is again passing amber-coloured urine, slightly acid, and of sp. gr. 1016. With heat and nitric acid there is the same reaction as before, that is, the urine contains no albumen.

8th.—Since yesterday he has passed about 13 oz. of dark indigo-coloured urine, besides 20 oz. of amber-coloured urine. The urine is of sp. gr. 1030, and loaded with albumen. It presents no blood-corpuscles, but plenty of hæmatin-crystals.

10th.—This morning he has passed dark mahogany-coloured urine. This measures 10 oz., its sp. gr. is 1025. It deposits a thick brownish sediment on standing. The microscope shows hæmatin-crystals, oxalate-of-lime-crystals, and distinct casts, the matter forming which is of a dark colour.

13th.—The urine is of a dark mahogany colour, highly albuminous; quantity in twelve hours 16 oz., sp. gr. 1028.

14th.—Early in the morning he passed 16 oz. of amber-coloured urine, sp. gr. 1014, containing no albumen. Since he has been up this morning he has passed 2½ oz. of dark indigo-like urine, loaded with albumen, sp. gr. 1030. This contains no blood-corpuscles, but hæmatin-crystals and oxalates in abundance.

15th.—In the night he passed 21 oz. of straw-coloured urine, of sp. gr. 1012. To-day he passed 18 oz. of dark mahogany-coloured urine, of sp. gr. 1017. Each kind of urine possesses the same characters as before.

16th.—This morning he passed 14 oz. of amber-coloured urine, of sp. gr. 1025. The reaction with heat and nitric acid is the same as before.

18th.—From 5 p.m. until 9 p.m. he passed about 14 oz. of dark mahogany-coloured urine, of sp. gr. 1020, containing no blood-corpuscles.

19th.—During the night he passed 20 oz. of amber-coloured urine, of sp. gr. 1010. He has been out of bed this morning. Up to 2 p.m. he has voided 18 oz. of dark indigo-like urine, of sp. gr. 1023. The microscope shows no blood-corpuscles, but numberless crystals of oxalate of lime, and uric-acid crystals, as well as granular casts and crystals of hæmatin.

20th.—The urine passed in the night is amber coloured; that voided this morning is mahogany coloured.

21st.—He has not passed any mahogany-coloured urine to-day, the secretion being of a dark amber colour; it is not coagulated, but it is cleared by heat, and no precipitate is thrown down on adding nitric acid. On standing it deposits an abundance of pink lithates.

22nd.—His darkest urine is now of an amber colour, and but very little darker than the urine that he ordinarily passes. Its sp. gr. is 1026. No change is produced by heat or by nitric acid. Boiled with liquor potassæ, it does not become darker. On standing it gives no appreciable deposit.

23rd.—Yesterday, before going to bed, he passed 14 oz. of clear amber-coloured urine, of sp. gr. 1014. In the night he passed 18 oz. of light amber-coloured urine, sp. gr. 1005. This morning he passed 15 oz. of urine of a clear amber colour, and of sp. gr. 1020. On standing this gives no deposit.

24th.—His darkest urine is now of a clear amber colour, and of sp. gr. 1020.

25th.—No dark urine is now passed.

26th.—The morning urine is of an amber colour, and of sp. gr. 1020. It has an acid reaction. It is not changed by applying heat or nitric acid. On standing twenty-four hours it throws down a flocculent deposit, consisting of oxalate-of-lime crystals, granular-looking cells, and pavement epithelial cells. The lymphatic glands below and behind the left angle of the inferior maxilla are enlarged and tender, and there is a blush on the skin over them.

27th.—The urine passed this morning is of a clear amber colour, and of sp. gr. 1022. There is none which is darker.

28th.—On getting up this morning he felt faint and trembling, and passed some urine, of sp. gr. 1020, not much darker than usual, but rather smoky. It is rendered faintly opaque by heat, and is not cleared by nitric acid. On standing it deposits numerous crystals of oxalate of lime, seen by the microscope.

March 1st.—The glandular swelling has not diminished in size. He has had some shivering this morning and pain in the loins, lasting an hour or two. During this time he passed $6\frac{1}{2}$ oz. of dark port-wine-coloured urine. It coagulates on applying heat and on adding nitric acid. Under the microscope no blood-corpuscles are seen, but oxalate of lime, granular cells, and granules.

2nd.—His urine presents three varieties—that passed yesterday is smoky, of sp. gr. 1016, and amber coloured; that passed in the night is of a light mahogany colour, and of sp. gr. 1014; that passed this morning is of a dark mahogany colour, and of sp. gr. 1020. Each variety is coagulated by heat and by nitric acid. There is very little deposit on standing. No blood-corpuscles are seen.

3rd.—His darkest urine to-day is of a smoky amber colour, its sp. gr. is 1016. It is coagulated by heat or nitric acid. It contains crystals of hæmatin, but no blood-corpuscles.

4th.—The urine passed in the night is of a dark amber colour; that passed this morning is of a dark mahogany colour. Ordered Tr. Cinchonæ co. ʒij, ex Julep. Menthæ ter die.

5th.—This morning's urine is of a dark mahogany colour, its sp. gr. is 1020. It is coagulated by heat and by nitric acid.

6th.—The darkest urine is of a clear amber colour, sp. gr. 1020. It is unchanged by heat and nitric acid.

7th.—The darkest urine is of an amber colour and turbid, its sp. gr. is 1024. It has an acid reaction. It is cleared by heat, but not cleared by potash, and is not coagulated by nitric acid. The deposit consists of amorphous granules of lithates and oxalate of lime.

8th.—All the specimens of urine are clear, sp. gr. 1012. No change by heat or by nitric acid.

10th.—During the 9th he passed 20 oz. of clear amber-coloured urine, sp. gr. 1016. During the night of the 9th 16 oz. of clear amber-coloured urine, sp. gr. 1018. This morning, 16 oz. of clear amber-coloured urine, sp. gr. 1020. Each specimen is unchanged by heat or by nitric acid.

11th and 12th.—The urine is of a clear amber colour, sp. gr. 1017 and 1022.

13th.—The urine is of a clear amber colour. That passed in the night has a sp. gr. of 1013, that during the day of 1024. There is no change by heat or nitric acid. No trace of sugar is to be detected.

14th.—This morning (the temperature being much lower than it was yesterday) he shivered, felt faint, and trembled when getting up.

Yesterday (on the 13th), from 11 a.m. to 1 p.m., he passed 17 oz. of clear amber-coloured urine, of sp. gr. 1016. Last night, from 8 p.m. to 6 a.m., he voided 26 oz. of urine, sp. gr. of 1012.

This morning, from 6 a.m., he has passed 11 oz. of urine, of a reddish-amber colour, sp. gr. 1028. The last specimen coagulates when heated and when nitric acid is added to it. On standing twenty-four hours a flocculent precipitate is deposited, which, under the microscope, is seen to consist of oxalate-of-lime-crystals, crystals of hæmatin, squamous epithelium, granules, and granular-looking cells; no casts nor blood-corpuscles are to be discovered.

15th.—Yesterday, 11 a.m. to 8 p.m., he passed 17 oz., of a clear amber-coloured urine, sp. gr. 1015. In the night, 8 p.m. to 6 a.m., 13 oz., of a clear amber colour, sp. gr. 1024. This morning, 6 a.m. to 11 a.m., $7\frac{1}{2}$ oz., of a pale amber colour, sp. gr. 1024. These specimens are unchanged by heat and nitric acid.

16th.—Yesterday, 11 a.m. to 8 p.m., he passed 17 oz., of a clear amber colour, sp. gr. 1019. 8 p.m. to 6 a.m., 24 oz., of a clear amber colour, sp. gr. 1018. 6 a.m. to 11 a.m., 10 oz., of a clear amber colour, sp. gr. 1024. Each of these specimens is clouded by heat, but cleared by nitric acid.

17th.—The urine is in all essential particulars the same as yesterday.

18th.—All the urine passed is of a clear amber colour. That voided in the morning is neither lighter nor darker than that passed at other times.

19th.—Yesterday, 11 a.m. to 8 p.m., he passed 18 oz., of an amber colour, not very clear, sp. gr. 1022. 8 p.m. to 6 a.m., he passed 22 oz., clear, of an amber colour, sp. gr. 1020. 6 a.m. to 11 a.m., 13 oz., pale amber colour, sp. gr. 1007. These specimens are all unchanged by heat and nitric acid.

20th.—From 11 a.m. yesterday to 6 a.m. to-day he passed 29 oz., of a clear amber colour, sp. gr. 1022. This morning, 6 a.m. to 11 a.m., 13 oz., pale straw-coloured urine, sp. gr. 1010.

21st.—All his urine is clear and of a normal colour.

22nd.—The urine presents exactly the same characters.

23rd.—Yesterday, 11 a.m. to 8 p.m., he passed 1 pint, of a pale amber colour, not very clear, sp. gr. 1020. 8 p.m. to 6 a.m., 1 pint, of a straw colour, not clear, sp. gr. 1015. This morning, 6 a.m. to 11 a.m., 8 oz., of a straw colour, clear, sp. gr. 1014. Each of these specimens has a faintly acid reaction, and is rendered cloudy by heat, but cleared by nitric acid.

24th.—Urine all normal in colour.

25th.—Urine all normal in colour.

26th.—Urine normal.

He leaves the hospital, being convalescent.

In this case we had a good opportunity of observing the intermittent character of the disease. The attacks generally took the following course. During two or more days the patient voided urine containing hæmatin; then, suddenly, the urine changed, and assumed its normal characters. At another time he passed this dark urine for one day only, the urine being normal on the following day.

These changes in the urine were, however, still more striking and sudden than would appear from the above description; for while the urine that was passed in the early morning was found to contain hæmatin, that which was passed at noon or during the latter part of the day was healthy, and so it continued for as many as eight days. Such is the usual clinical history of these intermittent attacks.

In the first case recorded in Dr. Harley's paper it is stated that the patient suddenly passed five or six ounces of urine of a dark red, or chocolate colour. This used to occur once in the twenty-four hours during two or three days, and then as suddenly disappeared. On one occasion this patient passed in the morning normal urine, at two o'clock he voided urine containing hæmatin, and in the evening of the same day he again passed healthy urine.

In the second case recorded by Dr. Harley it is stated that the urine assumed the colour of blood, a symptom which greatly alarmed the patient, especially as it occurred about three times a week during the whole of the winter.

This very dark urine has a high specific gravity. In the above case it was, at one time, as high as 1033. Dr. Harley has found that in these cases the urine contains an excess of urea.

The albumen present appears to be in an altered condition. On boiling the urine it coagulates and becomes opaque. On adding a few drops of nitric acid it becomes still more opaque, but if an excess of nitric acid be added the precipitate is dissolved. It is considered, therefore, that this precipitate is due, not to albumen, but to globulin. Thus, the great characteristic of this dark urine is that it contains hæmatin and globulin in a free state.

An important point for consideration is the cause of this disease. It appears that there is a predisposing and also an exciting cause. In this case the exciting cause was exposure to cold and wet. In the second case given in Dr. Harley's paper exposure to cold seems also to have been the exciting cause of the disease. In Dr. Fuller's case, brought before the Royal Medical and Chirurgical Society by Dr. Dickinson, it is stated that the patient had been exposed to cold and damp. The report further says that, if the patient, after getting up apparently well and going to his work, chanced to be exposed to cold, he was attacked with shivering and retching, and that an hour or so afterwards he passed urine black like porter. In other cases there has been a history of ague, and the disease has appeared to be in some way connected with malaria poison. But these are not the only causes. There is reason for thinking that a blow or injury to the loins may give rise to this complaint. Thus, a young lady, in getting into a railway carriage, fell, and hurt her back. Soon afterwards she passed dark bloody looking urine. I carefully examined the secretion by the aid of the microscope, but found in it no blood-corpuscles, and only the granular pigment matter of disintegrated blood-corpuscles.

The attacks usually come on with indications of considerable constitutional disturbance. It may be noticed that, in the above report, the patient is stated to have shivered, and to have had pain in his loins so severely that he was compelled to leave off work, and it was soon after this that he voided the bloody looking urine. Another time his attack came on with trembling and faintness. A third time he shivered, felt pain in his loins, and soon afterwards passed the very dark urine.

Besides the exciting cause there appears to be a predisposing cause in these cases, for we are well aware that many people may be exposed to wet and cold without, as a consequence, suffering from hæmatinuria. We are therefore led to assume that there is a condition of the system which, under certain circumstances, is favorable to the development of this affection; and it will probably hereafter be found that it is in this predisposition that the essence of the disease lies.

The question now arises, what do we know of the pathology of this disease? Some suppose that it is dependent on an altered condition of the blood, and that the primary changes take place in the blood itself. An argument in favour of this view is found in the fact that a somewhat similar condition of the urine is sometimes seen in scurvy, in typhus fever, and also after inhaling arseniuretted hydrogen.

In the present state of our knowledge it is impossible to say that the primary change does not take place in the blood, yet there are positive indications that the kidneys are at fault. The pains in the loins, the condition of the urine, and the casts which are present, all clearly show that the kidneys are affected. In order, however, to point out what appears to me to be the pathology of this disease, I must here briefly allude to the functions of the kidneys.

I will not discuss the large question of the physiology of these organs, but will simply confine myself to a few observations on the subject.

The theory of the present day is, that water permeates the walls of the capillaries forming the Malphigian tuft, and runs down the urinary tubule. The epithelial cells which lie in the tubule are supposed to attract urea, uric acid, and other solid constituents of the urine from the blood. These solid constituents are then dissolved in the water coming down from the Malphigian tufts. Now, I do not believe this theory, nor did I when I lectured on physiology some years ago. I consider that the great function of the kidneys resides within the Malphigian bodies, and also that the tubules are infinitely more dynamical than is generally supposed. It appears to me that the tubule is secretive and absorptive, but not excretive. It is thought that the epithelial cells lining the tubules extract the solid constituents of the urine from the blood, and that, while doing so, they are themselves shed from the basement membrane. If, however, we examine healthy looking urine, we find that it does not contain any evidence of such a process of desquamation, for in normal urine it is very rare, indeed, to observe the epithelial cells of the kidneys.

As is well known, one of the great functions of the kidneys is to form the colouring matter of the urine from the colouring matter of the blood. How that change is accomplished we do not yet know; but we have good evidence to show that the kidneys must be in a healthy condition in order that the colouring matter of the urine may be formed. This we see illustrated by watching what occurs when the kidneys are affected after scarlet fever. We frequently see hæmatinuria as a sequela of this disease. The usual history is as follows:—After an attack of scarlet fever the child passes albumen and blood in its urine. The microscope shows that blood-corpuscles are present. The affection is a simple hæmorrhage from the kidneys. But when the child advances towards recovery, and the kidneys begin to resume their functions, although albumen may be present in the urine, we no longer find blood-corpuscles, these being replaced by hæmatin. The urine is dusky, but contains no blood-corpuscles. The kidneys have regained their functions so far that they can now break up the blood-corpuscles. In the next stage the urine, still containing albumen in small quantities, presents uric acid and urates, and we then know that the kidneys are beginning to recover themselves. In the fourth stage the urine contains no albumen, but urates, urea, and its natural colouring matter. The kidneys have then totally regained their functions, and we have seen, step by step, the dynamical power of these organs return.

We may illustrate these changes by the following diagram—

Urine, after scarlet fever, contains—

1st. Albumen and blood.
2nd. Albumen and hæmatin.
3rd. Albumen and uric acid.
4th. No albumen, but urates, urea, and the colouring matter of the urine.

In hæmatinuria some of the dynamical properties of the kidneys appear to be lost. I say *some,* because the organs still retain sufficient power to cast out an excess of urea, but have lost the power to convert the hæmatin into the normal colouring matter of the urine. They ought to eliminate the hæmatin in the condition of urine pigment; instead of that, they eliminate the hæmatin itself.

As, however, the kidneys regain their functions they again convert the hæmatin into the ordinary colouring matter of the urine.

Thus, then, it appears to me that, whatever may be the primary change in cases of this disease, we have at least good evidence to show that the kidneys are affected.

This is the local disease, and the one that must attract our attention. For everyday experience shows that a man may have his tissues greatly diseased, and his blood greatly altered (as in gout), and yet that he may be free from symptoms until some vital organ is affected. Moreover, even then the patient is in danger, not because he has diseased blood or diseased tissues, but because an organ essential to life is losing its functions.

CONTRIBUTIONS

TO THE

PRACTICAL SURGERY

OF

NEW GROWTHS OR TUMOURS.

SERIES V.

CARTILAGINOUS AND BONY GROWTHS.

By JOHN BIRKETT.

In an essay on 'Exostosis'[1] Sir A. Cooper has included under that term all growths springing out of the medullary cavities of bones, or from their investing fibrous tissue, the periosteum. Thus, he writes, "Exostosis has two different seats—it is either periosteal or medullary." In another paragraph he states, "Exostosis is of two kinds, either cartilaginous or fungous." In this passage (read by the light of our present knowledge) it is quite clear that diseases are placed together which, in their elementary structures as well as in their growth and results, are perfectly different. A reader of this Essay, at the present day, will at once see that the author has introduced cases which belong to the class of cancers.

In the first series of these papers ('Guy's Hospital Reports,' 1857) I have published cases in which cancer was developed

[1] 'Surgical Essays.' By Astley Cooper, F.R.S., and Benjamin Travers, F.R.S. Third edition, 8vo, 1818.

in relation with a metacarpal bone (Case 3), with the scapula (Case 4), the femur (Case 5), and a very remarkable case of osteoid cancer of the forearm (Case 14). In all of these the details correspond precisely with those stated by Sir A. Cooper to characterise "Fungous Exostosis."

From the series of cases to follow I have carefully excluded, therefore, every one in which the cancerous element was developed, but I shall have to relate how even a tumour composed entirely of cartilage may destroy life as cancer does, when there happens to be but one growth only of that disease. (Page 407, case 5.)

I have headed this paper "Cartilaginous and Bony Growths" because it is extremely rare to meet with a tumour composed solely of cartilage without bone, or with one consisting of bone without some trace of cartilage. In those enormous masses of new tissue resembling more or less closely fœtal cartilage, to be described in this paper, the bony element occurs usually more especially in connection with the bone from which the growth originally sprouted. But this is not always the case, for sometimes, although the cartilage growth is firmly attached to a bone, no osseous tissue is developed.

The best examples of bone growth without cartilage or with but slight traces of it are seen in some of those cases in which outgrowths of bone take place from the apophyses of the flat or long bones, and remain in their original site some time before removal. But probably in all such cases cartilage tissue was associated even with these in the earlier stages of their development (See prep. 1152^{48}.) Solid hard growths of compact bone-tissue occur under the name of ivory exostoses, and are generally developed upon the bones of the skull.

The tendinous attachments of muscles are also sometimes changed into bone, but whether a development of cartilage precedes that change I have not yet had an opportunity of investigating. When exostoses grow from long bones, even in youth, cartilage is not always found. Such was not the case with a long bony process extending from the trochanter minor which I found many years since in the dissecting-room during the dissection of the body of a young man. In this instance there was not any cartilage. (See prep. 1152^{5}.)

I shall commence with a description of the growths chiefly composed of cartilage. The cases are arranged according to the usual division of the bones of the skeleton into those of the Head, Trunk, and Extremities.

The bones of the head and face.—I place these together because, although the growth of cartilage may spring in the first instance from those composing the face, it sometimes extends to the base of the skull, and even invades the cranial cavity.

A fine example of a cartilaginous growth involving the right side of the face has already been published in the First Volume of the 'Guy's Hospital Reports,' in 1836. The growth is termed "exostosis," but it really consists of cartilage containing spiculæ of bone. The completion of the case is related in the Seventh Volume, 1842.

The man, æt. 24, was fifteen years old when he first noticed something hard in the right nostril. In nine years it had reached the size represented in the drawing 4^{50}, and from which the plate in Vol. I was copied, when Mr. Morgan partially removed the growth by cutting off that part which forms the preparation 1666^{32}. The growth increased slowly, the man survived the operation seven years, and the discovery of the disease sixteen. I believe that in this instance the growth did not invade the cranial cavity, but there is a case recorded in which this happened.[1] A young woman died at the age of twenty-eight years, the disease having been first observed when she was seventeen. She therefore survived the discovery of the growth about eleven years.

Mr. Moore, of the Middlesex Hospital, showed a young man whose face was affected with a similar disease, at one of the meetings of the Royal Medical and Chirurgical Society. The sufferer subsequently died, and the disease was found to extend into the cranium.

A cartilaginous growth, removed by Key from a patient twenty-nine years old, together with the right half of the lower jaw from which it had been growing nine years, is seen in preps. $1091^{15, 16}$.

The vertebræ.—In the museum of the College of Surgeons there is a preparation (No. 207) of "the section of a tumour,

[1] Mr. T. Holmes, 'Trans. Path. Soc.,' vol. x, p. 250, with a plate.

thirteen pounds in weight, which grew in front of the lumbar vertebræ of a soldier thirty-seven years old. It was loosely connected with the vertebræ by its investing fibro-cellular tissue."[1]

Ribs.—Also, in the College Museum is a preparation (No. 200) "of a nodulated cartilaginous tumour, seven inches in diameter, which formed on a man's ribs."[2]

I allude to this preparation as it supplies a parallel case to the one to be described next.

CASE 1.—*Enormous mass of enchondroma developed on the front of the Thorax; about ten years' growth; death.* (Plate II, Drawing 403^{71}.)

For the opportunity of seeing this remarkable case I am indebted to my former dresser, Mr. Edmund H. Galton, of Brixton. It is, perhaps, one of the most extraordinary examples of enchondroma on record; and it so closely resembled some of the diseases of the breast that I thought that organ had been the original seat of the disease.

In April, 1861, M. B—, between sixty-five and sixty-six years old, stated that a large tumour then occupying the left side and front of her chest began to grow about nine years since. From the manner in which the mammary gland seemed to be involved in the growth, it was assumed that the disease commenced in that organ, but upon closely interrogating the patient there was good reason to believe that the first lump was felt through the breast, and was really developed behind it.

The woman stated that she had always enjoyed very good health; she was the mother of eight children; the catamenia ceased thirteen or fourteen years since; she suckled with the affected breast; and the discovery of the first lump was quite accidental, it having been painless for several years. At this time the general health of the patient seemed as good as ever; the pain arising from the growth was scarcely severe enough to complain of, but its size and weight began to be very inconvenient. The drawing from which the plate has

[1] Descriptive Catalogue,' vol. i, p. 90. [2] The same, p. 87.

been taken was made by Mr. Hurst at this date, and it gives a very faithful representation of the external appearances then observable. During the year preceding this date the growth had increased very rapidly. In some portions of it there were places so yielding and elastic when pressed that there seemed to be cysts containing fluid; so much so, that I proposed to attempt to diminish the size of the tumour by opening one or more at a time at intervals. In other parts the larger nodulated masses were hard and resisting. The woman would not, however, submit to any interference with the mass, saying that "as it had been growing so long, she would die with it."

During the following twelve months the tumour increased, and, becoming worn out by the changes which took place, as described by Mr. Galton in the following words, she died about ten years after the discovery of the first lump.

"April, 1862.—The patient's general health appeared good, although the tumour had increased rapidly since her attendance at Guy's Hospital. It presented the appearance of a solid mass, with numerous superimposed cysts of various sizes, in which fluctuation was distinct, and which one by one softened down and ulcerated through the skin. Each discharged first a white glairy fluid, and after a few days pus. Fresh cysts constantly and sometimes almost suddenly appeared. In this way the mass increased in size, until it covered the left side of the thorax, extending in front across the sternum to the opposite breast, towards the back to the posterior border of the axilla; above rising over the edge of the clavicle, and below extending to the last true rib. The pain was not very acute at any time, being rather of a dull wearing character. There was not any œdema of the left arm; the radial and ulnar pulses were not perceptibly affected. The superficial vessels of the neck were slightly distended. There was not at any time serious hæmorrhage from the ulcerated openings. The general health of the patient failed very gradually until the middle of September, when the tumour had increased by about one third since the time that it was drawn at Guy's Hospital. At this date very severe rigors commenced, and after a few days an opening in the skin below the breast was formed by the processes of nature, from which a profuse discharge of pus took place. This continued for a fortnight, during which time the prominence of the tumour rapidly diminished. She continued to decline in strength, and died at last from exhaustion on September 30th, 1862."

"*Necropsy*, twenty-four hours after death.—The body was emaciated, but otherwise generally healthy. The left side of the thorax presented an enormous mass of disease, greatly decomposed, which in parts had evidently been gangrenous before death. The tumour occupied the entire left side of the parietes of the chest, extending from the clavicle above to the margin of the last true rib below, and from the posterior border of the axilla behind to the right edge of the sternum in front, beyond which small cysts encroached upon the right mamma, and some extended below the lower angle of the axilla, and even as far as the middle third of the upper arm. In the principal mass three openings were visible, one about three inches below and in a vertical line with the site of the nipple, another just below the lower angle of the axilla, and the third near the mesian line of the chest opposite the sternal end of the sixth rib. All these openings communicated with each other and opened into a central cloaca, formed by the walls of an enormous cavity, which measured about eight inches in diameter, and contained a large quantity of decomposed pus, blood, and débris of the tumour. On cutting through the entire tumour this largest cavity was exposed, and in endeavouring to dissect it from the walls of the chest a cartilaginous mass was noticed, continuous with and inseparable from the cartilage of the fourth rib, forming what had evidently originally been the pedicle of the tumour. The fibres of the pectoral muscle could not be clearly made out, and no bands or septa of fibrous tissue could be distinguished throughout the growth, which consisted of innumerable smaller cysts extending in all directions from the larger one. Some of these were entirely broken down; some contained a puriform fluid, and others a white glairy fluid. The nipple was healthy, but the ducts were dilated and appeared to open into the largest cyst. Immediately around the nipple a layer of healthy skin and adipose tissue covered the gland. There was no opening into the thoracic cavity, but the intercostal muscles between the fourth and fifth ribs were attenuated, and the sternal end of the fifth rib partially necrosed."

"I am indebted to Mr. Brennan for the report of the patient's state before death, as she was under his care from April last. He kindly allowed me to be present at the necropsy."

Bones of the pelvis.—Passing on to these bones, we have a fine example of a growth of cartilage arising from the os innominatum, and invading the cavity of the abdomen.

Case 2.—The man, æt. 26, was under the care of Mr. Morgan in 1845. The tumour was of about three years growth, and weighed twenty-two pounds. (Prep. 1132^{52} and 1132^{53}.)

Necropsy.—The right leg was distended with serum. The tumours occupied the right half of the abdomen, extended beyond the mesial line, especially below the groin, and pressed up the liver, &c. The caput coli was adherent to it anteriorly and above. It was made up of nodules of cartilage, with cavities containing two or three pints of ropy brownish mucus and coarse grains of softened cartilage. A long cut downwards removed the inner and anterior third of the mass; another removed the upper right and back third, with but traces of bone. The base (third) was then left and brought away with the chief part of the ilium; it contained more bone, and a thin whitish body, coarsely, feebly, and darkly cancellated and buried in an excess of soft varying cartilage.

The pelvis of the right kidney was dilated and distended with water.

The preparation (1132^{52}), having been macerated, shows the bony elements composing that portion of the growth more immediately in connection with the ilium. A large coral-like bony projection is growing from the crest of the bone, and another circular growth is springing from the dorsum, and having traversed the bone is visible on the venter also. These now appear to have been osteophytic outgrowths of bone into the cartilaginous growth. They are beautifully delicate, without, however, the common appearance of ordinary bone.

In the College Museum (prep. 206) is a very interesting cartilaginous growth removed from a woman 34 years old. "After death the tumour was found closely connected with the sacro-iliac symphysis and the adjacent bones. It projected also at the back of the pelvis."[1] This woman had had several children, each labour being more difficult than the preceding one. The last labour, thirteen months before death, was very severe and protracted. A large immovable tumour was felt in the left side of the pelvis.

A case similar to this one was for some weeks in the Obstetric Ward in Guy's Hospital during 1865. The patient, a well-formed, healthy looking woman, about thirty years of age, had a hard nodulated mass occupying the venter of the left ilium. As the growth enlarged she experienced increasing pain from compression or stretching of the anterior crural nerves. As it was not considered practicable to remove the tumour, she left the hospital.

Mr. Holthouse has described a cartilaginous tumour originating in the left os innominatum of a male, æt. 35.[2] It seems to have been of about three years' growth when he died, worn out with pain and suffering. "The tumour occupied nearly the whole of the abdominal cavity, but its outline could be felt on the right side, and it had pushed the intestines over into the right hypochondriac region, where they could be recognised on percussion. The weight of the tumour was thirty pounds." The details of this case are most interesting, but I must refer the reader to the book in which they are published.

Bones of the upper extremity.

The scapula.—In the essay before quoted Sir A. Cooper states that he does not "recollect to have met with any instance of this affection of the scapula." By reference to our museum we are now enabled to supply three cases. One of these, sent some years since by Mr. R. Nunn, of Colchester, affords a fine example of an enormous growth, which commenced rather late in life, was not observed until some weeks after the receipt of a blow upon the shoulder, and seems to have been developed in relation with the neck of the bone. The second case forms the subject for one of the illustrations of this paper; and the third I had entirely under my observation from very soon

[1] 'Descriptive Catalogue,' vol. i, p. 89.
[2] 'Trans. of Pathol. Society,' vol. viii, p. 367.

after the commencement of the growth to the death of the patient.

Case 3.—*Enchondroma of scapula of about seven years' growth; death.* (Drawing 5^{25}, prep. 1098^{20}.)

(The following report of this case was sent to the museum by Mr. Roger Nunn. Note-Book 2, p. 48.)

In April last I was requested to visit James Goody, who was residing about six miles from this place, and who had been suffering for some years from a tumour on the shoulder. Having seen this man about three years previously, I was much surprised to see the immense increase which had taken place in the size of the tumour, and upon making very particular inquiries of his wife and neighbours I heard the following particulars:

He was a carpenter and bricklayer, æt. 57, and had, until within seven years, enjoyed in every way good health. About that time, whilst assisting to move a piece of timber over a saw-pit, he was struck by it on the shoulder; this accident gave him considerable pain at the time (which he termed a wrench), and prevented him from following his employment for a fortnight, at which time he had apparently recovered from the effects of it. He continued perfectly free from pain and inconvenience for about two months, when he was seized with an acute gnawing pain at the top and back part of the shoulder, which gradually increased during three months, when a small swelling was perceived situated about the neck of the scapula. For this he applied to a medical man, who gave him liniments, &c. &c., which he applied, but without avail, and both the swelling and the pain increased for about three years, at the end of which time he visited the Essex County Hospital as an out-patient. The tumour had then gained a considerable size. He was leeched, and other remedies were taken to reduce it, none of which, however, had any effect, and it still continued to increase. The pain, which until now had simply been of a gnawing kind, had of late become lancinating, accompanied with a feeling of intense heat. He continued his work until the Christmas of 1834 (being four years after the swelling commenced), and was even able to follow his occupation of sawyer up to the summer of the same year. In the following April, 1835, he was advised to go to London, where he applied to Mr. Liston, at the University College Hospital, who, after some little time, determined upon removing it, which he then proposed to the man. I believe Mr. Liston thought it to be an exostosis. It then extended from the scapula downwards to the ninth rib, and upwards over the shoulder to the clavicle; backwards towards the median line nearly to the spinal column, and down the humerus, so as entirely to encircle the head of that bone and the scapula. The man, however, would not give his consent to have the whole removed, but would allow only three quarters being taken away. The operation was then, of course, given up, and he returned home to his family. From that time until November last it rapidly increased in size, when it had reached to that extent that when sitting he was obliged to have it supported upon a table, and, although with sufficient general strength, he was only able to walk with assistance, as the weight was so great it quite overbalanced him. After this he was obliged to keep entirely to his bed, not being able to support it in any but the recumbent posture. During this time his general health and appetite had been good, his spirits high; at the time I saw him he was evidently fast sinking; his appetite had failed him for several days. He had suffered much from alternations of chill and fever; had had no sleep; constant severe pain, with great restlessness; hands and feet constantly cold, and the back part of the tumour about to slough. As it was now so painful to him to be moved, I deferred making any minute examination of the swelling until after his death, which took place on the following morning.

Necropsy.—The anterior part of the tumour presented a very irregular surface, in some parts soft and yielding to the touch, as of fluid, in others firm and almost bony; some much distended and injected, others depressed and livid; on the whole much resembling a fungous growth. The posterior surface was regular, and in parts in some stages of disorganization. It extended anteriorly to the centre of the sternum, and posteriorly to the spine; superiorly above the shoulder and inferiorly to the right ilium, presenting somewhat the form of an irregular cone, the basis of which enveloped the whole side and three fourths of the humerus, the *apex resting on the crest of the ilium.* On dissecting off the skin I found it in many parts to consist of numerous cysts filled with a serous kind of fluid, intermixed with numerous portions of lymph, in others cartilaginous, and in others even osseous. Removed with it the scapula and greater part of the humerus, with a small portion of clavicle, to which it was attached; it had no strong attachment either to the ribs or crest of ilium."

The drawing and preparation in the museum merely show the texture of the growth, which is, in the piece of it preserved, rather more broken up into distinct lobules than we commonly see in these growths. This condition may, however, be explained by changes taking place in so large a mass as was somewhat rapidly developed in this particular instance.

Case 4.—*Very large enchondroma of scapula, about two and a half years' growth.* (Pl. I, drawing 5^{18}, prep. 1098^{25}.)

E. G—, æt. 36, was admitted into Lazarus Ward on the 10th February, 1866, under my care. The following history of the case and description of the man's condition were given to me by my dresser Mr. Gill, and Mr. S. S. Brown, the reporter.

About the end of the summer of 1863 the man discovered a small "lump" on the infra-spinous fossa of the left scapula. This seems to have been the point of commencement of the growth, as accurately as could be ascertained from the patient and his wife at this date. His wife said when it was first noticed it was "about the size and shape of a native oyster," but harder. He did not suffer from any pain in the swelling then nor until some months afterwards. The surface of the lump was quite smooth. During the first eighteen months of its growth the increase was gradual and slow, although at this time it had reached nearly the size of his head. Even now it was painless. About this period the tumour began to increase much more rapidly. The pain he now suffers from began about eight months since, is dull and dragging, and it was more severe than it is at the present moment. He has had less pain since the skin ulcerated. He never felt any difficulty in breathing whilst the tumour was growing, and the cough with which he is now suffering so severely commenced but a few weeks since. He complains besides of a very severe pain in his head, which causes more distress than the tumour. The pain arising from the growth has never interfered with sleep, and it has been so trifling that he continued to follow the occupation of a maker of the leathern parts of army accoutrements until within a few days of admission into the hospital.

It is somewhat remarkable that he never asked medical advice until the last week. He accounts for this strange and fatal neglect of himself by stating that his great anxiety had been to work for his family as long as he could possibly do so.

The man was short in stature and extremely emaciated; his skin was of sallow complexion, containing a considerable amount of dark brown pigment. There were several cicatrices of old ulcers on his back, which were probably connected with the syphilitic poison, as he confesses to having had the venereal disease. With this exception he states that his health has been always good, and that he has never been stinted of food. His mother died of consumption, but his father is at present alive and very healthy. He was brought up to and followed the trade of a painter, but he never suffered with any symptoms of lead poisoning. He cannot remember any injury of the bone affected.

Description of the tumour.—The whole of the posterior thoracic region and left side were involved in an immense growth, which measured 38 inches around its largest circumference, $23\frac{1}{2}$ inches in its vertical axis, and 24 horizontally. This growth extended backwards to within three inches of the spinal column, forwards in front of the axilla nearly to the left nipple, and the inferior border extended downwards to within an inch of the crest of the ilium when the patient was sitting upright. The pedicle of the growth was not attached further downwards than for about an inch below the nipple, and by moving the upper extremity it was clearly ascertained that the growth involved the whole scapula, without in any way implicating the humerus. Neither was the integrity of the clavicle in any way affected. The acromion of the scapula was distinguishable, and the contiguous portion of the spine of that bone was traceable for a short distance. The movements of the shoulder-joint were free, although limited. The tumour was not anywhere attached to the ribs. The axillary and cervical lymphatic glands were not diseased. The axillary artery traversed the anterior surface and upper border of the tumour in a groove or furrow formed between two masses of the growth. It was easily felt, being pushed close beneath the integuments, and the pectoral muscles were much wasted. The pulse in both radial arteries were of equal strength. There was not any numbness of the left arm. The new growth was an irregularly oval mass, the skin covering it being congested and having numerous dilated veins traversing it; but it was not adherent to it, nor in any way infiltrated except the lower part, and there with serum only. At two spots the skin had ulcerated, and through these openings a passage led into the substance of the mass. From these sinuses a very offensive sanious fluid continually flowed. The surface of the mass was remarkably irregular, and the whole of it seemed to be composed of separate lobes of harder or softer consistence, some of the lobes from their hardness feeling like masses of bone, others from their more yielding texture resembling cartilage, whilst others felt elastic like a cyst containing fluid. From the external indications and the history of the case, the conclusion was that the growth consisted of cartilage, and was growing from the scapula, having originally commenced in that bone.

The idea of removing the tumour was, of course, entertained, but the state of the patient's general health at the moment of his admission into the hospital precluded all hope of his recovering from an operation of a nature so formidable as would be required in a case like this.

The state of his general health was as follows:—He was emaciated to an extreme degree; he had passed sleepless nights, the tumour preventing him lying in any position of comfort or repose; he had total loss of appetite; his pulse was very feeble and very rapid, although the sounds of the heart's action were fairly normal. His respiration was short and hurried, he had constant but slight cough, his voice was feeble and interrupted, and he complained of pain at the epigastrium and on the left side. The blood-vessels of his face were congested, and the facial expression was distressed and anxious. Still, there were no marked indications of active disease in the lungs.

Food of every kind he could take was freely given to him, with stimulants, tonics, and sedatives, but in spite of all treatment he gradually declined, and died February 16th, the sixth day after admission.

After death I attempted to remove the tumour with as much caution in dividing important structures as I should have employed had the patient been living, and I believe I might have effected the removal of the growth together with the scapula without cutting the brachial plexus or axillary artery. An enormous wound was, of course, made, nearly the whole of the side and back of the thorax having the integuments detached, but the operation was practicable; so far as concerned the removal of the tumour, sufficient integument was saved to form flaps.

The weight of the tumour was about twenty pounds. It constituted a well-defined mass, over which the muscular fibres of the region were in some places stretched out. With the exception of the posterior surface, where there was a great hole leading into the centre of the growth, its surfaces were perfect. To a certain extent the surface in relation with the thorax was moulded to the shape of the parietes of that cavity, the extremely nodulated surface being the superficial one, which had not encountered much resistance to the development of the new tissue. The general outline of the scapula was entirely lost, although the glenoid cavity was visible and quite unaltered. Also, the acromion formed its normal prominence, and the supra-spinous fossa was well marked. A vertical section of the tumour was made at right angles with the spine of the scapula. Near to the superior extremity of the mass traces of the original spine of the scapula are visible in the preparation. With these a large osseous centre was somewhat indefinitely continuous. Ramifications of bony tissue spread outwards from this towards the surface of the growth, without, however, reaching it in any place, for the whole of the more superficial parts consisted of beautifully bright clear cartilage, of firm consistence and lobulated structure. The lobes were made up of lobules very even in size, which were about half an inch in diameter. Parts of the tumour were very soft, others entirely broken down and almost fluid, forming a jelly-like substance. Some of the tissues had died, and formed a very offensive semi-fluid grayish slough.

Necropsy.—Dr. Moxon recorded the following account of the after-death examination:—The body was spare, almost to wasting. The membranes of the brain were slightly thickened. The left pleuræ were adherent uniformly and universally by old adhesions. The right lung had a thin coating of recent lymph over its lower lobe. There were not any lobular abscesses discoverable. The lungs generally were emphysematous, all parts of their surface having elevations the size of small shot to split peas scattered about, each having a black zone about it, and each seeming to consist of distinct air-vesicles. At the edge was a fringe of vesicles of the size of shot to peas. The lower right lobe was tougher and firmer than the rest, like the indurated chronic pneumonia in low degree.

The right side of the heart was somewhat, but distinctly, larger in proportion than is proper; its texture was also increased and its substance hardened. There was a large, fleshy, pale clot in the right side; very little indeed in the left, which was contracted firmly.

The liver weighed sixty-six ounces. Its section was coarse, from the sharply defined distinction between the portal and the hepatic fields.

The spleen weighed seventeen ounces; it was very soft and grumous.

The kidneys appeared healthy, but they were of large size and weighed fourteen ounces.

CASE 5.—*Enchondroma developed on the scapula, but extending into the surrounding textures; removal of growth; recurrence, and death about two years after its discovery.* (Drawing 197^{95}, prep. 1098^{30}.)

I was requested by Mr. Kellock, of Stamford Hill, to see a patient of his, in March, 1861. I was introduced to a very healthy looking lady, forty-five years old, who had enjoyed uninterrupted good health; had had several children, the last some years since. The catamenia were persistent. I was told that, six months since, she accidentally felt a little above the inferior angle of the right scapula, and on the surface of the infra-spinous fossa, a firm circumscribed swelling. It was then about one inch in diameter. The swelling had never been very painful. There was not any difficulty in being certain that the tumour, whatever might be its nature, was intimately associated with the scapula, for it was immediately influenced by the slightest movement of that bone. When I saw it the shape was rather oval, the longest axis being vertical and measuring about two inches. It was remarkably circumscribed, projected about half an inch above the surface of the bone, was firm, resisted all pressure, and no complaint of pain was made under manipulation. At this time I suspected that the swelling arose from some chronic inflammatory effusion, and measures were adopted upon this assumption in relation to treatment.

I did not see the patient again until the next year, in February, 1862, an interval of one year. The growth had now reached a considerable size. It gave her great pain and trouble, but the integuments were unaffected, although it had advanced towards the axilla and occupied the whole of the infra-spinous fossa of the scapula, and was even identified with its spine. It felt very firm and resisting in some parts when pressed hard, in others softer, and even here and there disposed to fluctuation, or, rather, as if consisting of cysts tightly circumscribing some soft solid. The axillary lymphatic glands were healthy. Movements of the growth and scapula were perfectly coincident. Now, from the healthy aspect of the patient, the manipular indications of the tumour, its steady increase and intimate connection with the scapula, I diagnosed the growth to consist of cartilage.

During the preceding twelve months she had been under the treatment of another surgeon, who had employed various means in the hope of dispersing the swelling.

It seemed to me that there was no alternative but to attempt the removal of the tumour, together with as much of the scapula as was implicated in the disease. To this proposal the patient consented. Accordingly, Mr. Kellock and myself undertook the operation on February 12th.

The patient was placed fully under the influence of chloroform, and a vertical incision through the integuments enabled us to expose completely the surface and borders of the tumour, and to detach it together with the body of the scapula from the surrounding tissues. Then with bone-cutting forceps we detached the whole of the infra-spinous fossa of the scapula, or rather that part of the tumour occupying its site, for we were unable to find any vestige of that division of the bone. Upon examination we found that the disease was extending under the root of the acromion process into the supra-spinous fossa, and all that we could find we removed. There was not much hæmorrhage, and each divided artery was secured as soon as possible. The parts of the scapula we did not remove were the glenoid cavity, supra-spinous fossa, part of the spine, and acromion process.

That part of the growth below the spine of the scapula was contained in a well-defined fibrous envelope. The mass consisted entirely of cartilage, firm and solid in some parts, granular and easily breaking up on pressure in others. The section was beautifully hyaline. There was a capsule at one border, containing a material of semi-solid consistence, and, indeed, almost fluid, tenacious like very thick mucus. Not a vestige of the shape of the infra-spinous fossa was traceable in the section of the growth removed, although here and there the finger detected the roughness of osseous tissue when it was gently moved over the section. The cartilage-tissue was granular where it invaded the supra-spinous fossa and its muscle, and, indeed, we entertained a suspicion that these minute granules of cartilage extended into the surrounding muscular textures even to the subclavius muscle.

The wound healed favorably, and the patient recovered its effects very fairly, but about the end of May there were signs that the disease was again growing. These were swelling, or rather fulness, and pain at the upper part of the cicatrix. At the end of June there was a large tumour occupying the whole scapular region, and extending towards the spine, beneath the clavicle and into the axilla.

In July the cicatrix ulcerated at the upper two thirds, and the growth projected through the opening; its surface was curiously mottled, and small patches of cartilage were clearly seen on it. The edges of the ulcer were quite independent of the growth, and not attached to it, but were merely lying upon it.

The general health of the patient was tolerably good, and, although she complained of shortness of breath and weakness, there were not any signs of either thoracic or abdominal visceral complications.

At this date the repetition of the operation was considered. From the wide-spread ramifications of the growth, it seemed useless to attempt any further operation unless the whole of the upper extremity was sacrificed, a proceeding, of course, of considerable risk. Neither the patient nor her advisers felt disposed to urge another operation, and, as she did not suffer acute pain, she made up her mind to rest content with palliative measures.

After the skin had ulcerated there was a continued discharge of serous, sanious fluid, the patient lost her appetite, passed restless nights, and at last sunk from exhaustion in December, 1862.

An after-death examination showed a large development of fat beneath the skin. The muscular tissue of the heart was weak and pale; the lungs were collapsed, but their texture was normal, and not a trace of any new growth was found. The liver was large, pale, soft, and loaded with fat; the kidneys were soft and flaccid; there was no serous effusion in any of the cavities.

The cartilaginous growth was confined to the scapular region, and had not invaded either the ribs, the clavicle, or the humerus. The shoulder-joint was quite healthy; but the important soft parts, the brachial plexus and axillary vessels, were closely associated with the growth, which extended beneath the clavicle. This after-death examination therefore showed that considerable difficulties must have been encountered in any attempt to eradicate the growth. The arm might certainly have been allowed to remain, but, unless the glenoid cavity of the scapula had been taken away together with the tumour, a second operation would, I believe, have been futile.

The result of this case teaches the following lesson. It is clear that the patient died from the effects of the local growth as a primary cause, for there was not found a trace of any specific kind of growth in mass interfering with the function of any important organ. In a most decided manner it indicates the importance of performing an operation for the eradication of the local complaint at the earliest moment after the employment of all means which have failed to reduce the size of the swelling or arrest of its growth. Had an operation been performed in this case when the tumour was entirely circumscribed and perfectly confined to the infra-spinous fossa of the scapula, that part of the bone might have been easily cut away together with the growth, and a perfectly healthy wound left; as it was, when the operation of removing the tumour was accomplished, although a very large quantity was cut away, yet there seemed to be, in the space above the glenoid fossa and about the neck of the scapula, minute granular bodies, which, on examination, proved to be most tiny particles of cartilage. These were, I have little doubt, the seeds, as it were, of the second tumour, and I do not believe that any further prosecution of the operation would have prevented another growth. If I had another case of this kind to treat I think the following plan of treatment would be fully justifiable after the result of this case. There is something very characteristic or rather definite about the tumour, and it occurs in a region where we have the opportunity of forming a diagnostication of its nature by the method of exclusion; thus—Is the swelling inflammatory? There is a tumour for the origin of which the patient cannot account; perhaps there is a history of a blow upon the part. Then the swelling might depend upon chronic periostitis, with its characteristic products, or upon effusion of blood, or some such specific cause. Assuming inflammatory action to be the origin of the swelling, there would be almost certainly more pain accompanying its formation or pressure upon it, as we observe in nodes, for example. Supposing it to be caused by effused blood, decrease of its size rather than progressive increase would be noticed; but after steadily watching the progress of the tumour whilst under treatment, and being unable to associate its advance with either of the above-mentioned causes, we now arrive at this inquiry—Is it a new growth? The slightest irregularity of surface, a circumscribed outline, pressure causing little, if any, pain, a certain amount of firmness, and, at the same time, elasticity on compression, would lead the surgeon to suspect a new growth.

What now should be the next step in treatment? I would strongly urge an exploration. Even if the disease now turned out to be inflammatory, an incision would not be attended with harm; but if it showed itself to be dependent upon a growth of cartilage, or any other tissue, now would be the moment to eradicate it from the affected bone.

The humerus.—Sir Astley Cooper has published a very fine example of a cartilaginous growth attached to the humerus, on account of which he performed amputation at the shoulder-joint.[1] The preparation of this disease is in the museum at St. Thomas's Hospital (sect. C, 224). The patient was a female, æt. 30. "The tumour situated at the upper and external part of the left arm, so high up that on a superficial inspection it seemed to be connected, not only with the humerus itself, but also with the clavicle and scapula, rendering it probable that it had an attachment to the glenoid cavity of the latter bone." I have quoted this passage because it has an important relation to the operation of amputation at the shoulder-joint. The disease seems to have followed injury of the part, and to have been growing about three and a half years before its removal.

Mr. William Adams describes an enchondromatous tumour occurring in a male, æt. 61, which sprung from the right humerus, grew rapidly for two years, ulcerated, and destroyed the life of the patient.[2]

The following is of great interest:

Case 6.—*Enchondroma of the humerus, twenty-five years' growth (first observed when the patient was about fifty-five years old), reaching an immense size.* (Drawing 5^{15}.)

When on a visit to Mr. Rump, of Wells-next-the-Sea, Norfolk, he took me to see the man whose case is here recorded. It was then difficult to be certain whether the humerus

[1] Essay before quoted, p. 203; and 'Med.-Chir. Trans.,' vol. ii, p. 264.
[2] 'Trans. Path. Soc.,' vol. i, p. 344.

or scapula was the bone affected, in consequence of the great size of the tumour; but from the account given by the man it seemed to be probable that the growth sprung from the upper part of the humerus in the first instance, like the one described by Sir Astley Cooper.

The following account was written in July, 1848.

W. H—, æt. 68, was born in Burnham Market, Norfolk. At the age of fourteen he went to sea, and was on board the Bellerophon when Napoleon surrendered to the British flag. At the conclusion of peace he was paid off, and went to reside at Wells, about thirty-four years since. He obtained employment as a woodman on the Holkham estate. One night whilst in bed, after a day's work of wood-carrying, he was suddenly seized with great pain in the left shoulder, and for a time he lost the use of the corresponding arm. Soon after this he noticed an enlargement about the size of a walnut, and perfectly movable, on the summit of the shoulder. It increased very slowly in size and was not attended with much pain, so that he was enabled to follow the occupation of an oyster-man. This state of things continued until about ten years since, when the tumour began to grow rapidly, and the arm became powerless and the movements limited.

The upper division of the tumour increased rapidly, whilst the lower remained movable and grew but slowly. From this date the enlargement has steadily gone on, and his general health, which had been good hitherto, began to fail from continued pain and suffering.

In 1844 he was admitted into the Norwich Hospital under Mr. Crosse, where he remained two months. The integuments over the tumour were not discoloured, but in different parts they were traversed by large, blue, tortuous veins. Pressure upon the tumour does not cause pain, but it feels of a uniform stony hardness throughout.

Between July, 1848, and September 1851, the tumour had enlarged, and the integuments in places became slightly red; at these spots an indistinct sense of fluctuation is perceptible.

Measurements of the tumour were taken in 1848 and 1851, and were as follows:

	1848.	1851.
Vertical measurement, from above to below	18 inches.	21 inches.
Transverse or horizontal, from back to front	18 „	21 „
Circumference at base	28 „	30 „
Circumference at largest part	30 „	33 „

From these figures it appears that the growth increased at about the rate of one inch per annum in all directions.

This man survived until November, 1858. For the last two years of his life he had been confined to bed in consequence of the great bulk and weight of the tumour, which rendered him perfectly helpless. Before his death, which was occasioned by chronic bronchitis, at seventy-eight years of age, the forearm of the affected side became very œdematous, and slight ulceration from pressure existed in two or three places, but no disintegration of the tumour itself occurred. At the period of this man's death Mr. Rump was seriously ill, and therefore the tumour was not preserved. An after-death examination of it, however, showed that it was composed of cartilage in great part, with here and there patches of bony hardness.

The striking feature of this growth, as well seen in the drawing, is the subdivision of the great mass into four divisions, an upper, lower, and two lateral. Its remarkable prominence, too, is very characteristic, and the firmness and hardness throughout were good indications of its structure.

A case very much resembling the last one is related by Mr. Liston in 'Practical Surgery.' The tumour had been growing for nearly forty years around the superior three fourths of the humerus of a surgeon in the navy. "Amputation at the shoulder-joint was performed, and the patient recovered from the operation, but died two months afterwards with disease of the chest." The preparation is in the museum at the Royal College of Surgeons.[1]

In connection with the bones of the *forearm* I have not seen a cartilagious tumour.

The bones of the *carpus* are rarely, but those of the *metacarpus* and *phalanges* are very commonly affected by growths of cartilage developed upon their surfaces. In the majority of instances the cases which have been under my observation

[1] 'Descriptive Catalogue,' vol. ii, p. 167.

were boys and girls; and where the tumour was seen in this region of an adult the commencement of the growth was clearly traced back to childhood or youth. Very commonly, these tumours have a marked and very decided bony shell or capsule; their centre consists of cartilage, with granules of bone, and their base of attachment seems to be the surface of the bone upon which they grow. These conditions are illustrated by a series of drawings, numbered 29^{15} to 29^{19}, and preparations 1121 to 1122^{50} and 1124^{55}.

In one instance the growth seems to have sprung from the cancellated tissue of the head of a metacarpal bone (drawing 30, prep. 1124^{50}, and cast 3). This patient was under Mr. Aston Key, who was obliged to remove the whole finger in consequence.

A very fine example of these growths, which began in childhood, is shown in the drawing (5^{105}) of a hand which, from the neglect of the patient, nineteen years old, became excessively deformed in consequence. This young man was under the care of Mr. Cooper Forster, who was obliged to remove a finger. A section of the growth is delineated (drawing 5^{106}), and a preparation is preserved in the museum (prep. 1122^{25}), as well as a model.

Bones of the inferior extremity.

Femur.—There are in the museum some fine drawings and preparations to show cartilaginous growths about this bone.

One, a large enchondromatous tumour affecting the upper part of the thigh-bone (prep. 1160^{86}), was removed after death from a patient under Mr. Key. The growth reaches as high as the neck, and involves the trochanters as well as a large part of the shaft. In this particular instance the cartilage not only grows around the bone, but it has quite destroyed its form, so that only a few portions of the shaft are seen in the midst of it. This condition contrasts strikingly with the relations of the growth to the femur described in the next case.

Mr. Busk describes a case[1] in which a large irregular tumour, partly enchondromatous, was situated at the lower end of the right femur at its posterior surface. It was developed in a male,

[1] 'Trans. Path. Soc.,' viii, p. 375.

twenty-seven years old, and was first observed in boyhood, at the age of thirteen. It was therefore about fourteen years' growth. The tumour was removed by amputation of the member above the middle of the thigh. The lithographic drawing in the 'Transactions' of the society shows that at a short distance above either condyle a well-defined, nodulated, irregular-pointed growth was attached. For the full details I must refer the reader to Mr. Busk's description. The patient did well and is still alive now ten years since the operation. The preparation of the tumour is in the museum of St. Bartholomew's Hospital.

The following case is one of great interest, chiefly on account of the successful result of the operation. It is also a fine example of cartilage developed in the cancellated texture of a long bone.

Case 7.—*Enchondroma of the inferior articular end of the femur; amputation five months after its discovery; cured.* (Drawing 8^{50}.)

A married woman, æt 23, was admitted into Guy's Hospital, in July, 1848, under the care of the late Mr. Aston Key. She seemed to be healthy, although she looked very delicate. This appearance might be accounted for, as she was poor, lived in Southwark, and had had two or three children quickly one after the other, which she suckled. She had always enjoyed good health.

About four months before admission she noticed that her left knee-joint became enlarged. This was attended with considerable pain, and at last the limb became entirely useless. The inguinal lymphatic glands were not affected. Her general health, however, had not suffered materially, and, as the cure of the disease seemed hopeless, Mr. Key performed amputation through the femur, rather above the junction of the lower with the middle third of that bone. The stump healed favorably, without any untoward symptoms, except that a sinus remained open when she left the hospital, about two months after the operation.

The drawing in the museum represents a section of the knee-joint made through the patella from before to the popliteal space behind. The head of the tibia, the part of the femur affected by the new growth, and a portion of the shaft of that bone, as well as a part of the patella, are seen. The only bone-tissue involved in the disease was the inferior articular extremity of the femur. Both the condyles of that bone seemed at first to have been expanded by a growth of cartilaginous tissue, which at one part was still confined by the original articular cartilage covering the end of the bone. The cut edge of this cartilage is well shown in the drawing, forming a crescentic white outline. At the anterior and posterior horn of this crescent the new growth has burst the confines of the condyles, and in front it has extended forwards and upwards between the femur and the extensor muscles and tendon, which are stretched over it. Behind, the growth pushed into the popliteal space, and downwards a process grew into the knee-joint, where it was lying between the patella and articular surfaces of the tibia.

The whole of the new growth consisted of cartilage-tissue, showing a very pretty foliated outline and its characteristic hyaline appearance. It was somewhat granular in texture, with here and there bony spiculæ or granules. It was invested generally by a well-defined envelope, which in some parts of its circumference was very strong. The inter-articular ligaments seem to have restrained the progress of the growth in some parts. The shaft of the femur was remarkably healthy, the texture being very hard and compact for the bone of a female. In close proximity to the cartilage growth, and for about an inch and a half above it, some new bony tissue was developed, which, like new bone in general, was rather spongy.

From the description above given it is quite clear that the disease was originally developed within the condyles of the femur, the cancellated tissue of which was, so far as visual observation extended, entirely destroyed by its invasion. It is a point of some interest to connect the development of the cartilage growth with changes which were probably taking place at the point of union of the condyles with the shaft of the bone, for, most certainly, the limit of the growth of cartilage did not exceed that of the line of the epiphyses. I was unable to obtain any history of an injury of the part, and the opposite limb was perfectly healthy, so that there was not any symmetrical action excited.

When, after careful examination of the tumour, it was pronounced to be cartilage, and the elementary tissues of cancer were not found anywhere, Mr. Key still doubted the innocence of the growth, and seemed to be fully prepared for its recurrence in the stump.

Some two or three months after leaving the hospital this patient returned as an out-patient under my observation with the stump enlarged, inflamed, and very painful. Her general health also seemed much affected. I well remember Mr. Key's expression when I showed him his old patient—"Yes, I thought it would be so." At that time she was not again admitted, but she continued from time to time to come to the hospital, the stump still inflamed and giving her much suffering. It became, however, quite clear before the summer of 1849 that the condition of the stump was entirely dependent upon some disease of an inflammatory nature, for suppuration took place, and as the induration of the soft parts subsided the enlarged end of the stump of the femur could be felt. Mr. Key died in the autumn of 1849. I kept sight of this patient, but could not persuade her to come into the hospital to have the dead bone removed from the stump until May, 1851. At that time her health and strength were very much reduced by many causes, but it soon became re-established after the stump had been opened and the source of irritation entirely removed.

I have frequently seen this woman since that time. The stump has continued perfectly well, and the general health has been as good as it usually was before the amputation. I saw her last year, in December, 1865, seventeen years since the operation, when she was quite well.

Tibia and Fibula.—I place these bones together because they are sometimes associated with cartilaginous tumours of the leg.

CASE 8.—*Enchondroma growing from the tibia; amputation; recovery.* (Models 52^{40}, 52^{41}, prep. 1336^{10}.)

The following case was under the care of Mr. Poland. A healthy man, æt. 32, had observed a swelling in the popliteal space for some months. The tumour had slowly increased without causing much pain until lately. It was very hard and lobulated. Mr. Poland amputated the limb through the middle of the femur.

The models and preparation show a most beautiful example of a growth of cartilage from the posterior surface of the tibia. The characteristic outline of these growths is well displayed in the nodulated masses composing the whole. A section shows how the growth had sprung from the surface of the original bone, a portion of the compact texture of which still remains. The medullary cavity and cancellated head of the bone are not involved in the disease, nor, apparently, in any way affected. The cartilage growth may be seen running round between the head of the fibula and tuberosity of the tibia. The knee-joint was quite healthy. Now, if this tumour had been seated upon a more superficial surface of the bone, and the patient had applied early for advice, I believe the removal of the cartilaginous growth might have been effectually removed. The possibility of this simple attachment of the new growth to the bone surface only should therefore be remembered.

Case 9.—*Large enchondroma of the leg, of many years' growth; amputation; recovery from the operation; death subsequently from disease of lungs.* (Drawing 33^{37}, prep. 1336^{90}.)

A patient was under my care in Martha Ward in March, 1866. She was a healthy looking person, æt. 51, the mother of several children, and had been a good deal distressed by social vicissitudes. For many years she had been troubled with a hard swelling of the right leg, which commenced about fifteen years since in what she termed a rheumatic attack. Of late the tumour had caused excessive pain, so that she passed sleepless nights and lost her appetite. Besides, the limb had become almost useless from the weight of the growth. The limb from the foot upwards to about the upper third of the leg was very much enlarged, and the healthy looking integuments raised into great bosses by a very lobulated mass beneath. The growth projected most distinctly at the sides. The tibia was distinguishable in front. In some parts the growth was very hard, in others soft, and here and there the sensation of fluctuation was perceptible. From the length of time the tumour had been growing, the healthy appearance of the patient, and the palpable indications, the diagnosis of the disease was clearly that it was a growth of cartilage. The patient being told that amputation was the only means which could relieve her, she consented to the operation, which I performed below the knee-joint. The section of the bones and soft parts was quite healthy. She recovered from the operation, the stump healed, and she left the hospital about six weeks after her admission.

The tumour is a magnificent specimen of a cartilaginous growth. It appears to grow from the posterior surface of the tibia, near the ankle-joint, the texture of that bone being quite healthy. It forms a mass of several pounds' weight, and consists of lobes and lobules of cartilage, in a most healthy growing state. A vertical section was made through the tibia and foot, which should be examined to understand the relations and beauty of the textures of these growths. One fact is very remarkable. The cartilage growth has made its way completely through the os calcis into the sole of the foot, among the muscles there placed. One might suppose that the os calcis was the centre or nucleus of the tumour at its origin; but as far as the statement of the patient may be considered as a guide, I am of opinion that it commenced at the lower end of the tibia, and grew between the bones and the posterior muscles upwards and downwards.

The condition of the patient, as regards her general health, was not quite satisfactory when she left the hospital. She complained of a cough, which occurred in troublesome paroxysms; the mucus was sometimes slightly tinged of a brownish tint; occasionally this colour was certainly produced by the admixture of blood. She passed sleepless nights, lost her appetite; the pulse was feeble and powerless; and in spite of every attention from her family, and her medical attendant Dr. Henderson, and other friends, she gradually died exhausted in August of the same year. Unfortunately, it was not possible to make an after-death examination; but so far as the indications of the nature of the disease from which she died were manifested during life, it appeared that the whole of the pulmonary mucous membrane was affected by some chronic inflammatory action. The pulmonary tissue did not break up, nor were there signs of any tumour compressing the lungs.

The foot and toes.—The bones of these regions are not so often affected with cartilaginous growths as those of the hands and fingers. There is in the museum a drawing (24^{88}) and a preparation (1285^{85}) of a cartilaginous growth between the great toe and next, removed by Mr. Hilton; and one on the little toe (drawing 24^{89}), removed by Mr. Bryant.

The length to which the description of the cartilaginous growths has extended precludes any account of the bony ones in this paper. That will follow in the next volume.

DESCRIPTION OF PLATES

Illustrating Mr. Birkett's Paper on Enchondroma.

PLATE I

Illustrates Case 4, and shows the comparative size of the growth, and the ordinary position of the patient shortly before death.

PLATE II

Illustrates Case 1, and shows the general appearance of the chest, and the comparative size of the tumour after about nine years' growth.

Plate 2.

W. Hurst ad nat lith

M & N Hanhart imp.

A CASE

OF

ABDOMINAL TUMOUR.

By S. O. HABERSHON, M.D.

Cancerous disease of both supra-renal capsules, simulating disease of the spleen and lumbar glands.

(Reported by Mr. G. B. STEVENS.)

CASE.—Elizabeth B—, æt. 38, was admitted into the clinical ward, under Dr. Habershon's care, on May 2nd, 1866. She was a spare, emaciated woman, with a sallow and careworn expression of countenance. The forehead and the upper parts of her neck, and also her hands, were partially bronzed from exposure, the discoloration resembling that arising from the action of the sun's rays, and having defined borders. Her life seemed to have been an anxious one, and for eight years her health had been impaired. She had been married twice, and had five children by her second husband; four of these children died immediately or soon after birth. She described them as "looking old and wrinkled," and said that they "died from wasting," but there was no eruption upon the skin. So far as could be ascertained, they were the subjects of congenital syphilis. She complained of a dry winter cough, but no history of hereditary tendency to disease could be made out. There was some doubt as to a previous attack of ague. She was brought to the hospital in an enfeebled condition. The chest was very spare and small, but the sounds on percussion and auscultation were normal and gave no evidence of disease. The action of the heart was feeble, and the pulse was very compressible, 88 per minute. On examination the abdomen was found to be generally wasted, but the left side, and especially the left hypochondriac region, was rather larger than natural, and the lower ribs appeared to bulge. In the left hypochondriac region there was complete dulness, and this dulness extended above nearly to the median line, and below nearly to the crest of the ilium. At the upper part a defined border could be made out, nearly in the position of the left lobe of the liver, but it extended into the splenic region. At the lower part of the tumour, midway between the ribs and the crest of the ilium, and towards the median line, there was resonance on percussion and gurgling, evidently due to the presence of intestine, and beneath this portion, still lower, another growth could be felt, rounded in form and nodular. There was considerable tenderness on pressure at the upper part of the tumour. The left loin was enlarged; the tongue was clean; there was no vomiting nor pain after food, but the appetite was almost lost. The bowels acted regularly. The urine was of normal quantity and colour, of sp. gr. 1022, and free from albumen. The patient could give no account of the abdominal swelling. Her residence having formerly been in an aguish district, and sensations of heat and chilliness being experienced, quinine was ordered. The distress was, however, increased by this medicine; it was therefore discontinued. On the 10th May she had severe pain in the left hypochondriac region, and had no sleep at night; gr. iij of soap-and-opium pill were ordered to be taken at bed-time, and gr. iij of bromide of potassium three times a day. On the 15th she had been unable to sleep; the quantity of opium was therefore increased to gr. j every night; still, although she took nourishment, wine, &c., the symptoms of exhaustion increased, and the pain became persistent.

On the 20th there was more general fulness on the left side of the abdomen, and a good deal of flatus in the intestine. For two days she had suffered intense pain at the epigastrium, with vomiting. She was ordered bismuth, with carbonate of soda and morphia. Severe pain now came on in the right lumbar region, and a fulness was perceptible at that part; it was apparent that there was also enlargement there. On the 27th she was much worse; the nights were passed without sleep, and she was frequently found in a profuse perspiration. The vomiting was relieved by the bismuth; but on account of the continued restlessness and want of sleep, the opiate was given in small doses several times during the day, and was continued for several days; it was then omitted, because of drowsiness. On June 1st there was some œdema of the ankles and loss of power of the legs, especially on the left side. Her strength continued to fail, but still she complained of severe pain, and the perspirations were undiminished. On the 9th spots of purpura were observed on the abdomen, and some of the glands at the angle of the jaw were enlarged. The abdominal tumours were more distinct. Scarcely any nourishment was taken, but stimulants were administered freely. The œdema of the lower extremities increased, and there was constant restlessness. On the 12th the pulse could scarcely be felt, and the hands and feet were cold; but for several days she lingered, the mind occasionally wandering, until the 15th, when she expired.

A post-mortem examination was made by Dr. Moxon, whose report is subjoined:

The brain was anæmic, but firm and natural. The pleuræ were healthy, and there were no adhesions. The lungs were very anæmic, and together only weighed 14 oz.; their texture was clear, their surfaces dotted with rings of black pigment, surrounding single emphysematous air-cells. There were no relics of tubercles at the apices.

The heart and large vessels were small, weighing only 6 oz.; the right ventricle was very thin.

Abdomen.—The viscera were raised and pushed forward by two large tumours, which were lying on either side of the spine, and were connected centrally by other masses, consisting, for the most part, of enlarged lumbar glands. The liver was flattened; and its under part presented a patch of cancerous matter, where it was in contact with the right tumour. After removing the diseased masses, and making a vertical section, they were found to consist of large cancerous tumours affecting the supra-renal capsules and extending into the kidneys. The supra-renal capsules were four or five times larger than ordinary adult kidneys. The masses had the appearance of soft or encephaloid cancer. The kidneys could be separated. The right kidney touched the capsule to a moderate extent; the left imbedded itself in the mass above. Both kidneys presented masses of cancerous deposit, apparently beginning in the cortical part, and there seemed very little healthy renal structure left. The liver had undergone partial fatty change. Both ovaries contained cancerous deposit. The spleen was small and perfectly healthy, so also were the stomach and the intestines. The peritoneum also was free from disease.

This remarkable instance of disease consisted of cancerous infiltration of both supra-renal capsules; for although the ovaries were also implicated, the enormous size of the supra-renal tumours led to the belief, that they were primarily affected. The lumbar glands were secondarily involved, and, by direct extension of disease, the contiguous surfaces of the kidney and liver. The health of the patient gradually failed, and her exhaustion increased from the implication of the centres of the vaso-motor nerve. The stomach was partially pressed upon, and this fact served to explain the occasional vomiting, which was also due, in some measure, to sympathetic irritation.

Nothing could be made out as to the *cause* of the disease. The patient had probably become poisoned with syphilis, but there was no hereditary tendency to cancer. Neither could the duration be ascertained; for although the health had been failing for eight years, she had no knowledge of the abdominal tumours till they were detected after her admission into the hospital. The symptoms were emaciation, a fixed pain in the region of the growths, irregular perspirations, complete loss of appetite, occasional vomiting, restlessness, want of sleep, extreme prostration; death being preceded by slight delirium. The physical signs were sallowness of the countenance and abdominal tumours in both lumbar regions and in the left hypochondrium, with bulging of the lower ribs on the left side.

Emaciation and loss of strength were the first symptoms, and were steadily progressive. On examination after death no direct cause of these symptoms could be ascertained; there was no evidence of pressure upon the thoracic duct, and we were led to refer them to general impairment of nutrition, such as is frequently observed in cancerous cachexia.

The pain was severe and constant, especially on the left side. The suffering was due to the great tension of the affected parts rather than to serous inflammation.

The irregular perspirations, sometimes attended with chilliness, and the doubt whether the patient had had ague, led, at first, to the supposition of miasmatic poisoning and splenic disease; but, in all probability, these symptoms were the expressions of general exhaustion, as in hectic. The loss of appetite was a form of functional inactivity from failing power, and the vomiting was probably due to direct pressure on the stomach, as we have just remarked.

The absence of sleep was greater than could be explained by the pain; and although the latter symptom was severe, it was mitigated by opium; at length the restlessness which often precedes the close of life became the predominant condition.

As to physical signs, the discoloration of the skin was remarked during life; but it was not that which has been so often observed in connection with disease of the supra-renal capsules (Addison's disease, with bronzing of the skin). The brownish discoloration was only on the exposed parts of the face and hands; it had a defined border, and was referred to the effects of the sun (*ephelis solaris*). Besides this partial bronzing there was the sallowness of countenance often observed in cancerous disease; but it would, we think, be incorrect to attribute this state of the skin to any disease of the supra-renal capsules, for the remarkable cases of discoloration of the skin have been in connection with strumous degeneration of the capsules or with chronic contraction. Numerous instances have occurred of cancerous infiltration of the supra-renal capsules without any discoloration.

The next symptom to which we have to refer is the character and position of the abdominal tumour. The mere *position of any tumour* is very apt to mislead us in diagnosis, and it should always be associated, as a sign, with *indications of functional disturbance.* Thus, a tumour in the region of the liver or of the stomach is of more certain diagnostic value when the functions of those parts are interfered with. In this instance the tumour was in the left hypochondrium, and there were variations of temperature, and doubt as to miasmatic poisoning, which led us to refer the disease in part to the spleen.

The bulging of the ribs, however, was much greater than we ever find in miasmatic enlargement of the spleen. The resonance on the left side, between the two portions of the tumour, was an indication that the colon passed in front and to the left of it, and indicated glandular or renal enlargement; and when deep-seated pain and fulness on the right side came on, the lumbar glands on both sides were believed to be affected.

The negative signs were remarkable; the urine was healthy, although the kidneys were encroached upon; there was no evidence of any primary affection of the stomach, or liver, or intestines. And although we were correct in believing the lumbar glands to be affected, they formed but a small part of the abdominal tumour.

We would especially remark upon the importance of ascertaining whether the functional integrity of the part is involved. But, when we are called upon to decide between disease of the lumbar glands and of the supra-renal capsules, these indications entirely fail us. We may expect more help in diagnosis when the functions of the supra-renal capsules are revealed by advancing science.

DESCRIPTION OF PLATE

Illustrating Dr. Habershon's case of Disease of the Supra-renal Capsules.

The plate shows the relative positions and comparative sizes of both supra-renal capsules and both kidneys.

a. Section of the supra-renal capsules.

b. Section of the kidneys, partially encroached upon.

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ON THE

URINE IN ACUTE RHEUMATISM.

BY THOMAS STEVENSON, M.D.

As but few quantitative chemical examinations of the urine of persons suffering from acute rheumatism appear to have been made, the following analyses of urine passed by patients in the wards of Guy's Hospital during the spring of this year may not be devoid of interest. These analyses were undertaken at the suggestion of Dr. H. G. Sutton, with whom I had frequently discussed the questions arising as to the composition of the urine in rheumatic cases. He was anxious that I should, if possible, discover if any data for prognosis could be obtained from the chemical constitution of this excretion whilst the disease was at its height. Circumstances prevented our carrying out the investigation jointly; but I have to thank him for a careful selection of the cases, and for having the different specimens of urine collected of which the analyses are here given,

The few recorded analyses of the urine in acute rheumatism, with reference to the quantities of its most important ingredients, may be shortly summarised. Vogel[1], who places the daily excretion of phosphoric acid in the adult male at 54 grains, a greater quantity than that obtained by other experimenters, found that in three cases of rheumatism affecting females 32¼, 34, and 35 grains respectively were excreted. These figures show, if the probably small weight of these

[1] Neubauer and Vogel, 'On the Urine,' New Sydenham Society's translation, p. 414.

female patients be taken into account, that there is a slight increase in the amount of phosphoric acid excreted. Vogel also determined that the mean daily excretion of urea in three men suffering from this disease was 602 grains, a quantity much above the normal physiological amount, especially when we consider how small a quantity of food is consumed during the acute stage. Dr. Parkes[1] states that he has observed from one fourth to one fifth more than the physiological amount of urea to be excreted; he also states that the uric acid is increased, for in two cases the average daily secretion of this substance was $9\frac{1}{2}$ grains. Moreover, according to the same observer, there is a very large increase in the excretion of sulphuric acid, the amount sometimes reaching double that passed during convalescence. In one case it reached $52\frac{2}{3}$ grains, afterwards sinking during convalescence to 24 grains, whilst in five cases the increase was constant so long as the joint affection lasted. Dr. Parkes also found an increase of sulphur—containing extractives; but he states that the excretion of phosphoric acid is probably not much influenced; that the water, as common observation shows, is diminished; and, finally, that the free acidity is excessive, that the total quantity of solids is above the normal amount, and that the extractives and pigment are increased. Dr. Beale also states that the excretion of urea is in this disease very much above the normal quantity. Beyond these, very few accurate analyses bearing on the question appear to have been recorded.

In this investigation my attention was chiefly directed to the urea, the uric acid, the phosphoric acid, and the sulphuric acid, and these substances were alone determined quantitatively. The analyses were made of the whole secretion collected during twenty-four hours, when the disease was at its height. The urine was examined in a similar manner in three cases, when the patient was convalescent. The results were then compared with each other. In every instance the urine, when first passed, was acid; when submitted to analysis it had, however, sometimes become alkaline; but it must not be supposed that decomposition had occurred to such an extent as to invalidate the results. Strongly acid urine, passed by rheumatic patients, will often become alkaline in a very short time, although no

[1] Parkes, 'On the Urine,' pp. 286—290.

change is visible in the appearance of the urine, and without any appreciable loss of urea. In all cases the urine was employed when apparently unchanged in every respect excepting its reaction to litmus paper.

The amount of urea was ascertained by Liebig's well-known process; the chlorides were not previously determined, but the usual allowance was made for them. The uric acid was weighed after precipitation by hydrochloric acid, every precaution being taken to secure accuracy in the latter rather delicate operation. The phosphoric and sulphuric acids were determined gravimetrically, the former being weighed as pyrophosphate of magnesium after precipitation as triple phosphate, the latter as sulphate of barium. In nearly every case the first analysis was checked by a second determination, and, when necessary, a third was made.

Inasmuch as the diet of the patients was in all instances uniform, the variations in the quantities of the various substances excreted cannot be in any way attributed to its influence. During the acute stage the daily consumption of food consisted in a pint of beef-tea, half a pint of arrowroot made with milk, and a pint of milk. When convalescence commenced this diet was changed for one consisting of fish (a sole) at dinner, 10 oz. of bread, $\frac{3}{4}$ oz. of butter, and a pint of milk. Afterwards middle diet was given, consisting of 4 oz. of dressed meat, $\frac{1}{2}$ lb. of potatoes, 12 oz. of bread, 1 oz. of butter, 1 pint of beer, tea and sugar, and either 8 oz. of rice pudding or a pint of broth, alternately. It was when the patients were taking this last diet, and when convalescence had been fully established, that the urine was analysed for the second time.

Each case is thus arranged:—After a very brief history of the case the daily excretion, in grains, of urea, uric acid, phosphoric acid, and sulphuric acid, during the acute stage, is stated, and by the side of these quantities are given the corresponding quantities excreted during convalescence whenever they were ascertained. When the weight of the patient *during health* was known a table is given which exhibits the proportionate quantities of the above substances excreted for each pound weight of his body. Lastly, I have drawn certain inferences from a general survey of the observations made.

I now proceed to give the details of the cases.

Case 1.—Thomas Neale, æt. 24, a bricklayer, under the care of Dr. Barlow. It was his third attack of the disease. He was rather a short, thick-set man, weighing 9 st. 11 lb. The heart was affected. He took no active medicine. The urine was analysed on the thirteenth day of the attack, during the height of the disorder, and again twelve days later, when convalescence had been established for nine days. The urine collected during the acute stage measured 31 fluid ounces in the twenty-four hours, its sp. gr. was 1·0289, and it speedily became alkaline in reaction. The urine during the convalescent stage measured 36 fluid ounces; it was clear, acid, and of normal colour, its sp. gr. was 1·0249.

Daily excretion, in grains.

	Acute stage.	During convalescence.
Urea	528·03	447·30
Uric acid	11·07	very little
Phosphoric acid	32·81	27·76
Sulphuric acid	45·20	37·74

When these amounts are calculated, so as to exhibit the quantities of the various substances excreted for every pound of the usual weight of the patient during health, we obtain—

Daily excretion, in grains, per lb. weight of body.

	Acute stage.	During convalescence
Urea	3·861	3·265
Uric acid	·081	—
Phosphoric acid	·239	·203
Sulphuric acid	·330	·275

It may be interesting to compare these tables with analyses made by me of the urine of a healthy man under different diets, which varied from those containing a moderate allowance of animal food to those composed nearly exclusively of vegetables. The weight of the individual was nine stone. Very little exercise was taken. The urea was determined on thirteen different days, and the phosphoric acid on seven different days. On the majority of these occasions an animal diet had been taken.

Amount, in grains, excreted daily by a healthy man.

	Maximum.	Minimum.	Average.
Urea	479·95	377·94	422·85
Phosphoric acid	35·50	24·54	31·24

Amount, in grains, excreted daily by a healthy man, per lb. weight of body.

	Maximum.	Minimum.	Average.
Urea	3·809	3·000	3·356
Phosphoric acid	·282	·195	·248

These quantities do not differ materially from those obtained by previous observers. I have myself made only one observation as to the excretion of sulphuric acid in the urine during health, but Dr. Parkes places it at ·214 grain per lb. weight of the body, or 31 grains daily in an adult of medium size. In the instance observed by me it equalled ·321 grain per lb. weight of the body, or 44·25 grains daily.

Case 2.—James P—, æt. 21, under Dr. Barlow. He was a delicate-looking, sallow man, of temperate habits, a carpet-beater by trade. This was his second attack, and he had had very bad health since his first, which occurred when he was twelve years old. His weight in health was 10 st. 11 lb. The heart was affected. He was treated with frequent drachm doses of bicarbonate of potash, made effervescent by means of citric acid. The urine passed in the twenty-four hours, when analysed thirteen days from the commencement of the attack, measured 34 fluid ounces. It speedily became alkaline in reaction, its sp. gr. was 1·0199. When examined twelve days later, and seven days from the commencement of convalescence, 52 fluid onnces were collected in the twenty-four hours; its sp. gr. was 1·0135, and it was normal in appearance.

Daily excretion, in grains.

	Acute stage.	During convalescence.
Urea	345·14	404·95
Uric acid	8·16	almost nil
Phosphoric acid	32·51	19·88
Sulphuric acid	31·19	20·37

Calculating, as before, the quantities of these several ingredients excreted for each pound weight of the healthy body, we have—

Daily excretion, in grains, per lb. weight of body.

	Acute stage.	During convalescence.
Urea	2·286	2·682
Uric acid	·054	—
Phosphoric acid	·215	·132
Sulphuric acid	·206	·135

Case 3.—James S—, æt. 15, a shoe-black. This youth was spare and muscular, and was suffering from his first attack of rheumatism; there was heart complication. He was treated by Dr. Barlow with lemon-juice. On the twenty-first day of the attack the urine was examined. The quantity passed in twenty-four hours was 21 fluid ounces, having its sp. gr. 1·0215. It was again analysed fourteen days later, and nine days after he had begun to convalesce; 34 fluid ounces were then passed in the twenty-four hours. At this time the urine was normal in appearance, and its sp. gr. was 1·0185.

Tabulating the results, we find—

Daily excretion, in grains.

	Acute stage.	During convalescence
Urea	211·26	252·10
Uric acid	7·71	1·79
Phosphoric acid	11·64	9·02
Sulphuric acid	15·74	22·65

In the remaining cases the urine was examined during the acute stage of the disease only.

Case 4.—George H—, æt. 24, a labourer, under Dr. Rees. Height 5 feet 8 inches, weight 11 st. 7 lb. in health. Though of intemperate habits, he had usually been healthy, with the exception of a first attack of the disease, nine years ago. The heart was affected. He was treated with lemon-juice.

The urine was analysed thirteen days after the onset of the disease. It was turbid with lithates, rather high coloured, and very acid; its sp. gr. was 1·0285; 37 fluid ounces were passed in twenty-four hours. It afforded the following results:

Daily excretion, in grains (acute stage).

Urea	691·95
Uric acid	9·83
Phosphoric acid	47·76
Sulphuric acid	50·21

Daily excretion, in grains, per lb. weight of the healthy body.

Urea	4·298
Uric acid	·061
Phosphoric acid	·297
Sulphuric acid	·312

Case 5.—George C—, æt. 21, an intemperate man, a costermonger, under Dr. Barlow. This was the second attack, the first having occurred two years ago; otherwise his health had been good. There was heart complication. He was treated with lemon-juice. The urine analysed was collected on the fourteenth day of the attack. It measured 32 fluid ounces, its sp. gr. was 1·0227; it was clear, acid, and of a normal colour. The following results were obtained:

Daily excretion, in grains (acute stage).

Urea	322·36
Uric acid	4·99
Phosphoric acid	18·66
Sulphuric acid	44·68

Case 6.—John C—, æt. 14, a spare but muscular youth; by trade a shoe-black. He had always been delicate, had suffered for the last four or five years from palpitation of the heart, and had been liable to syncope on exertion, but there was no history of a previous attack of rheumatism. The heart was affected. He was treated by Dr. Gull without medicine.

The urine, when analysed on the twenty-sixth day of the disease, measured 20 fluid ounces, its sp. gr. was 1·0289; it was acid, high-coloured, and very turbid with urates. It afforded the following results:

Daily excretion, in grains (acute stage).

Urea	362·65
Uric acid	9·13
Phosphoric acid	20·92
Sulphuric acid	29·20

Case 7.—Eliza B—, æt. 16, a well-formed young woman, under Dr. Rees. It was the first attack, the heart was unaffected, and she speedily recovered, having taken lemon-juice.

The urine, collected on the sixth day of the disease, measured 26 fluid ounces, of sp. gr. 1·025. It was clear, and not very high coloured. It gave the following results:

Daily excretion, in grains (acute stage).

Urea	386·04
Uric acid	2·21
Phosphoric acid	24·87
Sulphuric acid	34·43

Besides these, I have analysed the urine of another patient, a woman, in the acute stage of the disease and during convalescence; but as this secretion, in consequence of retention, had to be drawn off by the catheter, it was doubtful whether the amount of urine passed was correctly estimated. I therefore forbear quoting the results.

In reviewing these analyses the first and most striking fact is the extreme variability in the results, so that it would almost seem, on a superficial glance, that no general conclusions can be deduced from them. But this is not to me a cause of much regret, inasmuch as my object in conducting these researches was rather to test the conclusions of previous writers on the subject than to establish any general theory; indeed, I should strongly object to any sweeping generalities being deduced from such a small number of instances as are here given. Nevertheless, a few observations upon my analyses may, I think, be fairly made.

First, then, we observe that in the acute stage the excretion of urea, which is by far the largest channel for the elimination of nitrogen from the body, shows extreme variations in its quantity. The physiological amount was rarely very much exceeded, and in only two of the instances (Cases 1 and 4) did this substance appear in very great excess. These two patients did extremely well, and speedily recovered. If they had been actively treated these cases would, no doubt, have been spoken of as showing the remarkably beneficial effects of the treatment adopted; but as the one patient took lemon-juice (Case 4) and the other (Case 1) had no medicine whatever, no theory as to the effects of medicine is supported by them. In striking contrast with these are Cases 2 and 3, in which the quantities of urea excreted were actually less during the acute stage than during convalescence. These, more especially No. 3, were stubborn, lingering cases. It will be said by the advocates of eliminative treatment that the process of secretion required assistance; but to this it may be rejoined that in Case No. 2 the patient was long kept under the influence of large doses of alkali, and that when this remedy was withdrawn he began to secrete more, and to make better progress; whilst in No. 3 the patient, whose case was still more tedious, took lemon-juice as a medicine.

When we consider that gelatine, the most highly nitrogenized of all the constituents of food, is contained in large quantity in beef-tea, which formed a considerable part of the uniform diet of all the patients during the height of the disease, and when we further take into account the well-ascertained fact that gelatine has a very great influence over the secretion of urea,[1] these results are certainly very remarkable. I am inclined to believe that when there is, independently of treatment, an abundant secretion, the patient is likely to do well, and *vice versâ*, but that when secretion is scanty it is almost useless to attempt increasing it by the administration of large doses of alkalies.

[1] Dr. Buchanan has found a variation of 174 grains in the daily excretion of urea under the administration of strong beef-tea. Reynolds's 'Syst. of Med.,' vol. i, p. 542.

In the remaining cases the excretion of urea did not exceed the physiological amount. But it must be remembered that the patients were at rest, and were taking a scanty diet, and that, therefore, the excretion was, no doubt, really greater than it would have been under parallel circumstances if no disease existed. The progress of these cases presented nothing of interest, except that the young woman, aged 16 (Case 7), who passed the largest amount of urea—386 grains daily, a full amount for a girl of that age—made the most rapid and satisfactory recovery of all the patients whose cases are here recorded.

The excretion of uric acid during the acute stage was likewise extremely variable, rising in one instance (Case 1) to 11·07 grains, whilst in another instance (Case 7) only 2·21 grains were excreted; and between these limits the quantities secreted were very diverse. There appeared to be no relation between the amount of uric acid excreted and the favorable or unfavorable course of the disorder, for both these patients did remarkably well, whilst the patient No. 2 and the patient No. 4, who excreted nearly the same quantities of this substance (8·16 and 9·83 grains respectively), made, the former a very tedious, the latter a very rapid, progress towards recovery. It is impossible to fix limits to the physiological amount of uric acid contained in the urine; but it may be seen that the amounts excreted in these cases during the height of the disease were far in excess of those excreted during convalescence.

In Cases 1 and 2 the excretion of this substance fell, in the passage from disease to health, from 11·07 grains and from 8·16 grains respectively to a quantity almost too small to be estimated; and in Case 3, it sank from 7·71 grains to 1·79 grain. None of these three specimens of urine let fall much of this acid on standing, though several of them deposited urates. This may have been due, in the first two cases, to one of two causes—either the acid existed in combination with bases, or the speedily alkaline state of the urine, from decomposition of urea, held the acid in solution. I am inclined to think that the latter is the more probable explanation.

Very wide variations were exhibited in the amount of phosphoric acid excreted. It fell to 11·64 grains in the most chronic case, that of the youth of fifteen (Case 3), and rose to 47·76 grains in one of the most rapid cases (Case 4). In all those instances in which its amount was determined both in the acute stage and during convalescence, it was greater in the acute stage; but with the exception of the above-mentioned case, in which the large amount was excreted, it generally did not vary much from the normal physiological amount, which may be stated as 32 grains daily.

The sulphuric acid excreted during the height of the disease exhibited a more constant excess than any ingredient of the urine except uric acid. In all three cases in which it was examined during both stages of the disorder there was a marked excess in that passed in the acute stage as compared with that excreted in convalescence. Generally, however, the normal physiological amount was not exceeded, though, when we take into consideration that probably only a small quantity of sulphur was taken in the food during the acute stage of the disease, the excretion must be accounted excessive. I may add that the same rule appeared to hold good here as with the nitrogenous constituents of the urine—good excretion, good progress.

Finally, the bulk of the urine excreted was invariably diminished, and the density of the excretion was thereby increased. It has been too generally assumed that, because the urine in this disease is of high specific gravity and contains an increased *per-centage* of solids, there must necessarily be an increased elimination of effete material through the kidneys; but if we make due allowance for the increase of density due to the diminished quantity of water present, this will be by no means so apparent. If the specific gravity of the urine in the seven cases during the acute stage be tabulated, and also the amount of fluid secreted daily, we find—

	No. 1.	No. 2.	No. 3.	No. 4.	No. 5.	No. 6.	No. 7.
Sp. gr.	1·0289	1·0199	1·0215	1·0285	1·0227	1·0289	1·0250
Fluid oz.	31	34	21	37	32	20	26

But if the urine were diluted with water, so that the same quantity of solid material should be distributed through 40 fluid ounces (which may be taken as the average amount of urine daily passed by an adult), its density would, in the majority of instances, have been less than that which is normal.

	No. 1.	No. 2.	No. 3.	No. 4.	No. 5.	No. 6.	No. 7.
Sp. gr.	1·0224	1·0169	1·0113	1·0264	1·0192	1·0145	1·0163

These results are quite consistent with those drawn from the chemical analyses. It is true that there is usually a much larger excretion of solids during the acute than during the convalescent stage; but the quantity of urea excreted during the former stage rarely exceeds the quantity that would be secreted by an adult in health, and *doing work*, though it, no doubt, sometimes exceeds that excreted by a healthy adult *at rest;* and it must not be forgotten that these patients were at rest so far as work is concerned.

It appears probable that where a high density is observed after diluting the urine so as to make it up to the normal healthy bulk the patient is likely to make good progress. I have not as yet been able to work out this point fully, but if it should prove to be true it will be a valuable aid to our means of prognosis. I should add that this is in entire accordance with my other conclusion—that where there is a large excretion of solids the prognosis is favorable so far as the rheumatism is concerned.

By way of appendix to these analyses of rheumatic urine I will add that of the urine in a case of acute rheumatic gout, made in order to compare the amount of excretion in the two diseases. This case, that of a woman under Dr. Rees' care, was long and tedious; it will be observed that, though the bulk of the excretion was great, the amount of the various solids was small, both in the aggregate and separately.

The urine passed in twenty-four hours measured 51 fluid ounces; its sp. gr. was 1·0094; it was normal in colour, clear, and alkaline in reaction.

Daily excretion, in grains, in a case of rheumatic gout.

Urea	222·40
Uric acid	·36
Phosphoric acid	17·72
Sulphuric acid	12·84

Conclusions.

1. In acute rheumatism, when the excretion of solid materials in the urine is large, the patient makes, other things being equal, a rapid recovery; on the other hand, in lingering cases the excretion of solids is usually small.

2. As in this disease the urine is invariably scanty in bulk, but (generally from this cause only) of high density, a useful guide to the progress of the case may probably be found by diluting the urine to the normal bulk, and then ascertaining its specific gravity. According as it is now of high or of low density will the progress of the disease probably be favorable or unfavorable.

3. Though the excretion of urea is usually greater during the height of the disease than during convalescence, this is not invariably the case; the reverse sometimes occurs. Though the excretion of urea is greater during the disease than during the early stage of recovery, the urea in the former stage seldom very much exceeds in amount the normal physiological excretion.

5. The uric acid is always much increased whilst the disease continues.

5. The phosphoric acid is generally in greater amount during the progress of the disease than during recovery, but the quantity of this substance rarely much exceeds the quantity secreted in health.

6. The excretion of sulphuric acid is generally increased, and often largely. In one instance more was excreted during recovery than during the acute stage of the disease. The amount of this substance excreted is very variable.

CASE

OF

EXCISION OF THE SPLEEN

FOR

AN ENLARGEMENT OF THE ORGAN, ATTENDED WITH LEUCOCYTHÆMIA.

IN A PATIENT UNDER THE CARE OF DR. WILKS;

WITH REMARKS.

By THOMAS BRYANT.

(Reported by Mr. R. Eager, Clinical Clerk.)

Charles P—, œt. 20, a groom, was admitted into John Ward May 30th, 1866, under the care of Dr. Wilks. He states that from a child he has had remarkably good health, and has never suffered from any illness. His parents are alive and healthy. He was born near to Northampton in a marshy district, but has never suffered from ague. He had lived there all his life until a week ago, when he went to reside at Sunbury with a gentleman, as his groom. He has led a very regular and temperate life. About five or six months ago he noticed a tightness across the upper part of his abdomen, and found that he was increasing in size, and that, especially on the left side, there was a hard mass. From this time the abdomen and the hard mass in the side slowly enlarged, the tumour keeping up close to the ribs and xiphoid cartilage, and not reaching (as now) nearly to the pubes. Within the last few weeks it has left its first situation close to the xiphoid cartilage and to the margins of the cartilaginous portions of the seventh and eighth ribs, and has dropped down into the abdomen. He has been treated at his native place, and appears to have taken large quantities of different medicines, especially quinine; he has also been cupped between the shoulders, but has not improved under any treatment. He has not been incommoded by the tumour in his work, &c., and it has not much troubled his breathing. He has lately lost flesh to a great extent, and has suffered from heaviness at the forehead, dizziness of sight, and singing noises in the ears, and also from pain between the shoulders. He has never had epistaxis, nor have his legs swollen. His bowels have been regular, and his appetite good.

Symptoms on admission.—He is of average height, but of a slight build; his cheek-bones are high, and his cheeks and eyes deeply sunken. His eyelids are delicate and drooping, giving his face a sleepy aspect. His colour is of a peculiar yellow cast, which is extended over the body; the conjunctiva is of a pearly hue. The hair is black and straight. He has some colour in his cheeks, but he says that formerly he was of a much ruddier complexion. His body and limbs are rather wasted. There is an appearance of great listlessness about him, but he will not own to feeling ill or wearied, and says that he does not become quickly tired. However, he lies down a great deal, and sits about the ward, not seeming to care to get out of doors.

Upon examination of the abdomen a large, hard, smooth tumour is felt, passing forwards and inwards from below the ribs on the left side to the right side of the umbilicus for about the space of an inch. It fills up the whole of the left side of the abdomen, leaving a small space above its outer extremity at the right side, between it and the xiphoid cartilage, where the transverse colon is pushed forwards, and a small space below it between its lower border and the pubes. The tumour is very dull on percussion, and the dulness is continued up over the chest as high as the third rib, extending towards the axilla as high as the upper border of the fifth rib, and behind as high as the sixth dorsal vertebra. The dimensions of the swelling are as follows:

	Inches.	
Between the ant. sup. spinous processes of the ilia	18	
Between the xiphoid cartilage and the pubes	13	
From the most extreme point of the tumour which can be felt to the eighth rib	9	15 inches the whole length.
From that spot to where the dulness ceases	6	
At its broadest part in the abdomen	6½	

A notch can be felt in the margin of the tumour, just below the xiphoid cartilage. The upper margin passes from the border of the rib away to the right, above the umbilicus, and is then rounded away to the lower border. The finger can detect the margin easily, and feels it to be rounded and thin. The region of the abdomen over the colon is tympanitic, as also is the right side. There is no fluctuation, and he suffers from no pain. The liver appears to be somewhat enlarged. A slight bruit, probably due to the state of the blood, can be heard with the first sound of the heart. The apex of the right lung appears to be slightly less resonant than that of the left. The tongue is clean, and the pulse normal, but soft. The bowels are regular. The urine appears to be normal, but contains perfectly colourless, beautifully regular rhombic crystals, with rounded angles, mixed with some few coloured crystals, of a lozenge shape, and also flat square-shaped ones, striated at either extremity, with a clear space between. His breathing appears to be but little affected. He had, and still has, a slight cough.

His arm was pricked to obtain a specimen of blood. It was noticed that the blood did not readily flow, and that it coagulated directly. Otherwise it seemed to be healthy, and to possess no peculiar qualities. On microscopic examination

Fig. 1.

The accompanying figure shows the microscopical appearance of the blood.

the blood appears at first to consist altogether of white corpuscles. but when it is examined carefully this is found not to be the case. The white corpuscles, which look somewhat larger than those of healthy blood, are more numerous than the red corpuscles, and are collected together in masses, with the red in rouleaux between them, thus giving, at first sight, the appearance before mentioned. Acetic acid dissolves the red corpuscle, and brings into view in the white corpuscle a large well-defined nucleus, which does not exist in the same state in each corpuscle, but appears in some a single rounded or elongated body, in others as if dividing or divided into two or more portions.

To take for his cough Misturæ Cascarillæ comp. ℥j ter die.

He remained in much the same condition until the 7th June, when the report says:—"He does not appear quite well this morning. He seems to be heavy. Tongue foul. Bowels not open for two days. Pulse soft, 100. Skin very pungently hot to the touch. He complains very much of dizziness in his eyes and heaviness in his head. Eyelids puffy. Abdomen rather swollen, appearing more tense than before. He complains of a slight sharp pain, or stitch, over the upper part of the tumour.

To take Mist. Magnes. c. Magnes. Sulph. bis die donec alvus respondeat.

8th.—He is still very unwell. He complains of great pain over the tumour. Skin still pungently hot. Pulse 110. Bowels slightly opened. Urine clear, rather high coloured, with a slight deposit of uric acid.

9th.—He is still unwell and restless.

To take Liq. Morph. ♏v,
Sp. Æth. Chlor. ♏x,
Ex Aquæ Menth. Pip. ℥ss; ter die.

11th.—He is much better and brighter. Bowels not open. The skin continues hot.

To take Pil. Rhei comp. ij. Rep. mist.

[During the time since his admission Dr. Wilks often said in the patient's hearing, at his bedside, that an operation might be performed, and that he (Dr. Wilks) was of opinion that no drug would afford him any relief. Dr. Wilks also discussed the question of operation with Mr. Bryant, who was asked to see the case. They coincided in the opinion that there was no evidence of any organic disease except in the spleen; that any benefit from medicine was, to say the least, doubtful; and that death was imminent if the disease was left alone. They both regarded the case as a fair one for the removal of the organ. Mr. Bryant then laid the whole question before the patient, telling him of the incurability of the affection from which he was suffering, and of the prospect of an early death if he was left alone. He explained to the man that recovery might be secured by the extirpation of the spleen, and pointed out the dangers and the uncertainty of the operation. The patient, however, readily consented to the suggestion, and much desired that the operation should be performed.]

20th.—The operation was performed by Mr. Bryant to-day, in a private room in the hospital, at 2.30 p.m., Mr. Cock kindly rendering assistance. A vertical incision, measuring about five inches in length, was made on the side of the tumour, extending from the cartilage of the eighth rib to the anterior superior spine of the ilium, in the line indicated in the drawing.

Fig. 2.

The accompanying figure shows the position of the incision, &c.

When the abdomen was laid open the omentum was seen lying over the spleen. This was raised up and put on one side, and the spleen was exposed, looking of a deep slate colour. Mr. Bryant then introduced his hand along the tumour, between it and the abdominal wall, and found it quite free, with only one small adhesion near its posterior margin. The lower and posterior portion was then lifted out of the abdomen. Just as it was being lifted out there was a flow of blood from it, which did not enter the abdomen, and came from the lower and back part of the spleen. The upper part was then lifted out, and, after a careful examination of the pedicle, a clamp was fastened on. The pedicle was then perforated through the middle by a blunt needle, armed with a whipcord ligature, and tied in halves. The parts were carefully examined as to the existence of hæmorrhage, but none was discovered. The pedicle was therefore returned into the abdomen, the ligatures being cut short off. The wound was closed with silk sutures and a pad of lint applied, slight support to the parts being afforded by means of strapping. At the time of the removal of the tumour out of the abdomen the man's breathing became easier.

3.30 p.m.—He appears quiet and comfortable, and answers cheerfully. His breathing is easy. His pulse is good, but his skin has lost its pungent heat. To take only a little iced water. Suppositorium Morphiæ statim.

4 p.m.—He looks rather pale, and does not appear to be very warm. Pulse good. Skin moist.

4.30 p.m.—He has asked to be relieved of his water. He looks pale, and is not so warm as he was.

Five minutes later, when his water was going to be drawn off, he was found to be cold, his pulse was very low, and he was in a great sweat and very pale. A little brandy was given to him, and hot bottles were applied to his feet. At about 4.50 p.m., his pulse having become imperceptible, he expired, after two or three convulsions.

Sectio cadaveris, on June 21st, by Dr. Moxon.—There was no external scar upon the body except that of vaccination and two small punctures on the left arm, which had been made to obtain blood for microscopic examination. The one upon the external surface of the left forearm was rather swollen, and, upon cutting into it it was found to contain grumous clots infiltrating the areolar tissue around.

Dura mater and sinuses were free from morbid contents; the membranes, however, were injected with blood. The brain was firm, and equally so in all parts.

Right pleura.—There was a thin semi-tenacious coat of recent lymph, thickest over some patches of lobular pneumonia, which were subsequently found in the lung. There was but little liquid effusion.

Left pleura.—This was in a similar condition, but covered more thickly. There was also some turbid liquid filling up about one third or one fourth of the cavity.

Lungs.—The whole of the pulmonary tissue had a fawn-pink colour, due partly to anæmia, and every part was easily lacerable. The microscope showed the whole of the tissue, which appeared healthy, to be in a very remarkable state. The distended capillaries were full of white blood-cells, and the epithelium of the air-cells was much more developed than usual, so that the walls of the cells looked thick and were covered with capillary ramifications, the epithelium being seen in the meshes. Some small patches of lobular pneumonia also existed, some of which were in a state of gray hepatization, while others contained sloughs. They showed no tendency to suppurate.

Pericardium.—There were two milky patches on the visceral layer, the one near the base, the other near the apex; both were on the anterior surface of the heart.

Heart.—This was flaccid. In each cavity there was a clot looking more like strawberry cream than aught else, and of a brick-dust colour. The preponderance of white corpuscles in these clots was plainly manifest to the naked eye. There was no separation of a firm fibrinous clot, as would be the case after death from acute pleurisy; neither was there the emptiness or close contraction of the heart common in death from hæmorrhage. The microscope showed the muscular fibre to be very perfect, very few of the fibres being finely granular, with some yellow pigment here and there around the quadrate nuclei. The capillaries were full of white corpuscles. The valves were quite healthy.

Abdomen.—In the cavity of the abdomen, at its lower part, on the left side, a large clot of blood was found, weighing about one pound and a half. Water was injected into the aorta for the purpose of finding, if possible, whence the hæmorrhage had proceeded, but without success. The pedicle was examined, and the ligatures upon it were found to be perfectly safe. Above where the ligatures had been applied in the operation, a small piece of that portion of peritoneum which continues the gastro-splenic omentum into the ligament of the liver was seemingly torn. The clot was, like that found in the heart, of a deep brick-red colour, and not of the usual purple smooth and glossy appearance, but dull and granular on the surface; when squeezed it readily broke down into a grumous pulp. It was also peculiarly mottled with a network of a white beaded appearance.

Stomach.—Healthy, excepting a few bright patches of capillary ecchymosis.

Intestine.—Throughout its whole length the villi and lacteals were most beautifully injected with chyle. The microscope showed the villi to be bare of epithelium and full of fat-granules.

Receptaculum chyli and thoracic duct.—Normal. Not full of chyle. The thoracic duct opened into the right subclavian vein instead of the left. There was also a duct on the left side, opening as usual into the left subclavian; this did not enter the thorax, but received a small branch from the upper intercostal space of the same side. The glands generally might be estimated at twice their normal size, but the enlargement was not striking.

Liver.—This organ was bloated-looking and flaccid; on section it was yellow and thick-edged, with watery-looking portal fields. There was very little bile in the gall-bladder. The whole weighed 10 lb. 9 oz. The microscope showed the cells to be full of fat. Iodine produced no change.

Kidneys.—These were fatty and flaccid, but otherwise appeared to be healthy.

Bladder.—This contained about an ounce of cream-coloured urine.

Testes.—Both were healthy; there was slight varicocele on the left side.

Prostate.—Healthy.

Spleen.—This organ was enormously enlarged, especially in its longitudinal diameter, the lower part curving forwards. Longitudinally, it measured 25 in. round; transversely, $15\frac{1}{2}$ in. round at its widest part. It weighed 4 lb. 7 oz. It was very hard and solid to grasp, feeling much like a lung injected with tallow. The capsule showed patches of a yellowish-white hue here and there; the surface was rather shrunken at these spots. They varied from the one fourth of an inch to an inch in diameter. When cut into they were found to extend into the structure of the organ a little way (for a space about equal to half their breadth), and to be surrounded with a highly injected deep red zone. They were made up of a hard yellowish-white substance; their whole aspect was much like that of the embolic masses sometimes met with in the kidney. They were sixteen or eighteen in number. The spleen on section did not present its usual deep purple-red hue, but was of a paler colour and of a more solid appearance. It was very tough, being torn with difficulty, breaking with a ragged fracture under even pressure, and yielding but little "splenic pulp" when scraped with the knife. Here and there were seen large Malphigian corpuscles. Iodine produced no change in it. Very thin sections, when shaken ever so violently, retained their consistency and became in time pale coloured, and then appeared like pieces of fibre-tissue. Under a $\frac{1}{5}$th-objective a very large quantity of fibre-tissue was seen, with very firm fibres uniting and communicating, and having nuclei at some of their junctions. The corpuscles had large nuclei, and some were polynucleated. When water was added nearly every one of them spread itself out by imbibition into a watery cyst, in which a large and well-defined nucleus was situated.

Remarks.—Having decided that the extirpation of an enlarged spleen was a justifiable operation under certain conditions, and that it was expedient to make the attempt in a case in which there should be no evidence of disease in any other organ, and in which all treatment by medicine should seem hopeless, I undertook the case which has just been related with some amount of satisfaction, as it appeared to come before us under those very conditions in which such a proceeding could be entertained, and in which some hope of its success could even be realised. For in this case frequent examinations, carefully repeated, failed to yield any evidence of disease in any organ except the spleen; past experience told us too truly that medicinal treatment is utterly useless when applied to such a disease of the organ as was made out to exist, and it was quite clear to all observers that life could not be prolonged for many months were the disease allowed to run its course.

I am not disposed to go over on the present occasion the arguments which have been employed to justify the operation, for this has already been ably done by my colleague Dr. Wilks, in the last volume of these 'Reports.' It is sufficient for me to state that, to my mind, the facts and arguments he has adduced are sufficiently strong to allow of the operation being undertaken in certain cases with a fair probability of success, for it is true—

"That in the lower animals the operation of removal of the spleen has oftentimes been performed without apparent detriment to their general health.

"That the organ has been removed in man by accident, and without any untoward symptoms resulting.

"That the spleen is often found so shrunken and of so small size that, in all probability, its function has been long in abeyance."

These facts are indisputable; and if nature herself, by her own processes, at times causes the removal of the spleen without apparent detriment to health, it does not seem unfair to argue that the surgeon is justified in removing the organ when all evidence tends to show that life is threatened by its disease, and its disease alone. In the case under consideration these conditions appeared to exist, and it was on account of them that the operation was undertaken.

The operation.—There was nothing in the operation worthy of lengthened remark; it was far less difficult than I had expected, being, in fact, not more difficult than an ordinary case of ovariotomy. The incision selected gave abundant room for all necessary manipulation, and allowed of the ready extraction of the organ without force and without traction. There was but one small adhesion, which passed as a slight band from the upper and posterior portion of the spleen upwards towards the stomach; it gave way during the manipulation required to remove the organ; it was detected when the hand was passed towards the posterior part of the spleen, and it had disappeared when the whole gland was removed from the abdominal cavity; it appeared to be similar to and was not deemed of greater importance than the slight adhesions which are so commonly met with in ovarian operations.

The neck of the tumour was reached and commanded without difficulty, and great care was taken to make no traction or tension upon the pedicle. Mr. Cock kindly held the organ during this part of the operation and guarded against such a contingency.

The pedicle was at first secured by a clamp while the spleen was being removed; it was subsequently tied in halves by a sufficiently stout whipcord ligature. A blunt probe was employed to pass the cord, this precaution being observed lest a sharp instrument might wound a vessel, for the fear of hæmorrhage was present with me from the first, and every care was taken to guard against it, although without success. The ends of the ligatures were subsequently cut short off as in ovariotomy, and a careful scrutiny of the pedicle proved that it was safely secured. After the operation all the parts were examined, but no evidence of bleeding was observed, and I take it that the fatal hæmorrhage occurred from some small vessel, and probably from the vessel which the solitary adhesion contained.

After the operation there was no sign whatever of any shock to the system, nor of disposition to collapse. The relief to the man's breathing was immediate, and was very marked; the pulse rose and was of good power; the man spoke with force, and expressed himself as being relieved, and when I left him, two hours after the operation, I believed all to have been safe. The sudden death from hæmorrhage was unexpected, and took me by surprise.

The ultimate result of this case was certainly unfortunate, although it tells little or nothing against the operation. Should another example of uncomplicated spleen-tumour, by which the life of the patient is palpably threatened, come into my hands, I shall certainly make another attempt, for from this case I have learnt that the difficulties of the operation are not great;

that an incision such as I adopted is sufficient to allow of the removal of a spleen of any size; that the pedicle of the tumour can be readily found, held, and ligatured; and that the removal of the organ is unattended by any collapse or other immediate depressing influence.

I have also learnt that in a future case it will be well to deal with all adhesions, however slight, as they appear, and with the greatest caution; for it is still upon my mind that had I done so in the case related a different result might have been secured, and that the hæmorrhage which destroyed the patient might have been avoided.

SPONTANEOUS CURE OF ANEURISM OF THE AORTA.

By WALTER MOXON, M.D.

It is usual, in writing systematically of the management of internal aneurism, to describe its treatment as either palliative or curative.

This distinction has been made since Valsalva, by his determined use of bleeding, diet, and regimen, was said to have succeeded in curing aneurisms of the aorta.

His plan, which is distinguished as "curative," was never generally practised, and has fallen into universal neglect and under very general condemnation.

The "palliative" plan is stated to include the use of moderate bleedings, some limitation of diet, and the prescription of anti-spasmodic and sedative drugs, but it is fair to say that in this instance there is, as is too common in our time, some discrepancy between what is taught and what is actually done; blood-letting is commended, but it is scarcely ever practised.

The death of the patient, which the physician, in adopting the "palliative" plan, practically concedes to be inevitable, follows with unfailing certainty; and not only is this so, but death is very generally preceded by the most aggravated suffering, melancholy to witness.

When this disease, whose medical treatment is now so utterly worthless, is by chance found cured, it becomes a matter of great importance to consider the conditions under which such cure has taken effect, in order that these conditions may, if possible, be imitated.

The spontaneous cure of aneurisms in parts accessible to surgical operations is always treated of freely by surgical writers on aneurism, yet it must be very rare, for cases are difficult to find; and this, no doubt, arises because some decisive plan of treatment is soon put in practice when an aneurism is brought under the surgeon's care.

Mr. Porter, after describing in the usual manner the several ways of spontaneous cure of aneurism, says, "But we seldom see one of these exemplified; they sometimes suddenly disappear without our being able to explain the fact." He gives a case of a man propped up with pillows expecting death—an aneurism through his sternum—yet the man got well. Another man in Steevens's Hospital, supposed to have subclavian aneurism, likewise recovered without operation. In a third well-known case, after Mr. Porter had unsuccessfully tried to tie the innominate artery, the aneurism spontaneously disappeared. The first two cases are not accompanied by details. In neither of them was Mr. Porter able to explain the disappearance of the tumour.

Mr. Liston mentions a case in which an aneurism of the axillary artery disappeared, and on the patient's death it was found that another aneurism had compressed and obliterated the subclavian artery.

I have not found other cases of spontaneous disappearance of aneurism in the living subject. Several cases of aneurisms found cured during post-mortem examinations have been brought before the Pathological Society, and others are scattered about in books upon aneurism. To these I shall refer presently.

(Reported by Mr. Harris.)

Case.—Henry G—, æt. 46, a chimney-sweep, was admitted into Luke Ward, under Mr. Hilton, February 8th, 1866. In August, 1865, he suffered from cancer of the scrotum, which was removed by Mr. Cooper Forster. The disease was confined to the right side of the scrotum. Two or three months after the operation the glands in the left groin began to enlarge. These have continued to increase in size up to the present time. The tumour is now of the size of a hen's egg. The skin is ulcerated over the surface.

February 13th.—The tumour was removed to-day; the wound was deep, about three inches long and two broad; the aponeurosis of the external abdominal oblique muscle, the spermatic cord, and the sheath of the femoral vessels, were exposed. Severe hæmorrhage occurred about an hour after the operation; this was arrested at once.

14th.—He complains of great weakness.

Until the 10th of March the report is favorable. On that day it is said, "Numerous large granulations are springing up, but the wound has an unhealthy appearance; the integuments above the pubes are much thickened; the penis is œdematous. The general health is tolerably good."

20th.—The carcinomatous growth, which has infiltrated the integuments above the pubes, is extending into the right groin; the wound is gradually filling up.

22nd.—The growth in the right groin is extending rapidly. The skin over it is ulcerating. To-day some hæmorrhage occurred from the old wound, but ceased without any interference. The appetite is bad.

29th.—Hæmorrhage has again occurred; the malignant disease and ulceration continue to extend rapidly.

April 7th.—It is stated that he is in a very low state.

13th.—He died at 4.30 a.m.

Inspection, made eight hours after death.—Body considerably emaciated; slight icterus; a bedsore on the sacrum. A great excavated space involved both groins, the pubic region, and the left side of the remains of the scrotum; the edges much raised; large lumps projecting from the inguinal part of the floor; these proved on section to be cancerous lymphatic glands.

Head.—Brain natural; two large clusters of Pacchionian glands had grown through the inner table and diploe of the frontal bones, and raised the outer table, which was very thin over them; the larger cluster was of the size of a horse-bean.

Thorax.—The left pleura had on each lobe a patch of recent lymph; beneath the patch upon the lower lobe was a small lobular abscess in the parenchyma of the lung; this had all the characters of pyæmic pneumonia.

In each lung were several bean- to nut-sized nodules, pale and dryish on section. Microscopic examination showed in them the "bird's-nest" cells and adventitious epithelial scales which characterise epithelial cancer. The pulmonary tissue was still present for some distance within the margin of the tumour, the air-vesicles being occupied by the epithelial growth.

(The extremely rare occurrence of remote extension of epithelial cancer is here rendered more interesting by the coexistence of the cancerous products with pyæmic pneumonia in the same lung. Either the cause of the pyæmic abscess or the cause of the cancerous nodule cannot be a specific disease, or else a specific disease cannot be a condition of the body in which any "fibrin" exuded from the blood takes on one particular mode of action.)

Heart.—Into the left auricle in front of the septum projected a tumour (Pl. I, fig. 2 *b*) of the size of half a large walnut, somewhat constricted where it was continuous with the wall of the auricle. The sac was not full-looking, but rather flaccid, and soft on pressure; it was found to be filled with laminated and partially softened clot, of the colour and consistency of cream cheese, the lamination being concentric with the wall of the sac, and the centre soft and pus-like. No way could be found from the sac into the aorta.

The interior of the aorta was very much disfigured by cartilaginous and calcareous degeneration; close inspection was required to discover the mouth of the aneurism; it was situate in the posterior sinus of Valsalva, just below the level of the upper edge of the valve, and it was turned downwards, so that the point of a probe would just enter in an upward direction into a transverse recess about four lines long (Pl. 1, fig. 1 *a*); but the probe could not pass into the aneurism, for the lower lip of this recess had become adherent within the recess to the hinder face of the upper lip. The entirely colourless and softened state of the contents (which were undergoing the disintegrative changes that occur in the so-called polypous concretions common within the heart), and the complete and firm closure of the mouth of the aneurism, prove the unusual completeness of its cure.

The mitral valve was very perfect.

Abdomen.—The liver had beneath its upper surface two considerable patches of cavernous tissue. The other abdominal viscera were normal.

The bladder was much inflamed within, its submucous tissue being in a state of suppuration; no mechanical obstruction of its neck existed. Numerous thrombi were found in the prostatic veins.

The lumbar glands near Poupart's ligament were much enlarged and cancerous. Microscopic examination of the skin and glands showed numerous endogenous cells, but the forms held to be characteristic of epithelial cancer were not so well marked here as in the lung.

In passing, it should be remarked that the position of this aneurism is one of extreme rarity. The only instance of aneurism thrusting into the left auricle which I am acquainted with is that which Mr. Thurnam describes in his valuable account of "Varicose Veins" ('Med.-Chir. Rev.,' xxiii). The specimen is in the Museum of the Faculty of Medicine at Paris. In this case the aneurism also thrust into the pericardium, but it burst into the left auricle. No such case has been before the Pathological Society, nor is there any in the museum of Guy's Hospital, nor, so far as I am aware, in any other pathological collection in London.[1]

The great rarity of aneurism thrusting into the left auricle is doubtless due to the position of the left auricle in the concavity of the aortic arch. The chief distensive strain upon the coats of the artery is exercised upon the convex side of the arch, which has to turn the course of the blood stream.

[1] By a curious coincidence two instances of aneurism thrusting into the left auricle chanced to come to the post-mortem theatre at Guy's within two weeks. The second specimen was given me by my friend Mr. Hankins; it is fully described in the 'Pathological Transactions' for the present year, and figures of it are appended to the present paper (Pl. II, figs. 3 and 4). The aneurism is of the size of a large walnut, and extends from the aorta about three quarters of an inch above the posterior valve; its mouth is of the size of a fourpenny-piece. It has burst along the line of its continuity with the auricle by a rent one inch long (*d*).

The person from whom it was removed had been a seafaring man. He had for weeks had some uneasiness and pain about his præcordia, chiefly in the morning on awaking. He went out one morning and on returning in the afternoon he lay down, tactiturn and dejected; he was found at half-past six p.m. leaning over a pail, retching violently. He lived until ten p.m., and meantime was pale, restless, covered with cold sweat. He was not seen by a medical man until two minutes before his death, and therefore the auscultatory signs were not noted.

The completeness of the cure of the aneurism in Henry G—'s case is remarkable; the continuity of the arterial coat has been completely restored, and nothing remains of the mouth of the sac but a minute recess, much like the relic of the foramen ovale in a normal adult heart. The only case I can find recorded of so complete a cure of an aortic aneurism is one quoted by Mr. Hodgson from Corvisart. He found a remarkably hard tumour, at first sight like a bronchial gland in size and appearance, which proved to be a "fibrous sac that by its base adhered intimately to the coats of the aorta, with which, at the same time, it appeared to be in some degree connected;" it "was formed of a membrane, evidently of a fibrous texture, about two lines in thickness, and it contained a substance less firm than suet, of a deep red colour, very similar in other respects to the old clots of blood which adhere to the internal surface of aneurismal sacs. I conceived that the cyst communicated with the cavity of the vessel to which it was attached, but it was in vain that I sought for a communicating aperture. The external coats of the aorta, at the part corresponding with the cavity of the cyst, were disturbed, and the thickness of the vessel at that point was infinitely less considerable than in any other situation. Having opened the aorta longitudinally, I could not perceive any aperture of communication, but I saw a gray livid spot that corresponded with the base of the cyst.

"There was another in the course of the same vessel, just by the cœliac artery."

The manner of cure in Corvisart's case seems to have corresponded with that in the case of Henry G—, but the time which had elapsed since the cure was completed had effaced the evidence of the process by which the closure of the mouth of the sac at the "gray livid spot" was effected. In Henry G—'s case the lips of the orifice of the aneurism had grown over the orifice until the wall of the artery had its continuity restored by the coalescence of the produced lips of the aperture, the upper lip overlying the lower at the place of meeting and union.

This process is one of common reparative inflammation, a condition which does occur in the middle and inner coats of arteries, notwithstanding that they are destitute of nutrient blood-vessels. The belief in the inability of these coats of arteries to inflame was deduced from the belief that no structure which is non-vascular can inflame, and was a necessary consequence of the vascular theory of inflammation; but, in fact, the products of inflammation may frequently be seen in and upon the inner and middle coats of arteries, and the whole of this piece of deductive reasoning is but an illustration of the danger of reliance on the *à priori* method of arguing in matters of experimental inquiry.

This manner of cure of aneurism by union of the lips of its mouth has never been described. Yet I believe it is the only way in which a complete and satisfactory cure can be established where the channel of the affected artery is not obliterated; evidently it is possible only when the mouth of the aneurism is very small.

In all the cases of internal aneurism given as cured by Mr. Hodgson, and in all those to be found in the 'Transactions of the Pathological Society,' the affirmation of cure is founded on the complete filling up of the sac, the orifice of which, however, remains unhealed, except that the clot is in some instances smooth on its intra-arterial face, and that this face is sometimes covered with a uniform, almost membranous layer.

The instance of healed aneurism which has just been detailed was associated with great wasting of the body; three severe hæmorrhages and the constant discharge from an enormous open sore had reduced the patient to a state of emaciation. It is not possible to overlook the identity of the curative plan of Valsalva with these conditions, which, indeed, had healed the aneurism in spite of the surgeon's care. An examination of the thin wall of the sac, unsupported as it was, proves that, at the time when the healing process began to take effect, the aneurism must have been on the point of bursting, an accident which would have finished Henry G—'s life in a few hours. So that no reasonable doubt can be allowed that the cancer, with its attendant discharge and hæmorrhage, and the consequent lowering of the tension within the vascular system, had prolonged his life; and that, save for the cancer, he would have died some weeks before by the bursting of the aneurism.

By a general survey of the recorded cases of the so-called spontaneous cure of internal aneurism I shall show that this case is not exceptional in being associated with wasting disease, but, on the contrary, that it may be stated, as a general rule, that the spontaneous cure of internal aneurism, when it occurs, takes place during emaciation.

In proof of this I will adduce first the cases which are given in the 'Transactions of the Pathological Society.' These are the following:

1 (vol. iii, p. 303).—A case recorded by Mr. Barlow; a woman, æt. 24, pale, weakly, and of irregular habits, in whom an aneurism was found completely full of clot. The heart proved to be *very fatty* on examination by Drs. Basham and Quain.

2 (vol. v, p. 107).—A case recorded by Mr. H. Ludlow; a very large aneurism, seven inches by four. This aneurism was extending rapidly; the patient then took to living on beef-tea, milk, and light puddings; in three months the aneurism ceased enlarging, and hardened, but it was noted that, whilst these changes were taking place in the aneurism, the man became pale and emaciated, and so feeble as to require an improved diet. He had not left his bed for six months.

3 (vol. ix, p. 172).—A case recorded by Mr. Holmes; this patient had heart and kidney dropsy; the state of his nutrition is not mentioned; the sac was nearly filled with laminated and partially decolorized coagula. (The aneurism appears by the description to have been a uniform distension of the cœliac axis; it communicated with the aorta by a half-crown-sized hole, and the branches of the cœliac came from the front of it; blood passed freely through it.)

4 (vol. ix, p. 38).—Dr. J. W. Ogle's case; an aneurism of seven years' standing; patient at first in St. George's Hospital, and relieved; when readmitted after five years was worse as to the aneurism; "a decided pulsation beneath the inner end of the second rib on the right side," &c. Dyspnœa very urgent The aneurism was found at his death full of clot, and is reported as cured, but he had phthisis of both lungs. Dr. Ogle, in his remarks, expresses the opinion that the tuberculosis may have been suspended whilst the aneurism healed. Is it not, rather, probable that the development of the tubercle, and the consequent discharge and wasting due to the phthisis, caused the cure of the aneurism?

5 (vol. x, p. 83).—Case recorded by Dr. Bristowe; two aneurisms, one of them quite full of clot, the last layer of which

clot blocked the mouth of the aneurism. The patient had vomiting and icterus, and was very emaciated; his countenance was pale; his legs were œdematous.

6. Dr. Stokes ('Diseases of the Heart and Aorta,' p. 559), notwithstanding his determined opposition to the curative plan of treatment, gives a case of a similar kind.

In this case a vast aneurismal dilatation, presenting two or three pouches, one as large as a hen's egg, containing a quantity of dense and laminated fibrin, lay exactly in front of the trachea. It was completely filled with fibrin. The patient died of phthisis, without symptoms of aneurism.

7. The same author gives a case of pulsating tumour in the region of the arch of the aorta, causing absorption of the ribs and perforation of the integuments. In this case frequent hæmorrhages occurred, attended with signs of diminution of the tumour; subsequently there was great improvement of the health under a generous diet.

Mr. Hodgson, in speaking of the spontaneous cure of aneurism by filling up of the sac with fibrin, gives four cases, in all of which lessening of the tumour was caused by extensive bleeding and dieting.

Pelletan had remarkable success with Valsalva's plan of treatment; by it complete cures were obtained in at least two, if not three, cases out of six; the other cases were much relieved.

Now, such cases not being selected, but being all the cases of cured aneurism, or aneurism stated as cured, which have come to my hand in the course of such search as I have been able to make, may be taken as a fair sample of average experience of such cases. It may be affirmed that in all those of them in which the method of Valsalva was not actually employed the cure of the aneurism coincided with conditions practically equivalent to the deliberate employment of Valsalva's method. These conditions were—

1. Cancer of thigh, hæmorrhage, and wasting.
2. Pallor and weakness, with very fatty heart.
3. Pallor, emaciation, and feebleness.
4. Heart and kidney disease.
5. Double phthisis.
6. Phthisis.
7. Bursting of aneurism and large hæmorrhages.

8—11. The four cases by Mr. Hodgson, treated with bleeding and low diet.

12—14. The three cases, by Pelletan, treated with bleeding and low diet.

The only case which is open to objection is Case 4. This patient died of heart and kidney disease. It is not stated whether he was or was not wasted, but the rule in cases of such disease is that the body is very ill nourished; it is really much more wasted than is apparent, emaciation being concealed by dropsy.

These cases are not open to the objection (of doubtful fairness) quoted by Dr. Stokes, from Bertin, that the cases recorded as cured by Valsalva's plan of treatment were only aortitis or simulated aneurisms.

They justify the statement that the cure of aneurism is always associated with wasting of the body, or conditions otherwise equivalent to the method of Valsalva.

On the other hand, the aspect of a sufferer with internal aneurism in a state of activity is generally one of robust health; and this fact is commonly used in the diagnosis of these cases by practical physicians.

So far as this evidence goes, then, it would appear that the progress of aneurism is directly as the good nourishment of the sufferer, and that his chance of cure is directly as the wasting of his body.

In five aneurisms which came upon the post-mortem table at Guy's during the past year it was found that the quantity of clot present in the aneurism was directly proportionate to the wasting which the body had undergone. The case which most nearly approached a cure—in which, indeed, one of two aneurisms present was so far cured as to be actually full of clot—was that of a man under Dr. Rees' care, in Stephen Ward. This man was admitted with a pulsating tumour behind the sternal end of the clavicle on the right side. One aneurism extended towards the right lung, arising from the innominate artery, and another rose from the root of the right carotid; this latter was wholly blocked up with clot. During life the case was regarded as one of malignant tumour, and the diagnosis appeared to be confirmed by the wasting and cachexia which supervened during the lingering progress of the case, and by the concurrent disappearance of pulsation in the carotid tumour. In contrast with this were two cases, in each of which the body was quite fully nourished, and in which no clot was present in the sac. In the remaining two cases the nourishment had suffered, and there was a moderate proportion of clot in the aneurismal sacs.

It is usual to say, that the quantity of clot in an aneurism varies with these two conditions—the distance from the heart, and the size of the orifice of the aneurism. But in each of the cases above alluded to the aneurism was of the aorta or the a. innominata, and opened by a wide orifice. That at the root of the right carotid was hemispherical in shape, and its mouth was its widest part, yet it was entirely filled up with clot.

Quiescence in the contents of the sac is, no doubt, the primary requisite for coagulation, and this appears, from the facts stated above, to be superinduced most certainly by the feebleness of the circulation which goes with emaciation.

And yet, although the proposal to treat aneurism by a method conducive to wasting, has, for a very long period, been before the profession, that method is not now employed, and is very generally condemned by high authorities.

At first sight this appears a fatal objection to this plan of treatment, for it apparently signifies that the plan has failed after very general trial.

But while the importance of general conviction, when it is wide-spread and long enduring, must not be undervalued, it must yet be remembered that such general convictions acquire their value only from the supposition that numbers of competent observers have, throughout the time, tried and found that such convictions are in accordance with experience.

Now, an examination of the history of Valsalva's method convinces me that this condition is very far from having been fulfilled in regard of the general conviction, unfavorable to that method, which now undoubtedly rules. Nay, further, that the results of actual trial have been, on the whole, very favorable to the method. Those speak most highly of it who have tried it most perseveringly, and those who object to it, object chiefly on theoretical grounds.

Before proceeding to discuss these theoretical objections, I would remark that the distinction between curative and palliative treatment appears to me to have done much ill-service to the efficiency of the medical treatment of aneurism.

It seems to challenge the lowering method to show actual cures; it enlists against a method professing to cure aortic aneurism our belief in the hopelessness of that formidable disease, when, in reality, the "curative" and "palliative" methods only differ in degree, and effectual palliation is to be gained only by means which, carried further, form the so-called curative method.

If the histories of all cases treated by the modern expectant method were written, and contrasted with the histories of cases which have been treated by low diet and bleeding, the contrast would lead to the common adoption of the latter means, if only for the relief of the symptoms; and this more especially when we see that the relief of the suffering of the patient tends to his cure in proportion to the extent to which he will allow the practice to be carried.

No writer states more fully and forcibly than Dr. Stokes the objections that are urged against Valsalva's plan of treatment, and to the justly great influence of his opinion is due, in a great measure, the general aversion to the lowering method.

Dr. Stokes says, "We must be contented to deal with palliative rather than with curative treatment. It is to be doubted whether we are justified in adopting any measures which, while they are directed under theoretical views to the cure of a disease, materially interfere with the patient's condition." To this very general statement it may be answered, that if any plan of treatment is ever found successful in a disease which, when only palliated, is uniformly fatal, no purely theoretical views should be allowed to put aside a fair trial of that treatment in every case of such otherwise fatal disease. So that, as it can be shown that patients have been cured of aortic aneurism by wasting, if any physician has it in his power, by consent of his patient, to try that method, and yet neglects to do so, no theoretical views will supply a sufficient answer for such neglect.

Dr. Stokes says further—"(1) The patient's mind becomes excited and apprehensive; (2) his system weakened by depletion; (3) his digestive functions ruined by starvation; (4) the forces by which he can resist disease are broken down; (5) his blood becomes uncoagulable; (6) his tissues unresisting; (7) the force of the aneurismal throb is augmented; (8) and a disease which, under other circumstances, might have endured for years, with but little interference with the general health, is turned into a rapid and destructive malady."

I have numbered the particular objections, for convenience of reference; they have, collectively, a most formidable weight, and would, perhaps, exclude the debilitating method if there were any alternative, however bitter.

Against the treatment of aneurism in the limbs by that method they are decisive, for the surgeon possesses a plan which offers an almost certain cure, with little danger or pain. But it is quite otherwise with the physician, who finds in internal aneurism a disease which, save for this one hope only, will surely prove fatal. Every objection to the plan must therefore be scrutinised, and allowed no more than its due importance. As to the first objection, the reflection naturally arises that the patient must be aware that he labours under an otherwise hopeless disease, and may be made to understand that the lowering plan offers his only chance. (2) The weakening of the system and (3) the damage to digestion are in this plan of treatment what the removal of a limb is in the treatment of cancer of that limb—dangerous, but necessary—not a certain cure, but rather the only chance. (4) These forces, as represented by the force of the circulation which they cause, are the very forces that are destroying the patient. The mechanical tension within his vessels—the consequence, and the index of his bodily health—is urging its way to his certain death. (5) The facts I have adduced above are in direct contradiction of this assertion, which is based upon purely theoretical grounds. (6) "His tissues unresisting." Here my leading case comes in with peculiar force, for the aneurism which healed so completely during emaciating disease was WITHIN the left auricle, bathed in blood, and utterly unsupported. The idea of weakening the elasticity of tissues is put forth by Dupuytren, but it is not shown that depletion lessens the mechanical resistance of the connective tissues. No doubt the objection would be of importance in the case of aneurisms which, from their position, could be opposed by muscles, for the muscles would lose their tone by depletion. But this is not the case, to any appreciable degree, with the connective system of tissues; such tissues as have to resist the extension of internal aneurisms.

(The yielding of the scrotum and of such parts as are concerned in hernia is encouraged by debility, but this is due to the yielding of the muscles of those parts.) (7) The force of the aneurismal throb surely cannot be augmented if the state induced by the treatment be that induced by stricture of the œsophagus, or by phthisis, *i. e.* be *not anæmia, but wasting.* The distinction is important. The pallid but plump condition induced by mere loss of blood, as from hæmorrhoids, is not the state to be aimed at for the cure of aneurism, and it never need be induced. The objection holds with full force against pure anæmia, with its throbbing and excitable pulse; but it is surely in our power to superinduce wasting without disproportionate anæmia. Stricture of the œsophagus induces a condition which does not include excitement of circulation and throbbing pulse; no pathological condition is more easy of imitation than that induced by stricture of the œsophagus. (8) If the case be one in which the patient is comfortable, and likely to live long, he might be left alone; but what proportion of aortic aneurisms are of this favorable kind?

On the whole, then, these objections do not appear to be of moment enough to outweigh the twofold advantages of a lowering plan of treatment—the advantage of a chance of cure and that of the alleviation of the symptoms due to the presence of the aneurism.

I have avoided any allusion to the quantity of fibrin in the blood, and its supposed bearing on the prospects of cure in different modes of treatment of aneurism, because there is no proof that fibrin is an index of vigour or that the blood in states of malnutrition contains less fibrin. The state of anæmia which is commonly appealed to is not the state desirable in aneurism. My own experience is that the fleshy clot in the heart, which closely resembles the matter filling up aneurisms, is found toughest and in largest quantity in cases of lingering death by wasting disease.

Lowering the tension within the aneurism by direct means has been called *curative,* and so stigmatised as impracticable.

The *palliative* plan, as described in books, has, in practice, been deprived of the only beneficial parts of it—the bleedings and the low diet; partly by the natural tendency of the mind to cause the contrast in the respective facts of the two plans to correspond with the contrasts in their respective names, and partly by that modern aversion to all lowering measures by which we are now overruled in the way of reaction from the too-extended and indiscriminate use formerly made of them.

But the distinction of palliative and curative plans appears to me to be unnecessary and mischievous, for the measures which most effectually palliate are those which most tend to cure.

EXPLANATION OF PLATES.

PLATE I.

Fig. 1. Shows the small trace of the orifice of the aortic aneurism in the posterior Sinus of Valsalva.

N.B. *The line from "a" should have been continued on to the next sinus to that in which it is made to terminate.*

Fig. 2. The aneurism seen in the left auricle, and cut open at *b* to show the laminated clot within.

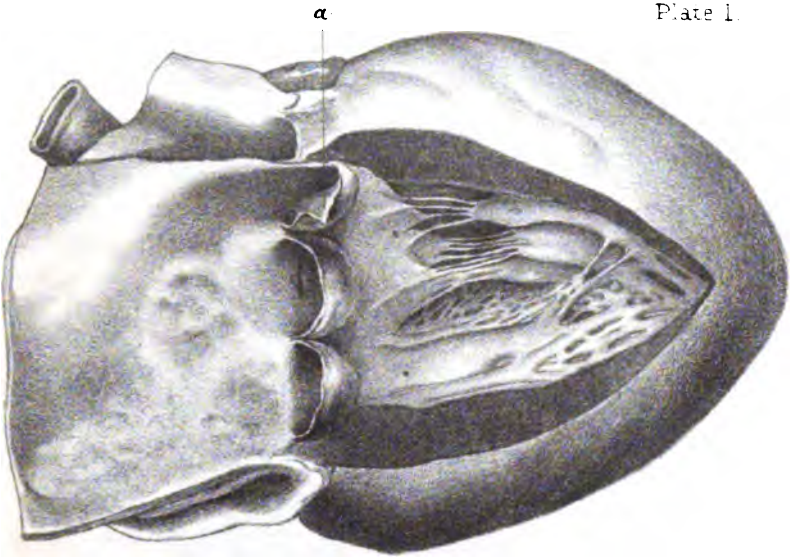
PLATE II.

Fig. 3. Showing the orifice of the aneurism at *c*.

Fig. 4.[1] The aneurism seen in the left auricle; *d*, the line of rupture.

[1] See footnote, p. 458.

Plate I.



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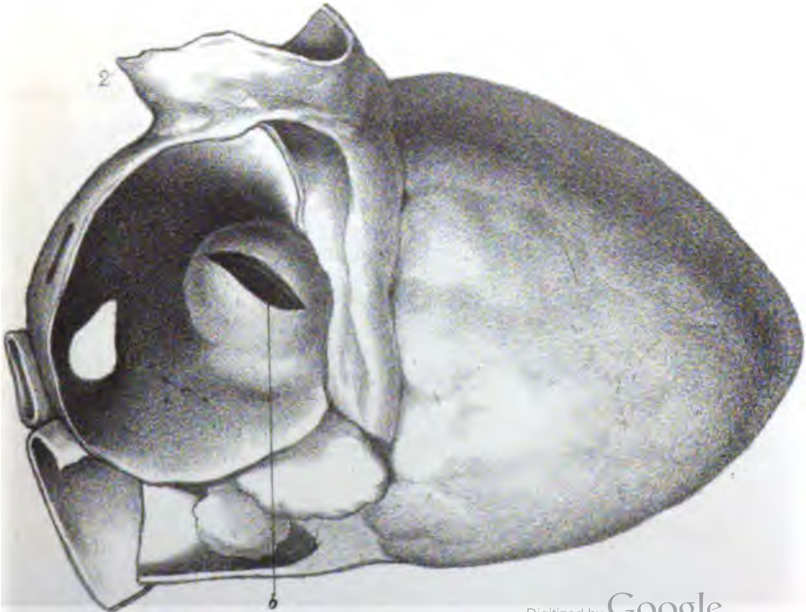

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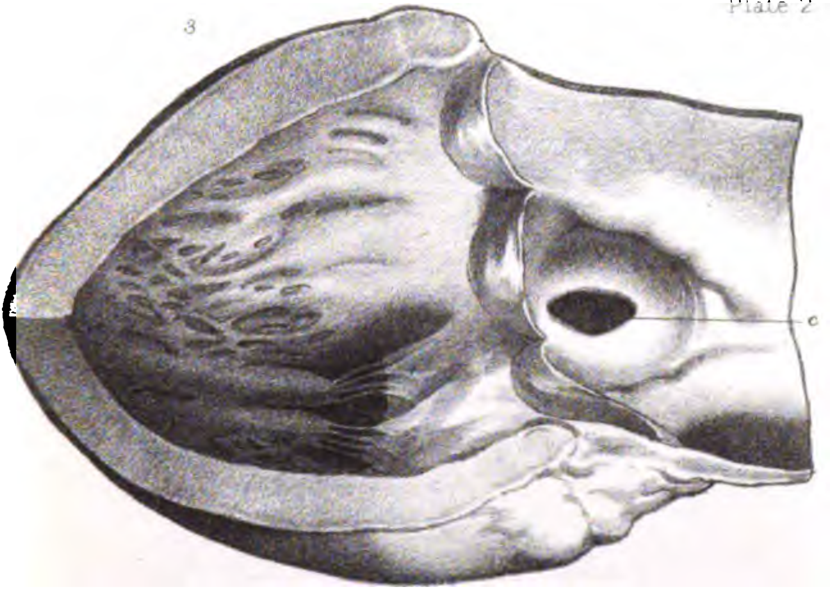
Plate 2

3



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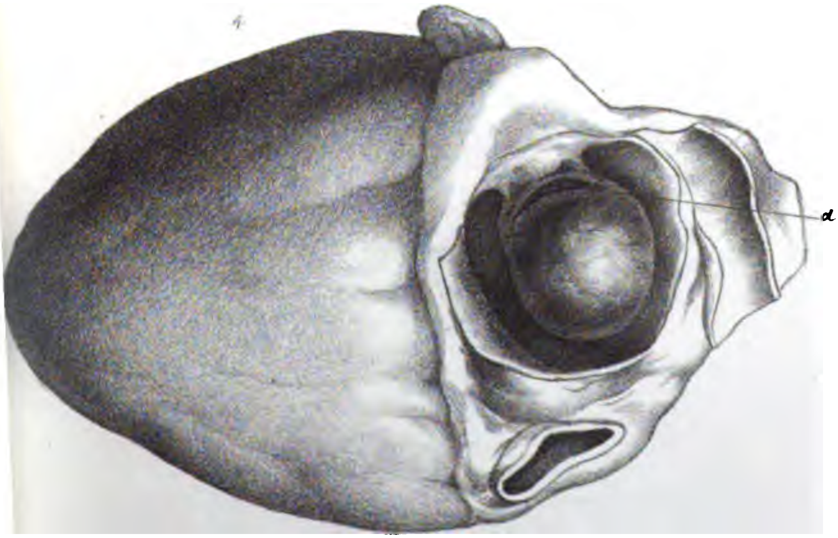

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NOTES ON CASES

CONNECTED WITH

OBSTETRIC JURISPRUDENCE.

By J. BRAXTON HICKS, M.D., F.R.S.

Fracture of the skull in new-born infants.

Fracture of the skull in the newly born has engaged the attention of medical jurists; and it is now admitted that by uterine contractions alone, and even when there is not much obstruction to the exit of the fœtus, fissures of the cranium can be produced. It is also known that in a standing labour fracture may take place from the child's head falling upon the ground, the facts connected with this subject having been well investigated by Dr. Klein. But the effects of lateral pressure on the skull of a newly born infant have not yet received any attention, at least to my knowledge. Yet this point is, to a certain degree, important, because violent manual pressure in a lateral direction may be employed by the frantic mother to deliver herself, if surprised in the midst of illegitimate labour, when the head only is as yet passing the vulva. My attention was particularly called to this subject by a case of supposed infanticide, in which my opinion was requested as to the cause of death.

A new-born child was found dead. A laceration of the angle of the mouth with a fracture of the lower jaw existed, and also a fracture of one humerus. There was a fracture through the arch of the skull, from one side to the other, with a fracture also of the frontal bones. There was some ecchymosis beneath the scalp, but external marks of injury were wanting. The peculiarity in the transverse fracture of the skull, combined with the laceration of the angle of the mouth and the fractures of the jaw and the humerus (the latter being meaningless for the destruction of life), led me to suspect that probably the injuries had been produced by the mother endeavouring to draw the child from her after the head was born. This would quite explain the fractures of the arm and the jaw and the laceration of the angle of the mouth, for the mother was a servant, and might have been put suddenly in fear of being discovered in the act of labour.

This manner of delivery would not, however, explain the cranial fractures. I therefore instituted some experiments upon the fœtal skull to ascertain whether lateral grasping of the head could produce a fracture of the cranium, in the same manner, as in some fœtal skulls, fractures of the cranium can be produced by the natural efforts of the uterus against the bony passages. This fact argues a very great degree of frangibility, and one must admit that the compression of the head between both hands, in violent attempts at delivery, would be at least equal to the action of the uterus in producing fractures.

I therefore tried upon eight still-born infants the effects of pressure with both hands.

The first skull which I tried was one of remarkable firmness and size (the size of the child having been the cause of its death). Firm compression with both hands produced no effect. I then placed the head on the floor on one side, and pressed firmly upon it. A fracture was afterwards found to have occurred, extending through the arch of the skull in a manner much resembling that observed in the child whose case is above mentioned.

In the *second* head I was able, by manual pressure on both sides, to produce a fracture through the arch not quite so complete, but still very marked and striking.

In the third and fourth cases no result was obtained. These experiments were made on small yielding heads, with loose sutures and soft bones, easily moulded into any form.

In the *fifth* a small fissure-like fracture of the margin of the left parietal bone was effected, which, however, was produced rather by the indentation of the bone under the immediate point of pressure than in the indirect mode in which it took place in the cases above mentioned.

In the rest of the cases no effect was produced, the skulls yielding so much as to render a fracture impossible.

Hence it appears that a certain number of fœtal crania are so constituted as to be capable of fracture under the circumstances of which I have been speaking. We might, indeed, have expected to find that such would be the case if we admit the possibility of the uterus fracturing the fœtal skull. Probably, however, the per-centage of these frangible crania is small.

The fracture in these instances was in the line of the temporo-bregmatic diameter, and, from the form of the head, it is highly probable that lateral pressure on the head would produce this kind of lesion. But, of course, where the resistance is great, the pressure would tend to act upon the part more immediately beneath it, and would thus produce depression of the bone and fracture at other parts, and not only in the line above indicated.

In character the fracture is, perhaps, more like a fissure than anything else. In such a case there would not be much contusion, although there would probably be a certain amount of ecchymosis beneath the skin in the line of fracture. There would be no injury of the skin, at least not in connection with the fracture.

Hence, supposing that a fracture of the skull were produced during childbirth by lateral pressure made for the purpose of extraction, we should probably find, according to the foregoing cases, points of difference sufficient to enable us to distinguish between a fracture so produced and one effected for the purpose of murder.

But there is probably not sufficient evidence to enable us to say, in a given instance, whether a fracture was produced in utero or by the lateral compression of the skull during birth.

Fracture of the fœtal cranium in utero by the midwife's manipulation.

I was called to assist a midwife in a difficult case of labour

The head was still above the brim. I detected a fracture of the cranium beneath a deep and extensive scalp tumour. Ultimately the child was delivered by the forceps; the instruments may, possibly, have increased the fracture, although, from the slight pressure I applied, I think that they did not, but there could be no mistake as to the existence of a long fracture before their use. On inquiry I found that the midwife had been endeavouring to press the child's head backwards from off the os pubis, on which it was lodged, into the hollow of the sacrum.

The bone beneath the cephalhæmatoma was examined after the death of the child, which occurred next day. The report says—"The vertex was covered by a large fluctuating tumour, which, on removing the scalp, proved to consist of semi-coagulated blood. There was a fracture of the right parietal bone, running from the eminence towards the temporal bone, and there was another fracture running from the same spot towards the frontal bone, thus leaving a triangular portion of bone quite loose and depressed. There were two or three other smaller pieces of bone broken off from the adjoining margins of the temporal and occipital bones. There was not much blood extravasated beneath the dura mater."

Now, supposing that in a similar case the child was illegitimate, and that after birth it had been put aside without much examination, and supposing a suspicion of ill-treatment to have arisen, the death of the infant having occurred in the absence of any witness, would it not be very difficult for the mother to clear herself of the imputation?—and in what way can a post-mortem examination help us in solving the difficulty?

Case of attempt to injure the child during the pangs of delivery.

The following case is of value as a means of explaining the frantic actions of the mother in childbirth in regard to her child.

I was attending in labour a highly respectable woman, who had had many children. The labour was rather severe as the head passed the vulva. Suddenly she raised herself up and called out—"Let me get at the child; I'll murder it." She made great efforts to reach it, and it was with very considerable difficulty that she was restrained. However, the expulsion soon occurred, and she then relapsed into a state of quietude, afterwards exhibiting no further sign of wishing to do otherwise than a mother should to her offspring.

The explanation of such actions is not very difficult. By some women pain is less patiently borne than by others, and by all its cause, whether animate or inanimate, is apt to be looked upon with a degree of anger. Hence, those who bear pain worst are apt, in the excitement of the moment, to overlook the relationship they bear to its cause, and even regard this as an enemy which they are anxious to get rid of.

Numerous instances of a similar kind, though in a less degree, can doubtless be remembered by all practitioners.

A case in which "apparent" death of a fœtus occurred between the birth of its head and that of its body; recovery by artificial inflation.

The following case (mentioned in Dr. Taylor's 'Princip. and Pract. of Medical Jurisprudence,' 1865, p. 921) possesses a certain amount of interest in jurisprudence.

A woman who had previously had eight easy deliveries was detained in her ninth labour by reason of the size of the child, which weighed 14 lb. Forceps were applied, and the head was delivered; the instruments were then removed. The child breathed four or five times, but the difficulty of delivering the shoulders and body was such that, before this could be accomplished, both the respiration and the action of the heart had apparently quite ceased. However, after inflation by the catheter through the larynx, the child breathed; it ultimately recovered, and was alive a twelvemonth afterwards.

This case, as Dr. Taylor remarks, nullifies the dictum of Casper, that when air is found in the lungs of a dead child the child must have been born alive. In this instance witnesses were present, but in another case delivery of the head might quite possibly occur, and the child breathe before the birth of the shoulders, and yet die before the remainder of the body escaped.

It is not very uncommon to witness in a natural labour, if of some considerable duration, the cessation of uterine efforts after the head has been expelled; under these circumstances the child generally breathes. Now, should complete delivery be postponed, either by the size of the shoulders impeding, or by the absence of expulsive efforts, it is not at all difficult to suppose that the fœtus might be still-born, and yet that air might, to a certain extent, be found in the lungs.

Cases in which attempts at inspiration were defeated by spasm of the larynx, &c.

Upon more than one occasion I have seen a new-born infant make several attempts at inspiration, but, from spasm of the larynx and retraction of the tongue, the air has been unable to enter. In one case in particular the chest was thrown open and arched; the tongue was retracted; the chest, when the inspiratory efforts took place, was lifted up in the centre, but the ribs on both sides were collapsed or flattened, so that a transverse section of the chest would, at this time, have been triangular.

In this case I pressed upwards and forwards the root of the tongue, lifting up the epiglottis; the air then entered easily and the child was saved. In some other cases I have been less successful. The exact cause of this condition is not clear. Sometimes, however, mucus, meconium, or some other foreign body, has been present and has irritated the larynx.

Now, those who consider respiration to be necessary to establish the true life of the new-born infant must admit also that, under these conditions, a child cannot be considered as a living one, notwithstanding that the heart is beating, that the attempts at inspiration are indisputable, and that the child is separate from the mother. On the other hand, if this occurred, a child wholly born might be murdered, and yet there would be no evidence of inflation of the lungs to prove its live birth.

Cases relative to the rapidity of the decomposition of the fœtus and placenta in utero.

Fœtus.—With regard to the rapidity of decomposition of the fœtus, I imagine that much depends—1st, upon whether the child has been already dead in utero before labour commenced; 2ndly, upon whether the child dies early or late in labour; 3rdly, and particularly, upon whether the membranes rupture early or late.

Now, the most rapid decomposition ensues when the child has been dead some time before the commencement of labour. It is not necessary that putridity should have commenced before the escape of the liquor amnii, but merely that the child should have been dead some days. In one such case I have known putridity established within twelve hours.

It is, of course, well known that a dead child may remain within the uterus undecomposed for some months if the membranes have not been broken.

On the other hand, in those cases in which the evidence has certainly shown that the child was alive within twenty-four hours of its birth, and yet that it was born more or less putrid, we have a clear proof that within that period decomposition of the fœtus can be set up.

These instances do not frequently occur, but I have met with at least three which may be relied upon. In all of them the membranes had been ruptured for some time.

I may here mention a fact which, perhaps, scarcely belongs strictly to the subject, though it has an indirect bearing on it. It is that the presence of offensive discharges before birth is not to be relied upon as absolute evidence of death of the fœtus.

I have myself known two instances of this kind. In each of them well-marked putridity of the discharges existed before the birth of the child, yet in one case the child was born alive and strong. In the other case the operation of craniotomy was performed in full confidence that the fœtus was dead, but after extraction there was a distinct movement of the child from the medulla not having been broken up.

Placenta.—I was called to deliver a placenta which had been left in the uterus and vagina. *Twelve* hours only had elapsed since the delivery of a live child, and yet the placenta was highly putrid.

I have, however, known it remain for some days in the uterus without decomposing to any great extent, and this is particularly the case if the placenta is firmly united to the uterine walls.

In those cases which have come under my own observation I have generally noticed that decomposition of the placenta has begun at the end of twenty-four hours. Outside the body the placenta will, in a moderately cool summer, commence to decompose two days after delivery. In very hot weather it may do so in twenty-four hours, but this is rare. In a temperature of about 50° Fahr. decomposition would set in after three days; some retarding influence in this respect would be exerted by exposure to dry currents of air.

Decomposition of both fœtus and placenta, doubtless, runs on much more quickly within the body of the mother than without, for warmth and moisture largely favour it. But certain conditions seem necessary to this end, these being the expansion of the os uteri and the rupture of the membranes.

CASES

OF

ACUTE RHEUMATISM TREATED BY LEMON-JUICE;

WITH REMARKS.

By G. OWEN REES, M.D., F.R.S.

On a former occasion, with the able assistance of Dr. Sutton, I published a few cases of acute rheumatism which had been treated by mint water, or, in other words, had received no treatment at all. The result obtained was very satisfactory, the patients recovering as rapidly and progressing in every respect as favorably under this expectant plan as upon most of those which have recently been adopted, and certainly doing far better than when subjected to the severe treatment in fashion some thirty years ago. When remarking upon those cases I stated my belief that had lemon-juice been prescribed the pain in several instances would have been relieved earlier, and the duration of symptoms probably curtailed. That opinion is strengthened by the cases here recorded, in which the average duration of the disease in the uncomplicated cases was 6·8 days.

If we take the uncomplicated cases treated upon the *expectant* plan, the average duration is 8·5 days. It is much to be regretted that we have not had the opportunity on this occasion of recording more cases of lemon-juice treatment, but it so happens we have had but few rheumatic patients admitted into our hospital during the past year, and I have not chosen to refer to old reports, which cannot pretend to the accuracy here attained. These old reports, however, taken for what they may be worth, are very favorable to the lemon-juice treatment, and show much the same results recorded many years ago, when I published a pamphlet on the subject.

A good deal of importance has naturally been attached to determining how far any given plan of treatment may be influential in preventing the occurrence of complications, more especially those of the heart-membranes. On this point I can only say that such complications scarcely ever occur during the lemon-juice treatment, and that when present in the cases they have existed before the exhibition of the remedy.

I do not consider, however, that this statement means very much in favour of lemon-juice, for I am persuaded that complications are not very common after patients are bedded and well cared for, whatever may be the plan of treatment adopted. When complications are present, however, I know no internal remedy that assists the patient so well as the free use of the juice. With regard to the existence or non-existence of complications in our hospital cases, *on admission*, we may be sometimes so placed that we cannot get at the fact. The hospital physician, perhaps, may not visit his cases on the day of admission, and then he may have to trust to the report of a senior student, his clerk, who is only commencing bed-side practice. Heart murmurs may be thus unheard and unappreciated, and on the physician's visit, twenty-four or thirty-six hours after any given remedy has been administered, may be detected by the more practised ear, and registered as occurring for the first time at that period after admission. Again, the observer who saw the patient at first may have had to deal with a pericardium full of fluid, and, consequently, no pericardial rub may have met the ear. Such cases are often reported as free from heart complication; and when a change occurs in the case, consisting in the absorption of the fluid, the pericardial rub may be observed at some considerable period after admission, and registered as the effect of pericarditis occurring at that date.

I have had frequent experience of the above error. Thus, when I have visited the case on the day of admission I have then been told that the heart was all right, and, certainly, no bruit nor rub could be heard; but the troubled action of heart, the increased area of cardiac dulness, and the dyspnœa, at once pointed to the existence of pericarditis, probably of some standing. Here, of course, the occurrence of a pericardial rub on absorption of the fluid may be foretold, and an instructive lesson afforded to the student. It is when our clerks are about leaving us that their reports begin to assist us, and we cannot expect more from those who are just beginning to observe; at least, that is my conviction, notwithstanding that I have had the advantage of dealing with the intelligent and industrious class of Guy's school.

It is owing to these conditions that I have availed myself of the daily attention of Dr. Sutton, in whose skill I have the greatest confidence. I expressed an opinion many years ago that the more acute the symptoms the greater the value of the lemon-juice treatment in rheumatism. From the experience I have now had, however, of the expectant treatment, I am almost certain that the very nature of the disease is such that in its most acute forms its duration is the shortest, though we must still allow, unfortunately, that some plans of treatment interfere with this desirable result being obtained.

Case 1.—Joseph H—, æt. 23, admitted, May 3rd, into bed No. 20, Stephen Ward, under the care of Dr. Owen Rees. He complains of pain in all his limbs; he is sweating freely; the rheumatic odour is well marked; his tongue is furred. He says he has had rheumatic fever before, and that he was in a hospital at Birmingham, where he was treated with blisters. Sumat Succi Limonis ℥ij ter die.

5th.—He is suffering great pain. Rept. Succus Limonis; Pulv. Opii gr. j o. n.

6th.—He has great pain in his wrists and shoulders; he is sweating very freely; no bruit; tongue coated.

7th.—Pain in the right shoulder, &c.; he says that he is in "a good deal of pain;" pulse 92; he is perspiring very freely; tongue clean. No increase in the area of cardiac dulness; no bruit. He states that he feels better to-day, and that he passed a good night. Urine 18 oz., of sp. gr. 1031, high coloured and acid.

12th.—He has been in much the same condition; he says he cannot raise his right arm, his shoulder is so painful; the arm is wrapped in cotton wool; the skin is cool and moist, not now perspiring, but he perspired a good deal in the night; tongue clean and moist; pulse 96. There is no increase in the area of cardiac dulness; the first sound is short, and feebly heard, the second unduly marked. The urine is clear, of normal colour, sp. gr. 1022.

13th.—Free from pain; doing well.

14th.—He is in no pain; tongue clean; skin cool; pulse 80; no bruit. Urine pale, clear, sp. gr. 1012; 19 oz. have been passed in twelve hours.

18th.—Doing well.

Case 2.—Jane S—, æt. 23, admitted March 12th, 1866, into No. 4, Esther Ward, under Dr. Owen Rees. She says that she has enjoyed good health, and has never had any serious illness, but has suffered at times with sore throat. This is her first attack of rheumatic fever.

On March 8th she was suddenly seized with pain in her back. Her ankles and feet became so painful that she could not stand. On the day of admission she had pain in both her ankles and in the right wrist. She was sweating freely.

13th.—She has very much pain in the right wrist, which is red, somewhat swollen, and very tender on pressure. She has pain in both her ankles. She is perspiring freely. The area of cardiac dulness is rather increased; a systolic creaking bruit is heard over the left third costal cartilage.

Ordered Succi Limonis ℥iij 4tis horis. Low diet.

14th.—The right wrist is very red and very tender, but not swollen. She is in rather more pain to-day in the foot, but less pain in the hand than yesterday. The left ankle is swollen. She says she feels better. The tongue is thickly coated with whitish-yellow fur. She is perspiring very freely. The perspiration smells sour, and turns litmus paper very red. Pulse 112. Heart—The apex beat is very distinctly felt a little below and a little to the right of the nipple; it is unduly marked. The rhythm is regular. The first sound is feebly heard over the apex, and a prolonged, murmuring, first sound is heard over the base. No creaking sound is heard to-day. No increase in the area of cardiac dulness. Cheeks flushed; thirst; no appetite. Cough frequent and hacking.

15th.—Her condition is much the same. She is still in pain, and perspiring freely. There is a faint systolic bruit at the base.

17th.—She is in no pain, and expresses herself as feeling much better. The first sound over the apex is rather prolonged.

19th.—She is in no pain, and feels much better.

20th.—She is in no pain. Succi Limonis ℥ij bis die. Middle diet. The cardiac dulness commences under the third rib. There is a slight systolic bruit at the base. The first sound at the apex is prolonged. She does not perspire much now. Rept. mist.

22nd.—She is in no pain, but had pain in her chest yesterday. Skin cool; appetite good; tongue clean. No perspiration now. The first sound over the apex is still prolonged; a systolic bruit exists over the base.

24th.—Going on well.

26th.—No pain in the limbs. She says that in the night she had pain in the chest, about the left breast, which kept her awake for some time. Heart—a faint murmur is heard over the base; the first sound at the apex is prolonged, but not so markedly as a few days ago.

31st.—She says, "My legs swell when I get up;" otherwise she appears well. The first sound is still prolonged, but not so much as it was some days ago.

She had no return of the symptoms.

Case 3.—Eliza B—, æt. 16, admitted May 24th, 1866 into No. 10 bed, Esther Ward, under Dr. Owen Rees. She says that three days before admission she was suddenly seized with pain in the little finger and in the inside of the left hand. The pain then extended to several joints. She went to bed, and next day was in such pain in her legs that she could not stand. On admission the pain was chiefly in the legs and ankles.

26th.—She complains of pain in both ankles and knees, and says she perspires much, especially about her back. The skin is cool, and not perspiring now. The tongue is moist, and slightly covered with white fur. Face natural. Pulse 88, very compressible. The ankles and knees are not swollen. The apex beat of the heart is unduly marked under the left nipple; the area of dulness is not increased; the first sound over the apex is sharp and short; there is no bruit. Ordered Succus Limonis. The urine is pale and clear, its sp. gr. 1014; she passed 34½ oz. in twenty-four hours.

30th.—She is free from pain, and has been so since the 27th inst. There is no bruit.

Case 4.—Alice M—, æt. 15, admitted into No. 9, Esther Ward, May 30th, 1866, under Dr. Owen Rees. She says that for about a fortnight she has been suffering pain, but not so much as at present. This is the third attack of rheumatic fever.

May 31st.—She has very severe pain in both ankles, and over both there is a blush of redness. She appears in very great pain and very much distressed by it; she is very restless, throwing her arms and head about; she says she cannot move her legs. Pulse 128. Tongue moist, coated with white fur. Face flushed; skin perspiring about the hands, but not much. A marked rheumatic odour. Heart—the area of dulness is normal; a faint systolic bruit is heard over the apex, but it is heard so faintly that its existence might be doubted; rhythm irregular once in eight or nine beats. Urine turbid, loaded with lithates; reaction acid, sp. gr. 1032; no albumen.

℞ Succi Limonis ℥iij 6tis horis. Low diet.

June 2nd.—She still has pain in her legs, but it is not so severe. She also has very great pain in each wrist. The right wrist is swollen and very red. She is sweating profusely. There is a strongly marked rheumatic odour. Tongue thickly coated with white fur. Bowels constipated. Pulse 120. Heart irregular in rhythm; systolic bruit; the area of superficial cardiac dulness extends as high as the upper margin of the third rib. She says, "I have had great pain all over the front of my chest, like a heavy weight, since yesterday." The Sister of the Ward remarks that she has complained very much of the pain in the chest. Urine turbid, high coloured, sp. gr.

1027, acid reaction. Pulv. Cal. et Rhei stat. Rept. Succus Limonis.

3rd.—She says that the pain in her chest has all gone, but her wrists are very tender, they are also red and swollen; the skin feels very hot and dry; the tongue is thickly coated; respiration 28—32. Pulse 116, feeble and irregular. The heart is very irregular; a well-marked systolic bruit is heard all over the region of the heart. The urine is very high coloured, of an acid reaction, sp. gr. 1032.

4th.—She has no pain in her chest; her skin feels hot; her tongue is coated; there is pain and swelling of the back of the hand; a systolic bruit is heard over the heart, of which the rhythm is irregular.

5th.—Says that she feels better, but is still in pain. Her skin is hot, but not at present perspiring; she states that she perspires very much at times. Tongue coated; pulse 120. Heart—no marked increase in the area of cardiac dulness; rhythm irregular; murmur at the heart scarcely audible. Urine loaded with lithates, scanty in quantity, of sp. gr. 1033. Bowels open.

7th.—She has great pain in her wrists, and also pain in her limbs, but she is not in so much pain as she has been. Tongue clean and moist; pulse 94. The cardiac dulness reaches as high as the upper margin of the third rib; a systolic bruit is still heard over the base as well as over the apex. She passed 25 oz. of urine in twenty-four hours; it is of sp. gr. 1029, high coloured, and acid.

12th.—She has been in much the same state, and is still in pain. Pulse 80; tongue clean; appetite bad; heart in much the same condition as at last report.

14th.—She is free from pain; her tongue is clean, and the skin cool. Pulse 100; a murmur is heard over the apex of the heart and also over the left scapula. The urine is straw coloured, of an acid reaction; 21 oz. were passed in seven hours; its sp. gr. is 1024.

16th.—She says she feels very much better and is free from pain. Pulse 98; skin cool.

17th.—She is out of bed.

19th.—She complains of no pain except of a little in her back; tongue clean; skin cool. Pulse 92; appetite returning; condition of the heart much the same.

21st.—She has been out of bed since the 17th, and complains of no pain except a little in the right hand; her appetite is good; skin cool; tongue clean. Pulse 96; systolic bruit over the region of the heart. Urine of natural colour, and of sp. gr. 1011; 40 oz. passed in twenty-four hours.

She had a relapse; remained a week in bed, and then recovered.

Case. 5.—Ann M—, æt. 27, married, was admitted April 14th, 1866, under the care of Dr. Owen Rees. She states that she has generally enjoyed good health, but has never been very strong; this is her first attack of rheumatic fever. Two months ago she was confined; she recovered well; she thought herself in good health until she went into her new lodgings, where a fire had not been lighted for some time, and the bricklayers had been engaged repairing and whitewashing the rooms. She moved on April 8th, and next day she did not feel well. She says, "On the 10th I had pain in my ankle very bad, but I had it well rubbed, which relieved me. On the morning of the 12th I had pain in my left foot and also in my knees, which continued and gradually became much worse."

April 5th.—The left ankle is rather swollen and red; she complains of great pain in it. A systolic bruit is heard faintly near the base.

16th.—She has great pain in both ankles, which are red and swollen. She says, "I feel pain all over my knees and ankles, so that I cannot move." Tongue slightly coated with white fur. No increase in the area of cardiac dulness; a systolic bruit is heard at the base. Her skin is moist; she says she sweats freely; pulse 112.

17th.—She has much less pain in her wrists, but has pain in both her ankles and there is great redness on the inside of each; but they are not swollen. Pulse 112. Urine turbid and very scanty, of sp. gr. 1037, and of acid reaction. A slight systolic bruit exists at the base of the heart; the tongue presents the same appearance as yesterday.

18th.—She says that she is in much less pain to-day. Pulse 100—104, full and bounding; tongue moist, coated at its base with yellow fur. She did not perspire so much last night. Skin moist. The area of dulness over the heart is not increased; the first sound at the base is prolonged, and at the apex also. Urine scanty in quantity, of sp. gr. 1037.

19th.—She says, "I am a great deal better; in no pain while I am still, but when I move I have pain in my knees and shoulders." The tongue is moist and clean; the left wrist is red, and slightly swollen; she perspired a good deal last night, and is perspiring a little now; no appetite; pulse 100, full. First sound of heart prolonged over the base. Urine very high coloured, of a very deep red colour, not albuminous; sp. gr. 1038; 30 oz. passed in twenty-four hours. Bowels open.

20th.—She says she feels better and is in much less pain; she can move all her limbs; she did not perspire near so much last night, and slept better. Pulse 96; tongue clean. A prolonged systolic bruit is heard all over the base of the heart, and a prolonged first sound over the apex; apex beat normal. Urine could not be collected; bowels open.

21st.—She says she is in much more pain to-day than yesterday. Pulse 100; she has pain in both arms; the tongue is slightly coloured with yellow fur; she is perspiring freely; says she "did not sleep much last night." Urine slightly acid, of sp. gr. 1032; 19 oz. passed in seventeen hours.

℞ Pulv. Doveri gr. v h. s. ℞ Suc. Limonis ℥ij 4tis h.

22nd.—She still has pain in her shoulder, but says she is not suffering so much as yesterday; the skin is rather hot; she perspired freely during the night; tongue moist and almost clean. Area of dulness over heart not increased; first sound prolonged all over both apex and base; at the base it might be considered a faint systolic bruit. Urine acid, very high coloured, turbid, sp. gr. 1035; passed 16 oz. in sixteen hours. Bowels not relieved since the 20th.

25th.—Pain in both wrists; she says she perspired very much last night. Her manner is rather excited; she says that her "back aches dreadfully;" she wishes to sit up in bed; pulse 108, rather feeble. The first sound of the heart, over the base, is heard markedly prolonged; it is also prolonged over the apex. Tongue moist, slightly furred. Urine, 17 oz. passed in twenty-six hours; acid, very high coloured, turbid, sp. gr. 1032.

26th.—She is not in so much pain and can move all her limbs. She perspired a good deal last night; pulse 112, feeble. A systolic bruit is heard at the base; the first sound is prolonged over the apex; the superficial cardiac dulness reaches as high as the lower margin of the third rib. Urine scanty in quantity, high coloured, but not nearly so much so as it has been; acid reaction; sp. gr. 1032.

27th.—Dr. Rees ordered middle diet.

28th.—Still in pain, but not so severe. She is suffering from retention of urine; she suffered in the same way after her confinement.

29th.—Urine has been drawn off, the quantity being 15 oz. in twenty-four hours; its sp. gr. is 1031; it is acid, high coloured, and clear; it becomes like milk on adding nitric acid, and clears on boiling. The superficial cardiac dulness reaches as high as the third rib; there is no bruit; the first sound is not so prolonged. There is pain in the right hand and shoulder; the skin is not perspiring now, but she says she does perspire; the appetite is pretty good.

May 1st. — She says she feels much better and is nearly free from pain; tongue clean; skin cool; pulse 88; appetite pretty good. Heart tranquil; a faint, prolonged, first sound over the base; dulness normal. Urine turbid, acid; sp. gr. 1033.

To avoid repetition, we may state that the patient continued steadily to convalesce from 1st May. On the 10th it is noted she is out of bed, free from pain, and going on very well.

12th.—She is free from pain; tongue clean; skin cool; pulse 92. Heart's dulness not increased; no bruit to be heard. Appetite very good. Rept. Succus Limonis ter die. Middle diet.

17th.—She is going on well and walking about the ward. Urine, sp. gr. 1026; about 15 oz. passed.

19th.—She went out well.

Case 6.—Emma G—, æt. 24, widow, admitted July 11th, 1866, under the care of Dr. Owen Rees. She says this is her first attack. She was taken ill fourteen days before admission. Before that time she had enjoyed very good health.

The attacks came on with great pain in the right ankle and leg, and the pain gradually extended all over her. She went to bed immediately she was taken ill, and had medical attendance at home, and took medicine.

July 12th.—She says she has pain all over her; greatest pain in the knees and ankles, not so much pain in the wrists or elbows; she can move her arms; she cannot turn herself in bed. Tongue moist, coated in the middle with yellow fur. She is sweating freely; pulse 104. Heart's dulness extends as high as the third rib; a systolic bruit at the base. Urine acid in reaction, clear; sp. gr. 1024.

Succi Limonis ℥iij 6tis horis. Low diet.

14th.—She says she is not in so much pain, and is able to turn herself over in bed on to her right side. Skin cool and moist, and not at present perspiring a great deal, but she states that she sweats a great deal at times. Pulse 104. The cardiac dulness reaches as high as the upper margin of the third rib; the apex beat is very indistinctly felt; a systolic bruit exists at the base. A prolonged first sound is heard at the apex, which is more and more prolonged as the stethoscope is carried towards the base of the heart. Urine, 68 oz. passed in twenty-four hours; sp. gr. 1023; it is rather high coloured, clear, and contains no albumen. Tongue cleaner.

16th.—She is not in so much pain.

17th.—She complains of no pain anywhere except of a little in the left arm, but she can move the arm; she says, however, that it hurts her when she does so. Tongue clean, moist. Pulse 84. Skin cool; no perspiration; says she does not perspire so much as she did. Heart dulness diminished; a loud systolic bruit at the base. Urine acid, no albumen, sp. gr. 1020.

18th.—She is entirely free from pain. Skin cool. Pulse 84. Tongue cleaner. A prolonged first sound exists at the apex of the heart, and a systolic bruit at the base. Urine acid, of specific gravity 1020; 25½ oz. passed in twelve hours.

19th.—She is free from pain and going on well. Urine acid, a little flocculent, otherwise clear; sp. gr. 1019.

21st.—She is free from pain. Tongue clean; pulse natural. Urine clear, acid, of sp. gr. 1022. Appetite very good.

26th.—She has been free from pain since the 21st. She says she feels much better. Skin cool; tongue clean; appetite good; pulse 80. Heart—A systolic bruit carried along the aorta, not heard over the apex. Urine flocculent, acid in reaction, of the natural colour, and of sp. gr. 1020. She has been out of bed.

30th.—Went out of the hospital well.

Case 7.—Emma H—, æt. 29, married, admitted April 18th, Esther Ward, under Dr. Owen Rees. She has had three children, but has not had one for six years. She has generally enjoyed very good health, and except during her confinement has never been laid up with any illness.

On April 8th she was seized with pain in the left foot, extending up her leg. Three days afterwards she had pain in her right leg, and has had more or less pain ever since. She says that this is her first attack of rheumatic fever. The urine is acid, dark-coloured, sp. gr. 1025.

18th.—She says she has pain in the right ankle, which is a little swollen; there is an extensive blush of redness on the outer side of the joint; it is tender when pressed; she has also great pain in her left knee and hip; she sweats very much when asleep; tongue coated with yellow fur; no appetite. Pulse 104, jerking, but not full. Heart—no increased area of dulness; no bruit; second sound over the base unduly marked; first sound rather prolonged over the apex. Skin moist.

19th.—Great pain in the right shoulder and left elbow, also pain, redness, and tenderness of the left ankle. Pulse 104, rather fuller. Apex beat of the heart very feeble; first sound over the base and apex very feeble. She says she slept very little last night. The tongue is moist, and coated with yellow fur; she is sweating very freely; the rheumatic odour is well marked. She is menstruating. The bowels are confined.

Succi Limonis ℥ij 4tis horis. Cal. gr. iii, Pulv. Rhei gr. xx statim. Low diet.

20th.—She says she feels better, and is not in quite so much pain; she still has pain in her right shoulder and in her legs, but is able to move them. Tongue coated with thick yellow fur, its papillæ red and prominent. She perspired very freely last night. Pulse 88, full; skin not very hot; no redness over the ankles to-day. First sound of heart feeble, second sound very sharp; no bruit. She is still menstruating.

21st.—The pain is much the same. Skin perspiring; pulse 100; tongue coated; condition of the heart the same.

22nd.—She has less pain in her arm, but her feet are very painful; her tongue is furred; she is perspiring freely; state of the heart much the same. Urine acid; 35 oz. passed in sixteen hours; sp. gr. 1025, no albumen.

25th.—She says she has a little pain in her ankles; appears to be doing well. First sound over the apex of the heart rather prolonged; no bruit. Urine acid; 11 oz. in eight hours; sp. gr. 1027, colour natural.

26th.—She is in no pain. Tongue clean. Urine pale, slightly turbid, acid reaction, sp. gr. 1020; 23 oz. passed in seventeen hours.

29th.—She has been free from pain since the 26th. She got out of bed on 28th. She was on that day ordered middle diet, and to take the lemon-juice twice a day only. Bowels regular; tongue almost clean; appetite very good. She states that she does not perspire much now. Heart's action tranquil; first sound faintly prolonged over the base and over the apex, second sound very sharp.

May 1st.—Apparently well; out of bed and walking about. Urine acid, pale, sp. gr. 1019; 27 oz. passed in eighteen hours. On careful examination of the heart no bruit is heard. The heart apparently healthy.

9th.—Went out of the hospital well.

Case 8.—Sarah P—, æt 17, admitted into Esther Ward, April 10th, under the care of Dr. Owen Rees, No. 1 bed. She says that she enjoyed very good health up to last Christmas. A few days after Christmas Day she was seized with pain in both legs, which lasted about two weeks; she afterwards had pain in her elbows; was at that time laid up about a month; afterwards she improved and was able to go about again, although not entirely free from pain. She further proceeded to remark that she was not "about" a week before she was again seized with pain in both knees, and she has since now and then had one or the other knee affected. She has had no medical attendance during her illness. She has pain in both ankles, which are rather swollen. No bruit is heard over the heart.

12th.—She still suffers from pain in both ankles, and pain and swelling in the right wrist. She was ordered "Succi Limonis ℥ij 6tis horis et Empl. Lyttæ sterno." She complains of pain in her chest.

14th.—Her condition is much the same.

15th.—Pulse 72; skin cool; tongue rather white, not much furred; a little pain in the ankles; sweats freely during sleep. There is no increase in the area of the cardiac dulness; the first sound is feeble and short, the second very sharp; there is no bruit. Her appetite is good; she says she feels better than she did when she came in; there is no swelling, but tenderness in the ankles on pressure.

17th.—She is in less pain; heart appears in the same state.

18th.—The pain is almost gone; she has a very little in both her knees, but nothing to speak of; tongue clean and white; she did not perspire much last night; skin cool; not perspiring at present. Heart's action quiet; first sound prolonged over both the base and apex, second sound very sharp; no bruit. Pulse 72. Urine acid and pale, of sp. gr. 1016; 18 oz. passed in about seventeen hours.

19th.—She is free from pain, walking about the ward. Urine pale and acid; bowels rather relaxed. She went out of hospital quite unexpectedly on April 20th, 1866.

Case 9.—George H—, æt. 24, admitted May 30th, 1866, into Philip Ward, under the care of Dr. Owen Rees. Some days ago, while going to work, he was seized with pain in his legs. He says, "It was in the sinews of my knees." He was obliged to turn back and go to bed. He states, "While in bed the pain worked all over my body, and flew from one part to another." Some years ago he had rheumatic fever, and was laid up for thirteen weeks; he says he has enjoyed good health with the exception of that attack—"no one better than I did; always good." "I drank a good deal of beer in my day, and now and then, on a Saturday night, a little drop too much." "When the sun is out sometimes my nose bursts out bleeding twice or three times a week; sometimes it will stop, and then not bleed at all. Father had rheumatic fever when twenty-five or twenty-six years old. Otherwise healthy, and drank a good deal."

His legs swell. His height is five feet eight inches; weight eleven and a half stone; he has a well-developed skeleton; his hair is curly and inclined to be thick; his features are regular; his nose is rather thick; his teeth are regular and sound in front, though much worn in the lower jaw; behind they are decayed. His mother had rheumatic fever and his sister has been in the hospital ward with rheumatic fever.

The cardiac dulness extends as high as the upper margin of the third rib, to the right of the right margin of the sternum, one eighth of an inch to the left of the left nipple, and one and a half below the left nipple. The apex beat is indistinct; a faint systolic beat is heard all over the cardiac region, and is very distinct over the apex of the heart, but not much more so there than elsewhere. He is sweating very freely about the limbs; the ankles are not swollen; the right wrist is painful, swollen, and red. The urine passed in the night and this morning, in nineteen hours, is turbid, coated with lithates, of an acid reaction; sp. gr. 1027; quantity in those nineteen hours, 29½ oz., about 37 oz. in twenty-four hours.

June 1st.—Passed about a pint (20 oz. imperial) of urine in sixteen to seventeen hours. He is in very great pain in his hands, and perspires profusely.

2nd.—The nurse says he perspired most profusely last night and this morning, so she gave him a bath. His hands are very painful and much swollen. Urine, 16 oz. passed during the night; it is of an acid reaction and of sp. gr. 1027. Heart appears in much the same state.

3rd.—He says he is much better. He has pain in both his hands, and says he has no use of the right arm; pulse 80; skin cool; tongue moist and still thickly coated with yellow fur; he did not sweat so much last night. Heart's dulness extends up to the lower margin of the third rib, and one eighth of an inch to the left of the left nipple; the systolic bruit at the apex is well marked. Urine, 20 oz. passed from 6 p.m. to 6 a.m. of the night of the 2nd; on the night of the 3rd, 6 p.m. to 6 a.m., 30 oz.

5th.—Urine, 24 oz. passed in twelve hours, from 6 p.m. to 6 a.m.; 16 oz. passed at 3 p.m., in my presence; sp. gr. 1014.

6th.—30 oz. of urine passed from 6 p.m. to 6 a.m.; sp. gr. 1015. He is much better, being nearly entirely free from pain; he can freely move all his joints.

7th.—30 oz. of urine passed from 6 p.m. to 6 a.m.; sp. gr. 1015.

9th.—He is free from pain and convalescent. Urine of sp. gr. 1017, and of an acid reaction. Systolic bruit at the apex of the heart; skin is cool.

11th.—He is out of bed and is going on well.

12th —He is going on well. Urine of an acid reaction, clear, sp. gr. 1018, no albumen.

14th.—He is in bed again; he says he went to bed for he had so much pain in his right knee when he stood; he has also pain in his right hand; pulse 92. Urine of an acid reaction, sp. gr. 1023.

15th.—22 oz. urine passed in twelve hours.

16th.—40 oz. urine passed in twenty-four hours. He is again out of bed. Dr. Rees considers this a relapse. The patient says he still has pain in his knees. Urine of an acid reaction, clear, sp. gr. 1025.

21st.—Urine, sp. gr. 1013. He appears to be going on well; is free from pain and walking about.

Postscript.—Since the above was written I have seen some strange remarks referring to the expectant treatment which we have been trying in Guy's Hospital. The writer concludes that because we have shown the uselessness of many remedies in vogue, we must have arrived at the belief that the best plan of treatment in rheumatism is to do nothing for our patients. I leave the writer and his logic to the mercy of the profession.

SELECT CLINICAL REPORTS.

(MEMOIR V.)

By G. H. BARLOW, M.A., M.D.

There are few practical questions upon which, as it appears to me, there has been greater diversity of opinion amongst medical men than that of the treatment of acute rheumatism. I do not at present allude so much to the now obsolete practice of indiscriminate bleeding and calomel, as to the later modes of treatment by chemical action, by elimination, and by merely expectant treatment. I am not prepared to say whether chemical remedies produce their effect by the decomposition or neutralization of the peculiar acid present in the blood in rheumatism, but it is nevertheless true that the most successful of these, the lemon-juice, contains a salt of potass, which may be an eliminant, and that citric acid in its uncombined state has not the same efficiency. The occasional success of the expectant treatment, or the giving no active medicine whatever, has been regarded by some as a proof of the inutility of all the other modes of treatment, and by some as an argument against the use of drugs in the treatment of disease in general. What, however, I would urge is that no one of these modes of treatment is most applicable to all cases, and that the success of the expectant treatment only shows that rheumatic fever, like all acute disease, is capable of spontaneous recovery; and that where this recovery is proceeding steadily and safely the officious use of drugs is to be avoided, and that the skill of the experienced practitioner never appears to greater advantage than in the discrimination of the cases which do not require his interference; but that, on the other hand, many cases will not proceed thus favorably if left to themselves, and in such instances there is occasion for the use, not of a specific remedy, for as yet we know of none such for rheumatism, but of medicines adapted to the exigencies of the particular case.

CASE 1.—*Acute rheumatism of ordinary severity, treated with moderate doses of lemon-juice, and afterwards of bicarbonate of potash; convalescence within three weeks from the first commencement of the disease.*

Thomas S—, living at 48, West Lane, Rotherhithe, admitted into the Clinical Ward, under Dr. Barlow, October 5th, 1864. Clinical clerk, Mr. Rendle.

History.—General health good before present attack, with exception of inflammation of the chest, as he says, five years ago; no rheumatic tendency in the family. He has been in the habit of getting intoxicated on Saturday nights; no history of gonorrhœa. Traces his present illness to getting wet through four days before admission; states that the right knee became swollen and painful first, and then the left, the right getting better meanwhile. Other joints affected in the following order—right shoulder, both wrists, left shoulder. Bowels open daily.

On admission.—Both wrists swollen, the right rather the worse, and the right shoulder is painful; but the weight of the bedclothes can be borne without causing pain. Ordered Mist. Senn. co. c. Tinct. Colchici ♏ xxv statim; Succi Limonis ℥ij 6tis horis.

October 6th.—Pulse 80, weak; tongue moist, slightly furred in centre and at the sides, red at tip; bowels acted twice in the night and once in the morning. Systolic murmur with the first sound of the heart, which appears muffled in comparison with the second, which is sharp. Urine scanty and high coloured.

7th.—Patient says he is better; swelling quite gone from the left wrist; the right is better, and both shoulders are painless. Tongue creamy in the centre; bowels unopened since yesterday morning; pulse 70. Urine neutral, dark straw coloured, and non-albuminous.

8th.—Better; right wrist less swollen, not so painful; tongue cleaner, slightly dry; bowels open once in the morning; urine clearer and lighter; pulse 66. Murmur (systolic) heard in the course of the aorta, not in the axilla or at the back.

9th.—Better; pulse 64; tongue as yesterday.

10th.—Pain in right wrist only; none in the chest. Heart sounds not so distinct; faint systolic murmur below and to the right of left nipple, not so much over aorta. Ordered Potass. Bicarb., Sp. Ammon. Ar., ex Jul., Menth. Pip, t. d. s.

11th.—Pulse 56, irregular; tongue clean; no pains anywhere; bowels open daily.

12th.—Pulse 56; pain in right shoulder, especially at night; heart sounds irregular. To continue Mist. Pot. Bic., &c., and to have Vin. Xer. ℥iv.

13th.—Pulse 48, less irregular; no pain last night; tongue clean; cannot hear the murmur now (?); bowels act every day once.

14th.—Pulse 50; tongue clean; pain in right shoulder returned. Distinct systolic murmur heard below the nipple at the apex; sounds are good to the right of the sternum; second sound good up the aorta. Ordered Inf. Cinchon. vice Julep. Menth. Pip. (vide die 10), and a chop.

15th.—Pulse 54; tongue clean; no pain in shoulder.

17th.—Pulse 50; tongue clean. Systolic murmur heard below and to right of nipple, and also towards the axilla, but not behind; less distinct in course of the aorta.

19th.—Pulse 64; bowels open; bruit still heard.

20th.—Pulse 68; tongue clean; bowels open regularly. Rep. Mist. Rep. Vinum. Middle diet.

22nd.—Improving; bruit still heard.

24th.—Is dressed this morning and sitting by the fire.

27th.—Getting stronger; complains of slight aching pains in legs and back, not in joints.

28th.—Improving; bruit not heard to-day.

November 2nd.—Ditto. To have full diet.

4th.—Presented; discharged cured.

CASE 2.—Gilbert C—, æt. 15, admitted October 18th, 1864, into Clinical Ward, under Dr. Barlow; clinical clerk, Mr. E. F. Turner.

He is a fair lad. He began to complain three weeks ago last Sunday of pain in his legs; a day or two afterwards his knees and feet became swollen and very painful, and since then his symptoms have varied a good deal; sometimes he has seemed better and sometimes worse. He has been under medical treatment at Sydenham. When nine years of age he had scarlet fever, but there is no evidence of other illness.

19th.—Face pale and slightly bedewed with perspiration; he lies on his back in bed and cannot move without severe pain in his left hip-joint; the left knee is a little tender, as are also both wrists and the left shoulder; the skin of the hands looks sodden and feels moist; his neck is quite stiff. Impulse of heart not distinctly seen; heart sounds quick and irritable, but not abnormal; præcordial dulness normal. Pulse 124, full, and slightly incompressible. Ordered on the 18th Pulv. Doveri gr v h. s. s. 19th, Pot. Bicarb. gr. x ex J. M. P., 4tis h. s.

20th.—Has passed a bad night in consequence of severe pain in the left shoulder, and in the left side and back of his head, and it is very painful for him to move his head. The left sterno-clavicular articulation is distinctly swollen and intensely tender; appetite bad; tongue coated with a yellowish fur; complains of thirst and slight dysphagia; no sickness; bowels open yesterday, but not as yet to-day (11 a.m.). Pulse 112; heart sounds normal.

21st.—Still great pain in the hip and shoulder, but the sterno-clavicular articulation is quite free to-day. Bowels open this morning; he slept badly. Urine normal colour, acid; traces of phosphate; stains of purpurine on the vessel. Aug. dos. Pot. Bicarb. ad gr. xx. P.

22nd.—Pulse 104; heart unaffected; left hip much less tender; skin of hands smell sulphureous; tongue coated yellow; bowels open.

24th.—He complains now only of pain in the right shoulder, ·n move his head about without pain in the neck; friction murmur heard under the nipple and quite localised; it is only systolic, and increased by pressure. Ordered Emp. Canth. parvum regioni cordis.

25th.—Pulse soft and full; murmur perhaps rougher, but not extended; spots of miliaria appear in crops upon the hands and face. He sweats a good deal.

26th.—Rub continues the same; pains in joints less.

28th.—The friction sound, still only systolic, remains as before. Pulse full and compressible, 96; tongue still slightly furred yellow; appetite pretty good; bowels open; sleeps well; sweats less than he did. Middle diet.

31st.—Friction sounds the same.

November 1st.—Urina sanguinis, pale amber, acid; traces of phosphates, not albuminous; a cloud of mucus at the bottom of the vessel. Pulse full, 108; heart-sounds the same; can move all his limbs about now without pain; sleeps well; does not sweat; tongue clean; appetite good.

3rd.—Temp. 100°; pulse 116, full (11 a.m.).

4th.—Feels very well; has been wanting to rise; pericardial murmur harsh.

7th.—Got up for first time yesterday; temp. 99·4; pulse 120.

9th.—About the same.

12th.—Gets up each day. First sound with a very harsh murmur under and to the right of left nipple.

15th.—Gaining strength daily. Has a slight herpetic eruption about the columna nasi from a cold.

16th.—Went out well.

Case 3.—Patrick M—, æt. 25, admitted October 10th, 1865, into Clinical Ward, under Dr. Barlow; clinical clerk, Mr. J. H. Walters.

October 10th.—General health good; habits pretty temperate. On September 25th, while working in a shipbuilding yard, was attacked with pains in his limbs and loins. The next morning the pain was so severe that he was quite unable to move. His knees were first affected, being greatly swollen and painful; after a day or two this subsided, but soon returned again. The right wrist was next affected. On admission his right wrist was slightly reddened, swollen, and hot; his elbows, knees, ankles, and shoulders, were very painful, but not swollen. A systolic murmur to be heard over the base of the heart, and præcordial dulness normal. Ordered Jul. Menth. Pip. ter die.

11th.—Better; pulse 52.

12th.—Bruit more distinct in course of the aorta.

15th.—Is walking about the ward; says he feels quite well.

He continued improving till the evening of the 20th, when the joints again became painful and swollen. On going to bed he had a severe attack of shivering. Ordered Pot. Bicarb. gr. x. Sp. Ammon. co. ♏xx, ex Inf. Cinchon. ℥j, ter die, s.

21st.—Continues very poorly.

23rd.—Feeling about the same; bowels not open. Ordered Mist. Sennæ co. c. Vin. Colchici, ♏xxv. M. Ft. hst. Pot. Bicarb. ʒj, Acid. Citric. ℈j, ex Aq. ℥iss, ter die.

24th.—Bowels well opened; skin acting freely; pulse 66, good. Rub heard with first sound, but most distinctly to left of the nipple.

25th.—Complains of severe pains in shoulders and arms; urine acid.

26th.—Feeling better; pulse 60; tongue moist; slight pericardial rub heard over left nipple; urine alkaline.

30th.—Pericardial rub under axilla imperceptible; acute bruit still distinct. Adde sing. dos. Mist. Ferri Amm. Cit. gr. v.

31st.—Feeling much better; is up and walking about.

November 2nd.—Going on well.

3rd.—Had but very little sleep during the night, owing to severe pain across the chest.

4th.—Pain considerably relieved.

6th.—Complains of shooting pains round the chest, especially at night. Ordered Quinæ Sulph. gr. ij, Ext. Colch. Acetici gr. ss, Ext. Conii gr. iij, ter die.

13th.—Is feeling stronger. Still complains of flitting pains about the chest; murmur still heard in the course of the aorta; pulse small.

16th.—Pains in chest continue, also the aortic murmur. Ordered Pot. Bicarb. gr. x, Pot. Iodidi gr. j, ex Aq., ter die.

17th.—Feeling better.

21st.—Gradually gaining strength; pain in chest still rather troublesome at night.

25th.—Went out to-day, feeling very well. There is a slight systolic bruit to be heard about one inch internal to left nipple; it is, however, very faint, and seems to be obstructive.

Case 4.—John A—, æt. 30, a labourer, admitted October 23rd, 1863, into the Clinical Ward under Dr. Barlow. General health good; fair complexion; temperate habits.

History.—Says he has been suffering from cold some few weeks, but continued at his work until last week, when he noticed several of his joints were swollen, painful, and inflamed, especially knees and shoulders. Perspiration smells very sour. He passed a large quantity of water of a very high colour, and containing a sediment of a brick-red colour; his bowels were rather confined. On Sunday, October 18th, the pain was rather more severe in the ankle-joints, and they were inflamed, and his wrist also. One brother died of rheumatic fever a year ago; his father was subject to gout and rheumatism. He has had rheumatism once before, but not rheumatic fever. The rest of his family quite healthy. Has not had good health since he came to London, eleven years ago, but before that time was a very strong and healthy man.

On admission.—Is rather emaciated, and has lately lost flesh; skin very hot, perspiring freely; perspiration does not smell very acid; countenance very anxious and depressed, eyes dull; tongue coated white; pulse full and 120. Heart sounds very indistinct, and a slight rub heard just beneath the right nipple; increased cardiac dulness. Has no cough; bowels open every day; makes large quantities of water, which is of a clear straw colour, and contains a trace of albumen. All his joints inflamed and swollen, but refers the pain to his wrist and shoulders. Ordered Pot. Acet. ʒss, Pot. Nit. gr. x, Vin. Opii ♏v, ex Decoct. Hord. ℥ij. Pil. Dov. gr. x h. s. s.

25th.—Passed a very restless night; pulse 130; respiration 42; bowels relaxed and severe pain in the region of the heart. Pil. Dov. gr. v 6tis h. s. Mist. rep.

26th.—Very ill last night; severe pain in the cardiac region; says he could scarcely breathe at times. Mr. Stocker was called to see him, and ordered Pil. Dov. gr. v 4tis h. s. Countenance very anxious; tongue slightly coated; breathing short and quick, 40; pulse full, regular, 120; skin hot and perspiring, acid smell; bowels relaxed; takes very little nourishment, is very thirsty. Heart's action very rapid; sounds indistinct; a very distinct double rub to be heard at the base of the heart, and may be traced up as high as the end of the sternum. He complains of most pain just above the left nipple, and it is at that part the friction sounds are most distinct. He does not complain of pain in his joints. There is also a slight pleuritic rub. Emp. Canth. reg. cordis applicand. Pot. Bicarb. gr. xxv, Tr. Hyoscyami ♏xx, ex Decoct. Hord. ℥ij, ter die. P. pil. Dov.

27th.—The blister gave great relief, and he got a little sleep during the night. Pulse 100, rather intermittent; skin very hot and perspiring; bowels open once last night. Adde Pot. Bicarb. sing. dos. mist. Pil. Dov. gr. x h. s. s.

28th.—On the whole a little better; got no sleep during the night; there is still a very distinct pericardial rub. Urine very acid, contains a brick-red sediment of urates; clears on addition of heat and nitric acid; no albumen. Complains of severe pain in the shoulders and wrist-joints. Got more sleep last night; complains of a very severe lancinating pain on the right side. At times I fancy I can hear a slight pleuritic rub. Pulse 100; respiration 30. Adde Sod. Pot. Tart. ℈j, sing. dos. mist. Opii gr. j ter die.

29th.—Still complains of pains in shoulders and right side; slept very well last night; tongue coated; pulse 100; countenance extremely anxious and depressed.

30th.—Same.

November 4th.—Pulse 126; slept a little last night; urine high coloured, contains no lithates; not so much pain in the region of the heart; the pleurisy on the right side is better; bowels rather relaxed; tongue furred. The calomel to be omitted from the pills. ℞ Opii gr. j bis die.

6th.—Bowels more regular; not so much pain.

7th.—Not so well; bronchitis came on in the night. Rhonchus heard in both lungs, principally at the upper part; pleuritic rub still audible on right side, in which place he finds pain when he coughs; the pericardial friction-sound is not so distinct as it was; pulse 132; bowels regular.

9th.—He is better; pulse 132; tongue moderately clean; cannot get much sleep; bowels regular; perspiration less; there is no pleuritic rub on left side; cough still bad; can't expectorate much. Is ordered a blister. Vini Xerici ℥iv.

10th.—Passed a restless night; cough very bad, but can expectorate; pulse 132; heart sounds rather muffled, but the rub cannot be heard very distinctly; not so much pain on coughing.

12th.—Passed a very bad night; there is not much rheumatic pain in joints except the shoulder. Ordered calomel again.

16th.—Much better; in no pain except when he coughs, when there is a little at the lower part of chest; sleeps well; tongue clean; not much expectoration; pulse 114; appetite good; bowels regular; not in much perspiration; has still a pericardial rub.

18th.—Discontinued calomel, but to have Pulv. Dov. gr. v ter die. There is now only a single pericardial rub.

23rd.—Improving; appetite good; tongue very clean; bowels regular; no perspiration. Pleuritic rub gone; there is a systolic bruit, most audible at apex; can be heard in axilla and in back; but pulse regular; consequently Dr. Barlow thinks that it is a pericardial rub.

26th.—Pulse 100, regular. Pot. Bic. gr. x ex Inf. Calumb. ter die.

28th.—Much better; appetite good. Discontinued Dover's powder as he sleeps well without it.

30th.—Got up for first time. Full diet.

December 11th.—Is now well, but weak; there is still single pericardial rub to be heard.

16th.—Discharged well.

Case 5.—Ward Clinical, No. 23, under Dr. Barlow; clinical clerk, Mr. E. F. Turner. Mary Anne S—, æt. 19, admitted October 26th, 1864.

October 26th.—Suffers from pain and swelling in all her limbs except right leg. On the 19th of this month she caught cold and her legs began to ache; she continued to get worse till the day of her admission. Two years and six months ago she had another attack, but a much milder one; she was in bed only four or five days, and then attended as an out-patient at the London Hospital for three or four weeks. For the last five months she has been nurse in a family who keep a public-house in the Borough. One of the children has been ill, and she thinks she has exposed herself by frequently getting up in the night to attend to it. ℞ Pot. Bic. ℈j, Pot. Nit. gr. v, Vin. Opii ♏v, ex Julep. Menth. Pip., 4ta quaq. hora.

27th.—She lies in bed on her back, and can only move her right leg without pain; face and body generally bedewed with perspiration; hands have a sodden appearance, pale, but the knuckles pink; there is the peculiar sour smell of rheumatism; there are sudamina upon the face; tongue covered with a thick yellow fur, red at the tip; lips parched; complains of great thirst; bowels open once yesterday. Heart's dulness normal; first sound only to be heard very faintly, second sound clear. Pulse 96, full and moderately hard; catamenia regular.

28th.—Urine loaded with lithates, acid, non-albuminous. The first sound up the aorta is not clear.

29th.—She can move her left hand a little to-day; rheumatic odour strongly marked; tongue covered with a little brownish fur; sleeps badly; is rather talkative the nurse says; heart sounds as before.

31st.—Feels better altogether; can move all her limbs now and turn round in bed; sweats less; face very pale; her tongue is coated with a dirty yellow fur on each side of the median line. Impulse of the heart below and to the left of the left nipple; there is another impulse to be felt in the left third interspace, close to the sternum, where the second sound is quite clear, but there is a systolic murmur. Breast plentifully covered with sudamina.

November 1st.—Pulse moderately full, but there are occasional intermissions. I find her lying upon her side and expressing herself as feeling much better.

3rd.—Temperature 99·3; pulse 86; the murmur has disappeared; she does not sweat now, and the sudamina are drying up; sleeps better; fur almost cleaned from tongue, which is red and pointed; bowels open once daily.

4th.—Ordered Pot. Bic. gr. xv, Sp. Ammon. co. ʒss, ex. Inf. Cinch. ℥j.

5th.—She felt very cold during the night, and this morning the left index finger is markedly swollen and tender, and she has pain behind the knees when she bends them; tongue clean. Yesterday afternoon she felt a pain in her right side, and does now when she breathes deeply; there is a creaking sound to be heard there with the stethoscope; temp. 99·5. Rep. mist. ex Aqua vice infus. Cinchon.

7th.—Temp. 98·6; heart sounds normal, but the pulse still intermits, though fairly full.

8th.—Temp. 99·2; he wants to get up, but feels too weak to walk.

9th.—Temp. 99.

11th.—Pains fly about her joints still; yesterday both her wrists were so painful that she could not help herself; to-day her left wrist is painful; her appetite is good; tongue clean; bowels regular; sleeps well; temperature 98·4.

18th.—First sound rather muffled at base of heart, but no distinguishable bruit; it is of better quality; began to eat meat the day before yesterday.

23rd.—Got up the first time yesterday afternoon; feels daily stronger; pulse not so intermittent.

29th.—Has been steadily improving; there is no bruit distinguishable up the aorta. Discharged well.

The first of the above cases was one of moderate severity. On the day of his admission there was no evidence of any heart affection, and he was, at the suggestion of my clerk Mr. Rendle, placed upon the use of the lemon-juice, a remedy which, as is well known, has been suggested and recommended by my colleague Dr. Rees, chiefly upon the ground of its probable chemical agency. It is one, too, which I have often found very serviceable. It is true, indeed, that the systolic murmur was first heard upon the day following the first employment of the lemon-juice, but, owing to the shortness of the interval, it would by no means be just to infer that therefore the remedy is less efficient than others in preventing the heart complication. The mixture containing the bicarbonate of potass was employed in moderate doses, and the patient left the hospital well at the end of thirty-one days. The period from the commencement of the treatment to incipient convalescence was twelve days.

In the second case the bicarbonate of potass was the remedy chiefly employed, as the skin acted very freely, and the urine was scanty and contained purpurine. This case, it should be observed, was one of a somewhat chronic character, the patient having been ill for three weeks before his admission. The time from the commencement of the treatment to decided convalescence was twenty-four days.

The third case, being not of a very acute character, was treated upon the expectant system, and under the use of it he at first appeared to be advancing favorably, and at the end of five days was convalescent, but at the end of ten days he had a relapse of a very severe character. He was then put upon the use of considerable doses of citrate of potass, with about twelve grains of the bicarbonate in excess, a combination in which I believe that the alkali enters the system more readily than when given in the form of simple bicarbonate. The period from the commencement of active treatment till the commencement of convalescence was twelve days.

The fourth case was treated with the acetatc of potass and nitrate of potass, a combination which is most efficient as an eliminant by the kidneys; and if we also give some Dover's powder at night, and apply cotton wool to the affected joints, we have generally a free excretion by the skin. In this case the improvement was at first very rapid, and would, probably, have continued so but for the accidental attack of bronchitis. In some severe cases of acute rheumatism treated by these remedies I have witnessed very speedy recoveries, more so, perhaps, than under the use of any other remedy.

It would require a very large number of cases, more, perhaps, than have yet ever been brought together, to test the superiority of one mode of treatment above another. Neither, for my own part, should I desire to make the observations requisite, since, uncertain as we appear to be as to the best remedy for acute rheumatism, I think that enough has been observed, especially if viewed by the light which may be obtained from the observation of other diseases, to show us that there are particular indications in each individual case, and that to use the same remedies indiscriminately in all cases would be productive of mischief.

We know that rheumatism is an acute disease, running a certain course, which it is rarely, if ever, in our power to stop, although the time in which this process is completed may be considerably controlled by remedies adapted to the exigencies of the case.

That a case of rheumatism should recover under the expectant treatment is no proof that such treatment is applicable to all cases. I have, indeed, found several do well in this way, but those were cases which, according to my views, presented the greatest probability of going on spontaneously to a favorable termination.

It may here be observed, that the process employed by nature appears to be the elimination of the morbid poison by the skin, and by the kidneys in the form of urates, and that whenever this elimination is going on freely and safely there is no ground for promoting it by active remedies; indeed, I would even go further, and maintain that it may be absolutely injurious to do so, and that there are good grounds for believing that the indiscriminate use of eliminants, especially in the form of diuretic salts, has often acted injuriously upon the kidneys, and led to structural change in those organs. But, on the other hand, we must remember the liability to the accidents of the disease, which are of very grave character, in the form of internal inflammation, chiefly affecting the heart, diaphragm, and pleura (though serous and fibrous tissues elsewhere do not always escape), and that these accidents are more liable to arise when the elimination of the morbid poison is suspended. For instance, a case occurred, and such are not uncommon, of a young woman who had arthritic pains, fever, scanty urine, and dry skin, so as almost to make it doubtful whether her disease was really acute rheumatism; there was at the same time a faint systolic friction sound. Besides the administration of active diaphoretics, in the form of frequent full doses of acetate of ammonia, with small quantities of tartarized antimony, and Dover's powder night and morning, a blister was applied to the region of the heart; the true rheumatic perspiration came on freely, and the patient recovered speedily and well; and I believe that the same principles apply to the use of diuretic eliminants, though it must be borne in mind that these are more apt to have an injurious action.

The chief danger, however, arises, as is well known, from the liability to internal complication, espceially in the form of pericarditis or endocarditis, or, as I would rather say, carditis.

Neither is the danger arising from diaphragmatitis to be lightly treated of. It has been a question whether, when these inflammations do arise, we ought to regard them as common inflammations or treat them as rheumatic. I believe that the latter is the safer plan, and that although, in the case, for instance, of pericarditis, the old combination of calomel, antimony, and opium, should not be discarded, and may be used advantageously at night or night and morning, the saline diaphoretics and diuretics, the latter in the form of salts of potass, should, under these circumstances, be prescribed, or, if previously given, be more actively employed. A blister to the region of the heart at the commencement of cardiac inflammation often appears to arrest its progress.

A SECOND REPORT

OF

CASES OF ACUTE RHEUMATISM

TREATED IN THE WARDS OF GUY'S HOSPITAL,

WITH REMARKS

ON THE NATURAL HISTORY OF THE DISEASE.

By HENRY G. SUTTON, M.B.

In the last number of the 'Guy's Hospital Reports' we reported several cases of rheumatic fever that had been under the care of Dr. Gull, and had been treated for the most part by mint water only. Since that time we have had an opportunity of observing several other cases similarly treated and of testing our previous conclusions, and we would venture to observe that this subsequent experience has tended to confirm what we have already stated.

The cases that have been treated by mint water during the past year have been few, for the simple reason that the cases of rheumatic fever admitted into the hospital have not been numerous.

A few cases have been treated according to different plans, and some of these, in which lemon-juice was employed, are recorded by Dr. Rees in the present volume. We have, however, thought it more advisable to introduce, in this paper, only those cases that have been treated almost by mint water alone.

The cases recorded below were all well-marked cases of rheumatic fever. Some of them were under the care of Dr. Barlow, and one was under the care of Dr. Wilks, but the majority were under the care of Dr. Gull.

CASE 1. *Severe rheumatic fever; pericarditis and pleurisy.*—Fred. C—, æt. 14, a brickmaker, admitted October 30th, 1865, into Stephen Ward, under the care of Dr. Gull.

He states that he has always enjoyed good health with the exception of catching cold. He has been, owing to his occupation, very much exposed to changes in the weather. He attributes his present illness to having got wet through. On October 23rd he was seized with pain in his ankles and hips, which has continued up to the time of his admission. He says that he has been under treatment for rheumatic fever.

October 31st.—He states that he is not in nearly so much pain as he was before coming into the hospital. His skin is cool and not perspiring; tongue almost clean. The condition of his heart is as follows:—The visible impulse is unduly marked and diffused; the rhythm is irregular; there is a slow prolonged beat, followed by three or four quick irregular beats; on percussion there is absolute dulness as high as the third rib and as low as the sixth rib, and extending a little to the left of the left nipple; a harsh, creaking, systolic bruit is heard over the second left costal cartilage, but it is not carried along the aorta. The pulse was 60 in the morning; in the afternoon it varied from 36 to 54; respiration 28. Urine acid, high coloured, sp. gr. 1015, no albumen.

November 1st.—In no pain. Tongue rather white and moist. Heart—the area of dulness is the same as yesterday; the first sound at the apex is prolonged, and the same creaking murmur is heard over the base; rhythm still irregular; pulse 60; respiration 24. ℞ Aquæ Menthæ ℥j, Ext. Taraxaci ʒss, 4tis horis. Milk diet.

2nd.—In no pain; pulse 72, irregular, soft, and compressible; tongue moist and clean; skin cool, clean, no perspiration. He says he sweated a great deal last night. Heart—the dulness is somewhat less than before; the impulse is diffused and unduly marked; the bruit at the base is more like a soft systolic murmur. There are sudamina on the chest, of which the contents are highly acid.

3rd.—He complains of a little pain in his finger-joints and also in the left knee; urine faintly acid, clear, straw-coloured, sp. gr. 1020, no albumen, quantity about normal.

4th.—Much the same; urine neutral, sp. gr. 1022, no albumen.

5th.—He has pain in the joints of the left hand, but they do not appear tender on pressure; he has also pain and tenderness in the left wrist and pain in the left knee; his tongue is moist and slightly furred at the base; he states that he sweated a little last night; pulse 68, of a jerking character, and irregular. Heart—apex beat unduly marked; visible impulse diffused; cardiac dulness as before; a systolic bruit over the left base.

6th.—He still complains of a little pain in the finger-joints of the left hand; the pain is evidently very slight, for he can bend all his joints, and there is no tenderness nor swelling of the joints; tongue slightly furred; skin cool, not perspiring; pulse 96. Cardiac dulness less marked; impulse the same under the left nipple; a creaking systolic bruit at the left base; second sound very sharp, first sound at the apex feeble. Urine very acid, with a deposit of lithates; he says that he feels better; appetite good.

7th.—Skin cool; he has a little pain in the right elbow; he perspired a good deal last night; state of heart much the same; urine alkaline, of a pale-colour, and of good quantity, sp. gr. 1014.

8th.—His skin is cool; tongue clean; appetite good; no pain in the joints; he complains of a little pain between the shoulders; skin is not perspiring; sudamina are present; urine pale, alkaline, with a film on the top, sp. gr. 1018.

9th.—He complains of pain in his left wrist and left elbow, and between his shoulders. Last night he sweated a good deal; his appetite is good; the contents of the sudamina are very acid; tongue slightly coated with light fur; skin rather hot, but not now perspiring; pulse 112; respiration 36. Heart—the area of cardiac dulness is the same as before; the impulse is very diffused, and visible over the base, as well as over the apex of the heart; a systolic bruit exists at the base. Urine free in quantity, alkaline, sp. gr. 1016, no albumen;

when boiled it becomes milky looking, but clears, with effervescence, on the addition of nitric acid.

10th.—Urine alkaline, sp. gr. 1017 (this urine was passed three hours ago); it is pale, with a phosphatic film on the top; it effervesces on adding nitric acid. He says he is not so well to-day; he has pain in the left hand; tongue clean and moist; left wrist swollen and evidently very painful; countenance anxious; skin not hot and not perspiring. Heart's visible impulse seen over a space extending from the second to the fifth rib; systolic murmur over the second left costal cartilage; dulness again somewhat increased.

11th.—He complains to-day of great pain in his wrists, knees, and ankles; his wrists and the backs of his hands are swollen; tongue moist and covered in parts with a thin white fur; skin hot, not perspiring; temp. 102·2; heart's condition much the same as yesterday; urine acid, no albumen, sp. gr. 1019. Rept. mist. Pil. Opii gr. ss bis die sumend.

13th.—He is in a great deal of pain; the wrists are swollen, red, and very painful; he cries out if the bedclothes are moved; the skin is hot, but not perspiring. Heart—a systolic bruit still heard over the base; impulse diffused. Countenance very anxious; he says that he is in a good deal of pain in his loins, knees, and ankles, and also in his heart. His own words are—"I thought I was going to die, the pain around my heart was so bad." Hot fomentations are applied.

14th.—He says that he is in a great deal of pain to-day in his joints, and that he has shooting pains through his chest, especially when he draws a deep breath; tongue dry; cheeks flushed; skin dry; no perspiration. Heart's visible impulse very distinct; on a level with and half an inch to the left of the left nipple there is a tactile friction fremitus; there is absolute dulness as high as the upper border of the third rib, and diminished resonance as high as the second rib; the dulness is carried half an inch to the left and is bounded on the right by the sternum; a confused to-and-fro murmur is heard all over the heart, and a loud systolic bruit at the base; the rhythm is regular. Urine high coloured, acid, of sp. gr. 1025; it becomes milky by boiling, but clears on the addition of nitric acid.

15th.—He says he feels better to-day than he did yesterday, but that he slept very badly last night; he has pains in his knees and groins, and also in the left shoulder; the tongue is moist and white; he is perspiring about the face; countenance less anxious; pulse 120, full and compressible; respiration 32. The heart appears to be in much the same condition. The urine is acid, of a deep orange colour, 20 oz. passed in twelve hours, specific gravity 1020, no albumen.

16th.—He feels much about the same. Tongue clean and moist; pulse 120; temp. 101. The cardiac dulness extends as high as the second rib. A creaking systolic murmur is heard at the base; no bruit at the apex. A pleuritic crackle is heard to the left of the nipple; respiration 40; urine acid, 30 oz. passed in seventeen hours, sp. gr. 1010. A very faint trace of albumen; no sediment on cooling.

17th.—He says he passed a better night. He complains of great pain in both his knees, and also in his elbows and left shoulder; his skin is hot; there is not much perspiration. Sudamina exist on the chest, the contents of which are intensely acid; countenance distressed; pulse 136; respirations 40. Heart apparently in much the same condition as yesterday. Urine of a pale straw colour, sp. gr. 1015, faintly acid.

18th.—He says he is not in so much pain, but he complains of a catching pain in his left side, especially when he draws his breath. Tongue moist, furred at the base; pulse 129; respirations 36. A to-and-fro murmur is heard over the region of the heart; tactile friction fremitus exists over the base. A pleuritic crackle is heard in the left axilla. The urine passed in the night is high coloured and acid, and contains a sediment; sp. gr. 1015; quantity passed in sixteen hours 52 oz.

19th.—Urine high coloured, acid, containing a very copious deposit of lithates.

20th.—The pain in his knees is a good deal better, but he complains of pain under his heart. His skin is dry, but not particularly hot; his tongue is moist, with a thin layer of white fur; pulse 116; respiration 36. Heart—dulness the same; to-and-fro murmur still heard. Urine—47 oz. passed in twenty-seven hours; that passed at 10 a.m. is alkaline, of sp. gr. 1014; that passed during the night is faintly acid.

21st.—Much the same. The urine passed at 12 at noon is neutral, of sp. gr. 1022; pulse 108. Dr. Gull says there is considerable pleuritic effusion on the left side. There is dulness all over the left chest, with bronchial breathing close to the left spine.

22nd.—Much the same.

23rd.—Pain in both knees, the right being markedly swollen; no pain in any other joints; tongue clean and moist; pulse 102; skin rather hot, perspiring; there is dulness all over the left chest except at the extreme apex; bronchial breathing and bronchophony are heard behind, but scarcely any respiratory murmur. Heart—the impulse is seen at the base as well as at the apex; it is difficult to define the exact position of the left apex; a harsh to-and-fro murmur is heard all over the cardiac region, and the dulness reaches as high as the first rib. Urine—25 oz. passed during the night; it is of an acid reaction and sp. gr. 1020.

25th.—He is in no pain except in the left side on drawing a deep breath; the cardiac dulness is diminished; the bronchial breathing is much less marked; the urine is acid, straw coloured, 19 oz. passed in twelve hours.

27th.—He is free from pain. Urine—sp. gr. 1014; it is acid, and contains a trace of albumen.

29th.—He says he is a good deal better, and complains of no particular pain anywhere; his tongue is clean; he says he did not perspire so much last night; cardiac dulness much diminished; no to-and-fro murmur audible, but a systolic bruit at the base; the first sound at the apex exceedingly feeble; pleuritic crackle during inspiration; dulness of the left side and bronchial breathing much less marked; ægophony at left base posteriorly. Urine—29 oz. passed in about fifteen hours, acid, rather pale, cloudy, but containing no actual sediment, sp. gr. 1015.

30th.—He is in no pain and rapidly improving. Urine faintly acid, pale in colour, of sp. gr. 1010, no albumen; 35 oz. have been passed in twelve hours.

December 2nd.—He is in no pain; he says he feels much better; the dulness on the left side is much diminished.

4th.—He is going on very well. Skin cool; area of cardiac dulness reaching as high as the upper margin of the third rib; first sound at the apex very feeble; ægophony at the left base posteriorly. Urine pale, cloudy, of sp. gr. 1009, slightly acid, containing a faint trace of albumen; 33 oz. passed in sixteen hours.

8th.—He is free from pain. A bruit is heard over the base of the heart; the first sound at the apex is more distinct. Urine straw coloured, cloudy; 26 oz. passed in fourteen hours, sp. gr. 1015, containing a trace of albumen.

12th.—He is going on very well, looks much better, and is in no pain; cardiac dulness much less than it was; first sound at the apex more distinct; a somewhat harsh systolic bruit at the base; urine pale, and 43 oz. passed in seventeen hours, sp. gr. 1015.

From this time he continued to do well, and he was soon discharged.

Case 2. *Rheumatic fever; no heart affection.*—Kesiah S—, æt. 20, admitted November 21st, 1865, into Mary Ward, bed No. 12, under the care of Dr. Gull. She says that this is her first attack of rheumatic fever, and that she has been ill nine days. She complains of very great pain in her wrists, ankles, and shoulders; she is perspiring very freely; tongue red; no appetite; a systolic bruit is heard over the base of the heart between the second and third left costal cartilages. ℞ Ext. Taraxaci ʒj ex Julep. Menthæ, 4tis horis. ℞ Ol. Ricini ℥ss stat. Milk diet.

October 22nd.—Suffers from great pain in the same joints.

23rd.—She is not in "quite so much pain."

24th.—She is in more pain. Rept. mistura.

26th.—Still in great pain.

30th.—She is free from pain; tongue clean; skin cool; convalescent.

December 4th.—She is going on well. ℞ Sodæ Sesqui-carb. gr. xv, Infusi Cascarillæ ℥iss, t. d. Middle diet.

She continued to do well, and was discharged.

Case 3. *Severe rheumatic fever; heart complication.*—David D—, æt. 23, a draper's assistant, was admitted November 3rd, 1865, Stephen Ward, bed No. 38. He states that he has enjoyed pretty good health, and has considered himself to be strong. Ten years ago he had a fever, and was laid up three weeks, but he does not know what kind of fever it was. He says that this is his first attack of rheumatic fever. On October 28th he was taken ill with pain in the left wrist. On the 29th the wrist was very painful and swollen. On the 30th he had pain in his knees and was compelled to give up work. After this he had pain in several joints.

November 3rd (the day of admission).—Dr. Gull has seen him and says that there is a systolic bruit at the left apex; the ankles are swollen and painful; the tongue is brown and dry; temp. 102; pulse 100; respirations 28. ℞ Ext. Taraxaci ʒj, Julepi Menthæ ℥j, 4tis horis. Milk diet.

November 4th.—He complains of pain in the back of the neck, in the knees, and in the right wrist; he is perspiring very freely; the rheumatic odour is strongly marked; tongue brown and dry in the centre, and covered with a yellow fur on the sides; pulse 96, full, but compressible; respiration 28. Cardiac dulness not increased; apex beat unduly marked, but in the normal position; systolic bruit over the left apex. Urine very highly coloured, depositing lithates, sp. gr. 1027.

5th.—He passed a restless night in consequence of the pain being so severe; the pain in the ankles and knees is not so great, but there is great pain in the right shoulder; skin perspiring; tongue still brown and dry; pulse 88; respiration 28. Urine highly acid, of sp. gr. 1030, and containing a copious deposit of lithates.

6th.—He says that he did not sleep much last night on account of the pain. This morning he has pain in all his joints, and cannot move; the tongue is coated at the sides and red in the centre; he is perspiring very freely, perspiration very acid; pulse 80; respiration 32. Urine high coloured, very acid, giving a copious deposit of lithates, sp. gr. 1030, no albumen.

7th.—He says he is "in a deal less pain," but that he passed a "shocking night" up to six this morning, when the pain left him suddenly. He has still some pain in his arms, legs, and back, but very much less than yesterday; skin perspiring very freely; tongue cleaner; heart in much the same condition as when last reported. Urine high coloured, very acid, of sp. gr. 1027.

8th.—He says he feels much better and is in less pain; tongue moist and cleaner; skin cool and perspiring. He is able to move himself in bed and to raise himself. He complains that his throat is sore when he attempts to swallow; the left tonsil looks very red, but there is no exudation upon it; pulse 80, less full and bounding; perspiration acid to test-paper; 20 oz. of urine are passed in twelve hours, it is acid, and of sp. gr. 1023, and contains no albumen; heart in much the same condition as when last reported.

9th.—He says he passed a very bad night; he complains of pain chiefly in the left arm; tongue coated with yellow fur, red in the middle. He says that the pain in the arm is as bad as ever; he perspired a great deal last night, and is now perspiring profusely; the perspiration is very acid; he says that he is very thirsty; pulse full and bounding, 80. Heart—no increase in the area of cardiac dulness; systolic bruit heard very distinctly over the apex. Urine copious, high coloured, acid, of sp. gr. 1026, containing no albumen.

10th.—Says that he has pain all over him this morning; his ankles are very painful; he passed a very bad night, on account of the great pain; tongue dry, reddish-brown in the middle; he is sweating profusely; the perspiration is intensely acid; pulse 80; urine not so high-coloured, acid, of sp. gr. 1025. Dr. Gull saw him to-day, and, finding that he was suffering very much, ordered him Opii gr. j hâc nocte, Pulv. Seidlitz. primo mane.

11th.—He says that he slept all last night; he is in much less pain this morning, but he has still some pain in his arm and also in his legs; the tongue is very red in the middle and coated at the sides with yellow fur; he is perspiring very much, but not quite so much as yesterday; perspiration acid; he says he is very thirsty; pulse 88, not so full and bounding, very compressible. Heart's impulse very distinct; systolic bruit heard over the apex, but not over the base. Urine —sp. gr. 1025, high coloured, acid, containing no albumen.

12th.—He has no pain, except in the arms; he says that he feels decidedly better, and that he passed a better night.

13th.—Says, "I am almost free from pain; still a little in my arms." He still sweats freely; he passed a better night.

14th.—He states that he slept very well last night, and that he has no pain except a little in the shoulder; he can turn any way in bed, and says, "I was able to wash myself this morning;" appetite much better; skin moist and cool; tongue almost clean; pulse 75 and soft. Heart—cardiac dulness not increased; a loud systolic bruit at the apex. Urine not so high-coloured, clear, acid, of sp. gr. 1021, containing no albumen.

15th.—He is still improving; urine acid, of sp. gr. 1022; 40 oz. passed in twenty-four hours.

16th.—He says he slept all night, and is free from pain; tongue clean and moist; appetite good; skin cool and moist, but not perspiring; pulse 72; heart apparently in much the same condition. Urine acid, of a deeper colour than yesterday, and of sp. gr. 1026; 34 oz. passed in twenty-four hours.

17th.—He slept well last night, and is in no pain; tongue clean; skin moist; he says he "does not perspire near so much;" perspiration still acid; appetite very good; pulse 80, feeble, no longer full and bounding.

18th.—He is free from pain, and had no perspiration last night; skin moist; perspiration acid. Heart—systolic bruit heard at the apex, but not at the angle of the left scapula. He had two teaspoonfuls of castor oil this morning, which operated well. Urine—that which was passed in the night has an alkaline reaction, that which was passed twenty minutes ago is acid, but not intensely so.

19th.—He says that he feels slight pain on moving the left shoulder, but that there is no constant pain in this part, and no pain anywhere else. He perspires very little now; tongue clean; urine high coloured and acid, of sp. gr. 1030. He got out of bed in the evening without leave.

20th.—He says he has a little pain occasionally in both shoulders; tongue clean; appetite good; skin moist, but without rheumatic odour; heart in much the same condition; urine acid, high coloured; 26½ oz. passed in twenty-four hours, sp. gr. 1027.

21st.—He is much better to-day; he was not quite so well yesterday; this was attributed to his getting out of bed without leave; his appetite is very good; he slept well last night, but perspired a good deal; he is now in no pain; urine not so high coloured, acid; 31 oz. passed in fifteen hours, sp. gr. 1019, slight trace of albumen.

22nd.—Much the same.

23rd.—He is going on well, is free from pain, and is to get up to-day. A loud systolic bruit is heard at the apex of the heart; the bowels have not been opened for some days; the tongue is clean; there was no perspiration last night; urine acid; 41 oz. passed in twenty-four hours, sp. gr. 1020.

24th.—He is out of bed.

25th.—He is feeling much better.

27th.—He is out of bed and doing well; he says that he is well, except that he has a little pain in his back; he sleeps well; appetite good; pulse 80.

29th.—He is going on very well, is out of bed every day, and is free from pain. There is a well-marked systolic bruit over the apex of the heart, but it is not heard in the middle of the axilla, nor at the angle of the left scapula; the apex beats between the fifth and sixth ribs, a little to the right of the nipple; there is also a systolic bruit at the left base. Urine acid, straw coloured, and of sp. gr. 1022, containing no sediment.

30th.—He is going on well, and says he feels stronger every day; urine acid, of sp. gr. 1013, no albumen; more than 20 oz. passed in twelve hours.

December 2nd.—He is going on well.

4th.—Urine pale, acid, of sp. gr. 1009, no albumen; bruit still audible over the left apex of the heart, but not so loudly.

5th.—Urine acid, of the natural colour, and of sp. gr. 1020. Bruit still heard, but less distinctly; cardiac dulness normal in extent; the apex beats between the fifth and sixth ribs and to the right of the left nipple.

7th.—He went out of the hospital.

Case 4. *Rheumatic fever; symptoms acute.*—Henry S—, æt. 19, a warehouseman, admitted October 28th, 1865, into Stephen Ward, No. 44 bed, under the care of Dr. Gull. He states that he has never had any illness until two years ago, when he had an attack of rheumatic fever, with which he was in Guy's Hospital for six weeks; after that time he continued well until the 23rd inst., when he felt a little pain in his back, but he remained at his work until the 25th. On that day he was seized with pain in the right knee. On the 26th the pain was so severe that he could hardly turn himself in bed. On the 27th he was still in great pain, and on the 28th he entered the hospital.

On admission. He complains of great pain in his ankles and knees; his skin is hot and he is sweating freely. The area of cardiac dulness is not increased; a systolic bruit is heard over the apex of the heart. ℞ Ext. Taraxaci ʒj, Aquæ Menthæ ℥j, 4tis horis. Milk diet. He is strictly ordered not to get out of bed for any purpose whatever.

October 29th.—He has pain in the shoulders. He states that the pain in the left leg has increased.

30th.—He says that he has much less pain in his ankles and knees, and that he can move his legs freely, but he complains of pain in the right wrist; sweats a great deal; appetite not very good.

31st.—Pain much the same; a blush of redness on the right wrist. He says he perspired a great deal last night, but he is not now perspiring. Tongue coated with white fur; a systolic bruit still heard. Urine high coloured, slightly acid, of sp. gr. 1028.

November 1st.—He is not in so much pain, but the right wrist is still painful, swollen, and red. Rept. mist. Diet as before.

2nd.—He passed a very good night, but perspired a great deal; he is in less pain; tongue moist, but covered with a white fur. Urine acid, of sp. gr. 1030, containing no albumen. Systolic bruit heard over the apex of the heart.

3rd.—He says, "I feel better to-day, but have a little pain in the wrist." He does not perspire except during the night; appetite pretty good; urine amber coloured, acid, sp. gr. 1028, no albumen.

4th.—He is in no pain and sleeps well; skin cool; tongue clean. Appetite good; pulse 68. Slight systolic bruit at the apex. Urine acid, of sp. gr. 1023, containing albumen.

5th.—He says he feels better and is in no pain; pulse 64. Urine acid, of sp. gr. 1025, containing a little albumen, but not so much as yesterday.

6th.—He is free from pain. Appetite good; pulse 60;

heart in much the same condition. Urine very pale, acid, of sp. gr. 1010, containing a very slight trace of albumen.

7th.—He is going on well. Urine acid.

9th.—Urine acid, of sp. gr. 1026; the quantity of albumen has increased.

11th.—He has been going on well; he is walking about the ward. Urine acid, containing no albumen, its sp. gr. 1022. From this date he continued to do well.

Case 5. *Rheumatic fever; symptoms acute; heart complication.*—David A—, æt. 16, admitted June 6th, 1866, into Stephen Ward, No. 25 bed, under the care of Dr. Gull. He is a carpenter. He says that he has always been delicate. Some time ago he had scarlet fever, and ever since that illness he has had a discharge from his ear. He has never been laid up with any other disease. This is the first attack of rheumatic fever, and he attributes it to exposure to cold. Fourteen days ago he was seized with pain in his left foot; the pain remained in this position for two days and then attacked several other joints. He has been perspiring a great deal ever since he was taken ill. The pain has attacked chiefly his knees and ankles. He has been in bed ever since he first felt the pain. No medical man has attended him. He has taken antibilious pills, but no other medicine.

June 6th.—He has very severe pain in both ankles; these joints are very red and tender, and the right one is swollen. He has also pain in the knees, and a little in the left wrist when he bends it. He is perspiring very profusely about the face. The skin over the body feels hot. The tongue is thickly coated with white fur, and is red at the tip and sides. The pulse is 112, full, bounding, and compressible. The area of cardiac dulness is not perceptibly increased; a very indistinct systolic bruit is heard over the left base, more like a prolonged first sound; the apex beat is unduly marked. Urine high coloured, acid. Ext. Opii gr. ss omn. noct. vel bis die. Julep. Menthæ ℥j 4tis horis. Ext. Tarax. ʒj sextis horis. Milk diet. He is not to get out of bed.

7th.—He says he is in more pain and that he has passed a very restless night. Pulse 100, full. He has very severe pain in his knees and in his left ankle; he is perspiring profusely; the tongue is thickly coated with fur. The cardiac dulness reaches as high as the upper margin of the third rib; the impulse of the apex of the heart is seen and felt to be unduly marked, but in the normal position; a systolic bruit is heard over the left third costal interspace, close to the sternum; the first sound at the apex is prolonged. Resp. 32. Urine high coloured, acid; 23 oz. passed in twenty hours, sp. gr. 1026.

9th.—He has great pain in all his joints; he lies on his back, and says that he is unable to move. He is sweating profusely, the perspiration standing in drops on his face. His left wrist is red and swollen; tongue thickly coated with creamy fur; pulse 101; resp. 32. Heart in the same condition as yesterday. Urine clear, rather high coloured, acid, of sp. gr. 1031.

12th.—He says that he feels better, and he looks less anxious. His legs are free from pain and he can move them freely; he has pain in his elbows and shoulders. Tongue almost clean and moist; skin sweating profusely; pulse 92, full and compressible. The cardiac dulness still reaches as high as the upper margin of the third rib; it is not absolute, but is rather a diminished resonance with a sense of increased resistance; a systolic bruit exists over the left base. Urine of natural colour, acid, and of sp. gr. 1028. Rept. mist. Milk diet.

13th.—He is in less pain, though he has still some pain in the right shoulder and right leg. Skin is cool; pulse 92. Heart in much the same condition. Tongue clean. Urine, sp. gr. 1031.

14th.—He has a little pain in both shoulders. He says he is better, and looks better. Tongue clean; pulse 96; resp. 28. He is not perspiring so much. Urine—16 oz. passed in ten hours; it is clear, acid, of a natural colour, and of sp. gr. 1018.

16th.—The pain is much less. He can move all his limbs, but he has still a little pain in his left shoulder. Skin cool and dry; tongue clean. The first sound of the heart is prolonged over the base, but no longer forms a decided murmur. The urine is acid, clear, of a natural colour, and of sp. gr. 1026; 12 oz. passed in seven hours. The bowels are open. Rept. mist.

19th.—He is going on well. Skin cool, not perspiring; pulse 96. He says he has no pain except a little in the left shoulder. Tongue clean. Urine clear, of sp. gr. 1021.

20th.—He is entirely free from pain.

21st.—Urine clear, of sp. gr. 1025. He is sweating a good deal about the face. Pulse very soft and compressible; tongue almost clean; heart in a normal condition. He says that he feels a great deal better. His appetite is good. He had no pain yesterday, but to-day he complains of an aching pain in the right shoulder. His skin is cool.

23rd.—He is in no pain, but had a little pain in his shoulder yesterday. No bruit can be heard; the cardiac dulness is normal. Tongue clean; skin cool; urine acid. Dr. Gull saw him yesterday and ordered him to remain in bed. Rept. mist. Milk diet and two eggs a day.

24th—He is free from pain. Heart in a normal condition. Urine rather high coloured, acid, of sp. gr. 1020; he passed 22 oz. to-day.

26th.—He is doing well. Urine acid and of a pale amber colour. Heart in a normal condition. Dr. Gull carefully examined his heart to-day, and stated that there is no evidence of any heart disease. From this time he continued to do well, and was discharged on July 16th.

Case 6.—Thos. N—, æt. 24, was admitted into No. 38 bed, in Philip Ward, on March 31st, 1866, under the care of Dr. Barlow. He says that this is the third time he has suffered from rheumatic fever. The first attack occurred two years ago, when he was laid up with it for eight weeks. The second attack occurred about a year ago, when he was in Guy's Hospital. His present illness commenced five days before admission (March 26th). He was seized with pain in his hips, knees, and feet, and the pain continued to get worse up to the time of his admission, March 31st.

April 1st.—We saw him for the first time next day, when there was pain and swelling in both knees, fluctuation in the right knee, pain in both ankles, and pain and redness in the left wrist. He sweats a great deal, and says that after his sleep the perspiration runs from him. The tongue is thickly coated with yellowish-white fur, and pitted at the sides.

Pulse 104, full, and abrupt. The cardiac dulness is not increased; a systolic creaking bruit is heard at the left base, the second sound is intensified, and there is a markedly prolonged first sound at the left apex. Ordered Julepum Menthæ ter die, and a low diet.

2nd.—He says that he is in great pain in his chest, going through to his back, and observes, "I can hardly breathe for it." He is in less pain in his legs. His breathing is quick, and his tongue is coated. The urine is very turbid, loaded with lithates, and high coloured.

3rd.—He says that he had a better night last night, that he is not in so much pain, and that he feels better this morning, but still has pain in his left wrist and both shoulders; the joints are swollen. Tongue moist, and coated with yellow fur; pulse 104; respiration 44. He has had a linseed-meal poultice applied to his side, which has relieved the pain. There is no increase in the area of cardiac dulness. The first sound is very feeble, and there is an exceedingly faint systolic bruit heard over the left apex. There is no pleuritic crackle. The urine is high coloured, acid, of sp. gr. 1028, and containing no albumen.

4th.—He is much about the same, but has suffered from epistaxis, and thinks that he has lost a pint of blood. The nurse and sister also think that he has lost about that quantity. The house-surgeon plugged his right nostril.

5th.—He says that he feels a good deal better to-day, that he is not in so much pain, but that he has some pain in his wrists. Tongue cleaner; skin moist; he did not perspire so much last night. Heart—apex beat in natural position; no increase in the cardiac dulness; a slight creaking sound over the left base, and a faint systolic bruit over the left apex. He has passed 16 oz. of urine during the night. It is very much loaded with lithates, very acid, and of sp. gr. 1030.

6th.—He is not in so much pain.

7th.—He says that he has a good deal of pain in the right hip, but no pain anywhere else; he otherwise feels better. The tongue is cleaner, and the appetite is a good deal better to-day. Bowels open; skin moist and cool. He says that he does not sweat so much as he did. Pulse 80. Urine clear, having an acid reaction, and a sp. gr. of 1026; he has passed 50 oz. in thirty-three hours.

9th.—Free from pain.

10th.—Free from pain; walking about the ward. He got up without the consent of Dr. Barlow. He says that he feels much better. The urine is clear, very acid, and of sp. gr. 1021.

11th.—Free from pain; out of bed; appetite good. He does not perspire so much. He has passed 40½ oz. of urine in twenty-four hours, of sp. gr. 1019, and of an acid reaction.

13th.—He is free from pain, and says that he is going on very well. He has passed 15¼ oz. of urine during the night, of sp. gr. 1025, and having an acid reaction.

14th.—Urine acid, loaded with lithates, 22 oz. in twelve hours, sp. gr. 1024. Going on well.

17th.—He has had a little pain and stiffness; more stiffness than pain in the right knee since the 15th.

18th.—In the same condition; very stiff to-day. Dr. Barlow thinks this should not be regarded as a relapse. Tongue moist; he perspired freely last night. He has passed 18 oz. of urine in twelve hours, having an acid reaction.

He continued to do well, and was discharged.

Case 7.—Elizabeth Ann W—, æt. 18, admitted December 20th, 1865, into Mary Ward, under the care of Dr. Gull. This is her first attack of rheumatic fever; she says she has been suffering pain five days. On admission the right wrist is red, swollen, and very painful; she cannot move her left leg, and has great pain in her knees; her tongue is moist and coated with a yellow fur; the rheumatic odour is well marked; the skin is hot and perspiring; the cheeks are very flushed. The cardiac dulness is not increased; a sound like the creaking of new leather is heard over the base of the heart; the sound at the apex is very feeble, but no bruit is heard there. Pulse 120, feeble.

21st.—She is in great pain, and says she cannot move herself in bed to-day; she is perspiring freely; the rheumatic odour is well marked; the tongue is moist and coated with fur; the pain is very great in the left leg and in the left shoulder, and also in the right shoulder-joint; her face is flushed; pulse 104; the heart is in much the same state as yesterday. The urine is high coloured, of sp. gr. 1029, turbid, with a copious deposit of lithates, the quantity passed in twelve hours being 21 oz., and of an acid reaction; on boiling it becomes slightly cloudy, and it remains so on adding nitric acid.

22nd.—She says that she has not slept an hour the whole night, and is now in great pain; she has pain in the left arm and she cannot move it; the pain in the right arm is somewhat better; she has pain in both legs, especially in the knees and ankles; she perspired freely last night, but not so much as the night before. The tongue is moist and thickly coated with a creamy fur in the middle; her face is not so flushed; pulse 118. Over the base of the heart a sound resembling the creaking of new leather is still heard; over the third left costal cartilage a slight systolic bruit; the area of cardiac dulness is the same as stated above. The urine is high coloured and of an acid reaction.

23rd.—The pulse is regular; the skin is perspiring; she is in less pain, but still has pain in the left arm; the tongue is thickly coated with yellow fur. The first sound of the heart is prolonged over the base, feeble and indistinct over the apex; the creaking sound is less evident.

24th.—Pulse 98, feeble, regular; she is in more pain in her legs, and thinks she has quite as much pain as ever. Tongue thickly coated; skin moist, but there is not much perspiration present; heart apparently in the same condition as yesterday.

26th.—She is not in so much pain, but says, "I cannot move myself in any way yet;" the right wrist is especially painful, but she slept "very nicely" last night; her tongue continues moist, and is much cleaner; her skin is cool and perspiring; she has no appetite; the first sound is prolonged over the base of the heart; pulse 86, compressible. Urine highly acid and very turbid, with a copious deposit of red lithates; the quantity passed in forty-eight hours being 36 oz., of sp. gr. 1032, and containing a trace of albumen. This urine has been kept for forty-eight hours, and is still acid. The bowels have not been opened since the 21st.

27th.—She says that she feels better, and that she is not in so much pain.

28th.—To-day she complains of a little pain in the left leg and foot; tongue is moist and almost clean; pulse 90; skin cool and not sweating much. The first sound of the heart is prolonged over the base, and also somewhat prolonged at the apex. The urine is very turbid, containing a copious deposit of lithates, of an acid reaction, and of sp. gr. 1032.

29th.—She is in no pain, but states that her joints feel stiff.

30th.—She continues without pain; the tongue is moist and much cleaner; she did not perspire last night, and the cheeks are not flushed; the pulse is feeble; over the second and third ribs on the left side the first sound of the heart is prolonged, and it continues slightly prolonged at the apex; she has no appetite yet, and the bowels have not acted for three days.

January 1st.—She is in no pain, and appears to be doing very well.

3rd.—Quite free from pain; tongue clean and moist; appetite much better; pulse 80; she does not perspire, even when asleep. Urine high coloured, acid, becoming somewhat milky when boiled, but clearer and deeper coloured when nitric acid is added; the quantity passed in twenty-four hours measures 26 oz., and has a sp. gr. of 1024.

5th.—She still continues free from pain, and is doing very well; there is no perspiration, and her appetite is very good; the skin is cool, and the tongue quite clean; pulse 80. A soft systolic bruit is heard over the apex of the heart, and also over the left third costal cartilage. The urine is faintly acid, and much less highly coloured, having a sp. gr. of 1028, and the quantity passed in twelve hours being 20 oz.; it becomes quite opaque by boiling, but clears on adding nitric acid; it therefore contains no albumen.

8th.—She is in no pain, but has had a bad headache this morning. The urine is paler, rather cloudy, and faintly acid, even now, *i. e.* twelve hours after being passed; the specific gravity of the 26 oz. passed during the last twelve hours is 1018. The cardiac systolic murmur is not so distinctly heard.

9th.—The urine is faintly acid; she passed 26 oz. in twelve hours, of sp. gr. 1020.

11th.—On carefully examining the heart no bruit, either over the base or at the apex, can be heard; the apex beat is in the normal position, and the area of cardiac dulness is normal; the urine which was passed eight hours ago is alkaline; that passed at 2 o'clock, while I was in the ward, is markedly acid, containing no albumen, and having a sp. gr. of 1021.

13th.—The patient is out of bed, and continued to do well till she left the hospital.

Case 8. *A severe case of rheumatic fever; early stage of pericarditis; sudden death.*—Emma L—, æt. 25, admitted January 7th, 1866, into Esther Ward, under the care of Dr. Wilks. She is a well-made woman, having large features, sound regular teeth, thick and dark hair, and a long, somewhat thick upper lip. Her father suffered from gout in the great toe, and her sister has had rheumatic fever. Until two months ago she herself had enjoyed good health, but since then she has suffered from a sense of languor, but has had no particular illness. About fourteen days ago she had a sore throat, and she could scarcely swallow anything. But it is only four days since she first felt pain in her ankles. On admission she is suffering a good deal of pain in her ankles, knees, and wrists. Heart's apex unduly marked; the area of cardiac dulness is normal, and there is no bruit. Ordered Julepum Menthæ ter die, and a milk diet.

January 8th.—She is in very great pain, and perspires profusely. The tongue is coated. She is very restless, and complains very much of the pain in her joints.

10th.—She is in great pain, especially in her wrists, and her tongue is furred. She is perspiring profusely, and the perspiration has a marked rheumatic odour. She is now menstruating. A creaking systolic bruit is heard over the base of the heart, and the first sound is somewhat prolonged at the apex. She still complains very much of the pain. Her manner is peculiar. She asks for something to give her sleep. Pulv. Dover. gr. x o. n.

11th.—She states that she passed a very restless night, and that she cannot move, the pain being so severe in almost every joint. The rheumatic odour is very marked. The tongue is coated, but not thickly, with a creamy white fur. Her wrists are red and swollen, and the pains in the wrists are especially severe. She has perspired a great deal. Pulse 112, full and somewhat bounding. The area of cardiac dulness does not extend above the third interspace. A sound resembling the creaking of new leather is still heard over the base of the heart, and over the surface and apex of the heart the first sound is rather prolonged. She requested Dr. Wilks to give her something to relieve the severe pain, and make her sleep. Accordingly she was ordered Pulv. Doveri gr. v h. s.

12th.—She is still in a great deal of pain, but not in quite so much pain as yesterday.

13th.—The tongue is coated with creamy fur, but is red at the edge. She had a much better night last night, but still has pain, though not so severe. Her bowels were relieved four times during the night. She has pain and swelling in both hands, also in the ankles and the back of the neck and shoulders. Pulse 116, not so full. A pericardial rub, which is harsh and well marked, is heard over the cardiac region, especially over the base. She is in so much pain that it is difficult to estimate the area of cardiac dulness. The urine is high coloured and turbid, and is loaded with red lithates. It becomes clear on heating it, but milky when boiled, and also when nitric acid is added, thus appearing to be albuminous. It is of an acid reaction, and has a sp. gr. of 1029.

14th.—She says that she had not so much pain last night, but she is in more pain to-day. Judging from the earnestness of her statements, she appears to be suffering severe pain, which she refers to her back and "all over her." Her hand is swollen and red, and there are sudamina on the chest. She sweats a great deal, and her skin feels hot. Pulse 120, regular and full. Her tongue is coated with creamy fur. Heart—the area of cardiac dulness is bounded above by the third rib. The apex beat is well marked and easily felt in the normal situation. A creaking systolic pericardial bruit is heard over the second left costal cartilage and over the top of the sternum. There is a harsh, grating, systolic bruit over the base, and a systolic soft bruit over the apex of the heart.

15th.—She says that she has a very severe pain across the chest, and that her chest was so tight that she was obliged to have a mustard poultice applied to it. Her skin is hot, but not perspiring. Pulse 120. She appears restless, and she complains of her head feeling stupid, and of pain in her forehead. She is very thirsty, and has a sensation in her mouth as if she had swallowed something burning hot. ("Before I get sound asleep, it burns so, and it wakes me; then I go nicely off to sleep.") She has had diarrhœa in the night. The urine is acid, having a sp. gr. of 1024. It is loaded with lithates, and is albuminous. She has passed about 20 oz. in twelve hours. She says she has always been nervous. There is a harsh, grating, systolic bruit over the base of the heart.

16th.—She has passed a very restless night, crying out "Lord have mercy on me." She seems in a very nervous condition. Her tongue trembles very much when it is protruded. Pulse 120, full and bounding. She is sweating freely. She does not seem to be in so much pain, but the hands are very painful. There is a red blush on the knuckles. The urine is loaded with lithates and very turbid, having a sp. gr. of 1024 and an acid reaction. Her bowels were relaxed during the night, and the sister of the ward says that she was delirious. About two o'clock at night the nurse observed her turning on her left side, as if to get sleep. The nurse spoke to her, and she was quite sensible. The sister stated that she heard a groan, and that she then got out of bed and spoke to the night nurse, who remarked, "No. — is come over very bad." The patient looked very white and breathed quickly, and died in a few minutes.

Autopsy.—The pericardium was healthy, except that there was a little recent lymph at the base of the aorta and pulmonary artery. The valves of the heart healthy. Muscular tissue of the heart looked dark and felt firm. A small pale clot in the right ventricle, a small dark clot in the left, but by far the greater part of the blood fluid.

The lungs were œdematous, rather congested, and tough everywhere. No emboli in the pulmonary artery. Brain healthy. The rest of the viscera were healthy. The synovial surfaces of the left knee and right wrist were partially coated by layers of yellow lymph, and there was a quantity of rather opaque fluid, with flakes of lymph in these joints. The cartilages of the joints were seen to be ulcerated.

It may here be noticed that all the above cases were treated, for the most part, with mint water only. When the patient was suffering very much pain a grain or half a grain of opium was ordered at bed-time, or twice a day, and when the bowels were confined a Seidlitz powder, with a dose of castor oil, was prescribed.

The average duration of those cases which were not accompanied with marked heart disease was nine days, and of those with heart affection of no great severity about eleven days. In the case attended with severe pericarditis and pleuritic effusion the duration was thirty-two days.

It will probably be admitted that the above record affords evidence to prove that a certain number of cases of rheumatic fever go on to recovery, and terminate in convalescence within from ten to twenty days. They also show that a patient may have extreme rheumatic pericarditis, and at the same time considerable pleuritic effusion on the left side, and yet that he may progress very satisfactorily, although no drugs be given, with the exception of a small dose of opium once or twice a day, this being prescribed with the object of relieving pain and quieting the nervous system, rather than of exercising any special influence over the inflammation of the serous membranes. The same thing may be said of endocarditis. These cases, moreover, prove that well-marked and even severe rheumatic inflammation of the heart may entirely subside and leave the heart apparently healthy, and this with little or no assistance from drugs.

We will now refer briefly to the case of Emma L——.

The first few days after her admission into the hospital this patient complained very much of the pain in her joints, and appeared to suffer a good deal. It was noticed that she was very restless, and was constantly complaining and desiring the nurse to do something for her. She also seemed very desponding. In fact, it was thought she might be labouring under some mental trouble. She asked for something to make her sleep.

The sister of the ward said, "She is the most troublesome patient we ever had; she keeps a nurse continually doing something for her." We retain this expression because it gives evidence of the restless distress under which the patient laboured. The day after her admission she began to complain of pain in her chest. In answer to our inquiry, she said, "It is not so much pain as that my chest feels so tight I can hardly breathe," and she was evidently suffering a great deal. This distress afterwards increased. The heart was most carefully examined, and there was no indication of endocarditis, but a sound like the creaking of new leather was heard over the second left costal cartilage; this appeared to show commencing pericarditis.

On the evening of the 15th she passed a restless night, and she started up in bed, crying out, "Lord have mercy on me." When we inquired next morning why she did so, she said that she suffered so much in her chest. Her own expression was, "My chest is so tight." It was noticed that her tongue, when protruded, trembled very much. During the night her bowels were relaxed.

On the evening on which she died she appeared to be somewhat delirious; she said that somebody was under the bed. It was particularly noticed by the sister that her manner was strange. The nurse stated that she was very restless that night, but that she was sensible when spoken to. About one o'clock she was observed to become suddenly very pale, and she died immediately.

In this case the pain in the joints was very great, and they were inflamed. The skin was hot, and perspiring profusely. The rheumatic odour was strongly marked. The pulse was quite full, and ranged from 116—120. The urine was scanty, albuminous, loaded with lithates, and of specific gravity 1024 to 1029. When it was examined under the microscope epithelial casts were seen.

We are next led to inquire as to the cause of this patient's death.

There was nothing to account for it in the post-mortem appearances. We may, however, notice that it is stated that the cavities on both sides of the heart were full of fluid blood. This, in itself, would tend to show that the death was sudden, and that there was a failure in the contracting power of the heart. When this fact is taken in conjunction with the sudden pallor which was observed immediately before she died, we conclude that death was due to syncope. We may next inquire to what the syncope was due.

We carefully examined the heart, in order to ascertain if there was anything in the condition of the muscular tissue which might predispose to death by syncope.

On examining the muscular fibre by the aid of a one-eighth-of-an-inch object-glass, of Messrs. Smith and Beck, we particularly noticed that the transverse striæ were not obscured by any minute sparkling granules, such as are so commonly seen in the muscular fibre of the heart; on the contrary, the transverse striæ were well marked; we therefore concluded that the muscular tissue was healthy. Thus, it did not appear that the patient's death was due to any organic change in the muscular tissue of the heart; and judging from the distressing sensation referred to the chest, we might be led to infer that it was the result of some deranged nerve action connected with the heart.

The evidence of pericarditis in this case was very slight, but its existence did not admit of doubt. During the patient's life a creaking sound was very distinctly heard over the left base of the heart, both by Dr. Wilks and by ourselves, and Dr. Wilks considered her to be suffering from slight pericarditis. The post-mortem examination showed that very soft lymph existed between the aorta and the pulmonary artery. The quantity of this lymph was small, and its presence might easily have been overlooked if the heart had not been very carefully examined.

Thus, both the physical signs during life and the post-mortem examination showed that there was an early stage of pericarditis. This patient was probably threatened with a violent attack of pericarditis, but death took place before the inflammation of the pericardium could be fully developed.

In support of this opinion we may refer to the following particulars of two cases which have been mentioned to us by Dr. Gull.

One of these cases is that of a gentleman who was suffering from rheumatic fever. The inflammation in the joints appeared not to be severe, but the patient complained of great distress about his chest. He said that he could not breathe, and that he felt as if he should die. Dr. Gull remarked that, although there was this very great distress about the chest, he could discover no abnormal sound to indicate either endocarditis or pericarditis. A few days afterwards, however, he detected a slight murmur, which increased; and as the physical signs became marked, the feeling of distress in the chest gradually subsided. At length all the signs of extensive pericarditis became present.

Dr. Duplex has kindly given to us the particulars of the following case, that of a lady who was under his care, and was seen by Dr. Gull also in consultation with him.

A lady, æt. 38, was suffering from her first attack of rheumatic fever. She was seized with pain in her ankles and knees. The pain was very severe. There was perspiration, but this was not profuse. During the first five or six days she appeared to go on well, except that she suffered very great pain in her joints. She then began to complain of a sensation which she described as a frightful feeling of suffocation and of inability to breathe. Her sufferings were most distressing to witness. Dr. Gull told us that he examined her heart very carefully. There were no signs to indicate that the heart was affected. Dr. Gull again saw her the next day, and still there were no physical signs of heart disease. On the third day he detected a slight to-and-fro murmur; after that time the to-and-fro murmur became each day more and more distinct, until there were all the signs of extensive pericarditis, with effusion into the pericardium. At the end of three weeks the heart symptoms began to subside. The total duration of this lady's illness, from the commencement of the attack to her total recovery, was eight weeks. The medicine given was Battley's Solution, in a dose of twenty drops, three times daily. A drachm, or even a drachm and a half, was taken in the course of the day. This treatment was continued for three weeks. The diet consisted of milk only, with 6 oz. of brandy.

In all the cases above recorded the symptoms were well marked and acute. Several of the joints were affected. There was pain in the joints, and also redness and some swelling. The pain was often very acute, the patient lying on his or her back, quite helpless, and unable to move. The tongue was thickly coated with yellow fur. The skin was perspiring freely, and in some cases profusely, and the rheumatic odour was well marked. The pulse was full, and the face flushed. The urine was high coloured, the quantity passed in twenty-four hours was diminished, and the specific gravity was increased, ranging from 1025 to 1032.

Cases belonging to this class tend to do well without the aid of any drugs; the patients simply require absolute rest in bed and a milk diet, and the symptoms usually subside in eight or ten days when unaccompanied by any heart disease. But, in order that they may do well and recover speedily, it appears to be absolutely necessary that the patients should rest in bed, that they should not be allowed to get out of bed for any purpose, and that the diet should be limited to light nutritious food.

It is interesting to notice how rapidly, even suddenly, the symptoms subside in such cases. Of this we have an example in the case of David D—, who had great pain in several of his joints, and was unable to move in bed. On the tenth day, about 6 a.m., the pain, as he stated, suddenly left him, and when we saw him next morning he was suffering much less, and was able to move all his limbs.

It is in cases of this kind that the remedies recommended by various physicians have appeared to be of so great service.

There is also another class of cases which tend to do well, but in which the duration of the attack is much greater. We refer to cases attended with the following symptoms—There is more or less pain in the joints; the skin is cool, and not generally perspiring, except during sleep; the tongue is slightly furred; the appetite is often good. The pulse is not full. The urine is often of a natural colour, and very soon becomes alkaline; its specific gravity is not at all, or very little, higher than normal. In these cases, nevertheless, there are often physical signs showing that the heart is affected with rheumatic inflammation.

There is a third class of cases in which the power of recovering is feeble, and which therefore require the aid of medicine. We refer to patients who have had rheumatic fever, and whose hearts are diseased in consequence. In such cases, as is well known, the rheumatic attack often recurs. If the heart has been very much damaged the joint symptoms are often slight and the chest symptoms severe. Cases are seen in which the inflammation in the joints is so little that it is difficult and even impossible to decide whether the changes in the heart are due to rheumatic fever. There are two things, however, that usually assist us in arriving at a conclusion. The dyspnœa has markedly increased, and often comes on in the course of a short space of time, that is, in two or three weeks. There is a history of a previous attack of rheumatic fever, and on inquiry, we learn that there is pain, though it may be but little, in one or two joints. Experience has shown that such cases run a rapid course, and not infrequently terminate fatally. The following example illustrates these remarks.

William H. C—, æt. 12, was admitted as an out-patient under my care at the Victoria Park Hospital, December 22nd, 1865. It was stated that a year and a half ago he had rheumatic fever, and that he was ill eight weeks with it. He appeared to recover well from the attack. During the last two months he appeared to have lost flesh. On admission he said that he had a catching pain in his chest, and he had a very troublesome cough. He looked particularly anæmic. He was short of breath if he ran or made any great exertion, but not otherwise. The cardiac dulness was much increased, lowered and moved to the left. A well-marked systolic bruit was heard over the apex of the heart, also at the angle of the left scapula. He appeared to be suffering from mitral imperfection, with an anæmic condition of the body. He was ordered citrate of iron and quinine and cod-liver oil. Fourteen days afterwards we saw him again, and he appeared to have improved and to be going on favorably. His friends thought that he was decidedly better.

On January 19th, that is, a month after admission, he was not nearly so well. His respiration was quick, and his cough was much worse. He complained of pains in his elbows and finger-joints. His father remarked, "I am obliged to carry him up and down stairs; he cannot walk, his breath is so short." Pulse 120; physical signs the same. From this time he got rapidly worse. On January 27th he was admitted into

Guy's Hospital under the care of Dr. Wilks. There was great dyspnœa; his feet and legs and scrotum were œdematous.

29th.—He spat up a quantity of blood, and on the same day died.

Autopsy by Dr. Moxon.—Pericardium healthy; the heart was very much enlarged; the left ventricle very much dilated, and its walls not attenuated; some soft vegetations on the aortic and mitral valves; the valves, with the exception of the vegetations, were healthy, but the mitral orifice was dilated, and had apparently allowed the blood to regurgitate. The lungs were very much congested. Liver had the nutmeg appearance. Kidneys slightly irregular on the surface, as if from commencing granular degeneration.

In other cases, as is well known, the post-mortem examination often shows some recent pericarditis or endocarditis which has supervened upon old disease of the heart.

For such cases as these the expectant plan of treatment is totally inadequate; they do not progress satisfactorily when treated by rest and light nutritious diet only; they require the assistance of some drug.

We have briefly recorded the last case not only because it is a good illustration of a certain form of rheumatic fever, but also to show the condition of the heart.

In the last number of the 'Reports' we endeavoured to point out that it is often very difficult and even impossible to say whether the heart has or has not been materially injured during rheumatic fever. At the time we referred to a case which had occurred in Guy's Hospital. The patient died of heart disease, apparently the result of rheumatic fever, and the post-mortem examination showed that the pericardium and endocardium were healthy, but the muscular tissue of the heart was diseased. The case was similar to the one which we have just mentioned. In both these cases the pericardium and endocardium were healthy and showed no signs of former disease. For the left ventricle to become so dilated would necessarily require time. Hence we are led to infer that there would have been no bruit during the rheumatic attack to indicate that the part was injured, unless we assume that there had been some endocarditis or pericarditis, all traces of which had disappeared, but this would be simply an assumption.

The urine during the acute stage was generally high coloured, diminished in quantity, and of increased specific gravity. In some cases there was a copious deposit of lithates when the urine cooled, but this was by no means constant. When the symptoms were acute the urine was acid. In the subacute cases the urine was occasionally noticed to be neutral, and sometimes alkaline. With respect to the alkalinity of the urine, we would observe that it is not so often that it is alkaline when passed as that it becomes so in an hour or two. We have several times seen the urine become alkaline in this way after it had been voided. When the symptoms are acute the urine has been noticed to remain acid for many hours, and even three days after it had been voided, thus strikingly differing from the urine passed in the subacute cases, which, as we have already stated, becomes alkaline quickly after being voided. It may have been noticed that in the sthenic cases the urine is, during the acute stage, usually of high specific gravity, and that it continues so while the acute symptoms last. As, however, the pain in the joints, heat of skin, coated tongue disappear, the urine falls in specific gravity and, by degrees, as the patient recovers, falls lower and lower until a very low point is reached (in one case as low as 1005), and then it rises again to its normal specific gravity.

A question may be asked—Does the urine always become alkalized as the attack subsides? That it often does, it would be difficult to dispute, but it certainly does not always. In some of the cases that have done very well, we might almost say, the best of any, the urine did not become alkaline; but day by day it remained acid.

Of twenty-nine cases of rheumatic fever admitted into Guy's Hospital during the last year, in sixteen there was evidence, on admission, of heart disease. Of these sixteen, eight appeared to have mitral disease on admission, and the same when discharged. Four of the sixteen appeared to have aortic valve disease (or there was a systolic murmur at the base, which was carried along the aorta). In the remaining four there was a murmur on admission, which disappeared as the patient became convalescent. In one case there was an anæmic murmur. In one fatal case there was early pericarditis. In one case there was no murmur on admission, but one was developed within two or three days, it appeared to be due to pericarditis, and in the same case a mitral murmur was heard when the patient was convalescent. In ten cases there was no murmur heard, either at the time of admission, during treatment, or during convalescence.

Twelve of these twenty-nine cases were treated with lemon-juice; seven with mint water; three with mint water and a little opium; three with the fixed alkalies; two with alkalies and lemon-juice; one with blisters. One patient was kept in blankets, no medicine being administered.

Of the ten cases in which the heart was free from any bruit, five were treated by lemon-juice, two by alkalies, one by blisters, one by blankets, one by mint water.

It may be noticed that in only one case was a bruit developed while under treatment, which may suggest the question as to whether it is common in hospital practice to find heart disease developed while under treatment.

It seems to be far from common according to the experience of physicians who advocate different systems of cure. But it appears to us, that until it is decided what is the proportion of cases which escape heart affection with treatment by rest and light diet only, it will be very difficult to decide as to the amount of influence any system of cure has exercised in preventing such disease.

CASES

OF

OPERATIONS ON THE LARYNX.

By ARTHUR E. DURHAM.

It was my intention to have discussed fully in the present volume of our 'Reports' the comparative merits of the various methods that have been adopted for the removal of growths, and for the extraction of foreign bodies, from the larynx. The necessary space, however, is not at command; I content myself, therefore, with giving the following details of two cases recently under my care.

In each case the larynx was freely opened by an incision in the middle line, through the thyroid cartilage and adjoining parts, and in each the result, as far as I am at present able to judge, has been most satisfactory and successful.

Case 1.—*Numerous warty growths in the larynx of a child, seen by aid of the laryngoscope; removal after complete exposure of the interior of the larynx; successful result.*

Mary F—, residing at Plaistow Marsh, was first admitted into Guy's Hospital on the 1st of August, 1862. She was at that time about nine years of age; her general health had been tolerably good, but she had been subject to attacks of "sore throat" and "loss of voice." For some weeks previous to admission she had been suffering from "one of her sore throats;" the symptoms had gradually increased in severity until she was scarcely able to breathe or swallow. On admission into the Clinical Ward, under the care of Dr. Pavy, her dyspnœa was so severe that her father states that "when he left her he never expected to see her alive again."

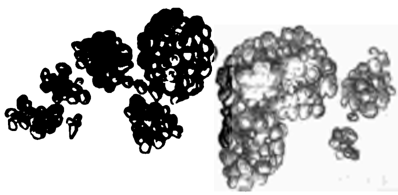
Within a few hours of her admission tracheotomy was performed by the then house-surgeon, Mr. Holmested. She breathed easily through the tube, and gradually recovered her health and strength, which had been considerably depressed by her sufferings. She left the hospital on the 22nd of September, comparatively well in health, but quite unable to speak audibly or to breathe through the larynx. She was readmitted on the 1st of October for the purpose of having the tube changed, &c., and remained in the hospital for about three weeks. During this period I was requested to make a laryngoscopical examination of the case. I attempted to do so, but utterly failed to arrive at any satisfactory result in consequence of the resistance of the patient. Since she left the Clinical Ward for the second time, on the 22nd of October, 1862, she has been more or less constantly under my observation. She has been admitted into the Hospital, under my care, no fewer than ten times, and on each occasion has stayed two or three weeks or more; she has also frequently attended among my outpatients.

From time to time I made numerous and persevering efforts to discover the precise condition of the larynx by the aid of the laryngoscope, but from time to time was defeated by the resistance of my refractory patient; at length, however, she became more manageable, and her tedious case was then speedily brought to a successful issue.

On the 16th of May, 1866, the patient, now about thirteen years of age, was admitted into Petersham Ward, under my care. She was much out of health; she complained of difficulty of swallowing, and of increasing discomfort and frequent pain about her throat; she was quite unable to breathe through the larynx; she could produce no audible laryngeal sound whatever, but was able, with some difficulty, to make herself understood by the motions of her lips and tongue.

In the course of two or three weeks she improved somewhat in general condition, and then gradually learnt to submit to the use of the laryngoscope. After repeated attempts I succeeded in seeing quite distinctly a considerable warty growth on the left false vocal cord, and in making out, though somewhat less positively, that the whole of the interior of the larynx was blocked up by similar growths.

On the 27th of July, with the assistance of my colleagues Messrs. Birkett and Forster, I proceeded to operate in the following manner:—Chloroform having been administered, I passed a small, curved, sharp-pointed knife straight through the skin, superficial structures, crico-thyroid membrane, and mucous membrane of the larynx, and cut directly upwards in the middle line through the thyroid cartilage, &c., thus dividing all the structures with one incision as high as the thyro-hyoid membrane, which was only slightly cut. I was subsequently obliged to divide the cricoid cartilage also, in order to expose the interior of the larynx to a sufficient extent. Some hæmorrhage followed the first incision, and the blood which was drawn into the trachea gave rise to somewhat severe spasmodic coughing. When the bleeding was arrested and the spasmodic efforts had subsided, the lips of the incision were drawn apart as widely as necessary; it was then seen that the whole of the interior of the larynx was occupied by warty growths. These growths were very numerous; they varied in size and situation. The largest was that already mentioned as distinctly seen by aid of the laryngoscope on the left false vocal cord. There were none, or only two or three minute ones, *below* the true vocal cords, but, with this exception, the mucous membrane of almost every part of the interior of the larynx was covered with them. All were carefully removed; some were cut off with scissors, others were twisted off with forceps. The slight bleeding which ensued was easily arrested by free exposure to the air, and temporary pressure by means of sponges.



The accompanying woodcut somewhat rudely represents the *appearance* of the growths. It conveys a tolerably accurate idea, however, of the *sizes* and *general forms* of the greater part of them; very many minute ones are not represented.

The wound was closed by sutures and strapping, and the patient was conveyed to bed. The tracheal tube, which had not been in any way interfered with during the operation, was left in. In the evening the patient was in every respect very comfortable; she passed a good night, sleeping well, breathing easily, and being only very occasionally disturbed by slight cough, accompanied by expectoration, now and then tinged with blood.

The next day, when the tracheal tube was closed by the finger, the patient could breathe perfectly well through the larynx, and could give utterance to various sounds, which, though feeble, were distinctly laryngeal. It was, indeed, difficult to prevent her from injuriously testing her newly recovered vocal powers.

Her progress was most satisfactory; no bad symptom of any kind occurred. On the fourth day after the operation the stitches were removed. On the seventh day the wound had entirely healed up. On the tenth day the tracheal tube was removed by my colleague Mr. Forster, who kindly took charge of the case during my absence from town. The opening, in which for just four years the tracheal tube had been worn, very speedily closed up; and on the 22nd August, not quite a month after the operation, the patient left the hospital in excellent health and spirits, breathing easily and well in the natural way, and able to speak quite audibly, without any exertion.

I saw her last on the 7th of September. She could not have been in a more satisfactory condition, and she said that her voice was improving daily.

Remarks.—The successful issue of this case appears to me to do even more than *justify* the apparent severity of the operation which was adopted.

I know no other method by which such a collection of growths could have been removed so speedily, so completely, and, I venture to add, so effectually and so safely.

CASE 2.—*Large cherry-stone impacted in the larynx; impending death; tracheotomy; artificial respiration; subsequent removal of cherry-stone by means of incision through the thyroid cartilage; perfect recovery.*

(I am indebted to Mr. H. MORRIS, B.A., the Dresser, and to Mr. JOSHUA DUKE, for full notes of this case.)

Henry B—, æt. $7\frac{1}{2}$, was admitted into Luke Ward, under my care, on the 26th June, 1866.

About three quarters of an hour before he was brought to the hospital this little boy was trying to "blow his hardest" through a "pea-shooter." Unfortunately, before "blowing," he "drew in his breath" with the pea-shooter to his mouth. The charge, consisting of an almond- and a cherry-stone, consequently passed into his air-passage. The neighbours tried their best to relieve him by the introduction of their fingers, and by holding him "topsy-turvy," and striking his back. It is stated that under this treatment the foreign body or bodies passed further down. Ten minutes later he was directed to cough, and it would appear that during coughing the cherry stone was forced up again, and became fixed in the larynx. Extreme difficulty of breathing came on, and he was brought to the hospital in a state of intense distress. He was blue and cold, but apparently quite conscious.

On admission it was evident that suffocation was impending; I therefore at once proceeded to open the trachea. Before this could be accomplished, however, all efforts at respiration had ceased. The operation was completed as quickly as possible, and artificial respiration by the Sylvester method immediately commenced; this was kept up for about ten minutes. The child gradually came round, and was soon sufficiently recovered to be conveyed to bed; both sides of the chest dilated well. During the operation there was considerable hæmorrhage from one of the much-distended thyroid veins.

At 10 p.m. the patient had recovered from the immediate effects of the operation and was pretty comfortable, but he could not breathe at all through the larynx, and I was unable to see *into* the larynx by aid of the laryngoscope. Chloroform was administered through the tracheal tube. When insensibility was perfectly produced, I took out the tube; I then passed an elastic catheter through the opening as rapidly as possible upwards, and my finger from the mouth downwards towards the larynx. Some fragments of almond were brought out of the mouth, but the catheter could not be passed through the larynx. We were obliged to discontinue our attempts on account of impending suffocation. The tracheal tube was re-introduced, and, by means of artificial respiration, the child was again brought round. During the night the tube became blocked up and fixed, and for the third time it was necessary to have recourse to artificial respiration.

During the next three or four days the child suffered from considerable constitutional disturbance, and indications of pneumonia appeared; he gradually recovered. It was considered expedient to allow him to regain his general health, as far as possible, before making any fresh attempt upon the larynx.

Laryngoscopical examinations were several times made during the ensuing fortnight. It was impossible to see *into* the larynx, the mucous membrane being swollen and œdematous; no air whatever passed through the larynx.

On the 28th July, chloroform having been administered, I again removed the tracheal tube, and endeavoured to push a catheter or probe upwards into the larynx. It was, however, quite impossible to do this without serious risk; I therefore resolved to open the larynx freely, to such an extent as might be necessary for the extraction of the foreign body, whatever it might be.

On the 30th July, chloroform having been administered, I made a vertical incision in the middle line on to the thyroid cartilage and crico-thyroid membrane. All hæmorrhage having been arrested by the ligature of three or four vessels, I proceeded to cut through the above-named structures as well as the mucous membrane, taking care to keep strictly in the middle line. On separating the parts the cherry-stone was at once seen completely filling up the middle of the larynx, stretching from ventricle to ventricle, and firmly held between the two false vocal cords above and the two true vocal cords below. It was easily extracted by means of a pair of dressing forceps; there was no hæmorrhage. The lips of the wound were brought together by sutures and strapping.

On the 2nd August the patient was up and walking about the ward. He could breathe easily through the larynx, but had not recovered his voice. The progress towards perfect recovery was uninterrupted, though, perhaps, not rapid.

On the 17th August he left the hospital. The wound was almost entirely healed. It was, however, considered better that he should continue to wear the tracheal tube for a time, although he could breathe very well through the larynx, and could both speak and sing when the orifice of the tube was closed.

On the 10th September he was readmitted, and on the day following I removed the tracheal tube. In the course of a few days the opening closed up, and on the 15th he went home in good health, and able to laugh, talk, and sing almost as well as ever.

The one remark I would especially make in connection with the foregoing case is this:—The event clearly showed that it would have been far better to have performed at first, and without hesitation, the operation which was only adopted at last as a *dernier ressort*. The cherry-stone, firmly fixed as it was, could scarcely have been removed in any other way until ulceration of the mucous membrane, and at least partial destruction of the vocal cords, had taken place.

ON

DISEASES OF THE RETINA;

WITH

REMARKS ON ITS STRUCTURE AND NORMAL CONDITIONS.

By C. BADER.

Anatomical and General Remarks.

The retina is spread out between the choroid and the vitreous substance, and extends from the optic disc to the ora serrata. It is thickest at a little distance from the yellow spot, where it measures about $\frac{1}{9}$th of an inch in thickness. It gradually becomes thinner as it approaches the ora serrata. Through decrease in the number of its component parts, half an inch outwards from the yellow spot it measures about 0·015 mm.

It is firmly attached along the ora serrata, and round the optic disc, but only loosely to the surface of the choroid. The opening through which the optic nerve passes is termed "the retinal aperture." The part occupied by the optic disc is termed "the blind spot." The region of the yellow spot lies to the outer or temporal side of the optic disc. In this region we distinguish the centre or "centre of the retina" (by some termed "the fovea centralis"). The colour of the region differs from that of the centre as well as from that of all other parts of the retina. The "centre of the retina" is about $\frac{3}{16}$ths of an inch from the centre of the optic disc, and lies somewhat lower.

The serrated line of attachment along the ora serrata is termed "the margin" or "periphery of the retina."

We further distinguish between the "inner surface" of the retina, which is next the vitreous chamber, and the outer surface, which is next the choroid.

Colour of the retina when viewed with the ophthalmoscope. —In fair persons the retina appears transparent; in those with brown irides, very faintly grayish; and in those with dark eyes of a bluish tint. This tint decreases from the optic disc towards the ora serrata, and is attributed partly to the pigmentation of the choroid, and partly to the effect of light reflected from the layer of optic nerve-fibres, which layer in dark eyes gives the surface, at the retinal aperture, a delicately striated appearance, the striæ radiating from the optic disc.

A peculiar colour is observed in most healthy eyes in the *region of the yellow spot.* This region, as regards colour, has a transversely oval shape, with the long axis of the oval horizontal.

The centre, looked upon as the most sensitive part of the retina, lies about $\frac{3}{16}$ths of an inch from the middle of the optic disc, and on a level with its outer and upper margins. In eyes with light blue irides this region sometimes differs but little from the rest of the retina; but sometimes a grayish halo, surrounding a reddish minute dot (the centre), of about one sixteenth of an inch in diameter, is observed. In eyes with brown irides, and especially in those with black irides, the centre appears as a dark spot, surrounded by a grayish-yellow halo, and the latter by a grayish semi-transparent one, which shades off into the transparent retina.

The retinal blood-vessels. — The blood-vessels of the retina enter and leave the eye at the optic disc. Within the eye they do not anastomose with those of the other tunics. All the blood, therefore, which goes to and comes from the retina has to pass through the optic disc. The walls of the vessels appear transparent. The arteries subdivide in the optic nerve before reaching the retinal aperture. Thus, from near the middle of the optic disc two large branches pass upwards, and two downwards and across the optic nerve fibres, into the retina. Close to the optic disc they subdivide into smaller branches in the retina; a large number of these turn towards the region of the yellow spot, above and below which they further subdivide. The arteries break up into capillaries. Besides these, there are, however, many small vessels which enter and leave the retina on the right and left of the aperture.

In young persons sometimes three or four arteries, and as many veins, can be seen passing through the optic disc. In middle-aged persons generally one or two large arteries only can be observed. The number decreases as age advances. The greater number of vessels are observed in the outer or temporal part of the retina. Peculiar twistings of the arteries are often seen, especially in the optic disc.

The veins which return from the retina also pass through the optic disc; they run near the arteries, their arrangement being similar.

The arteries can be distinguished by their bright red colour, and straight course. They are thinner, and not infrequently present a double contour, *i. e.* the blood seems to glide along the sides, while the central parts of the vessels appear empty. The veins are darker, more numerous, larger, and more variable in diameter. They are readily distinguished from the arteries by pressing gently upon the eyeball with the finger (while examining with the ophthalmoscope), when the pulsation of the arteries in the optic disc becomes at once apparent.

Pulsations of the retinal blood-vessels. — The relations between the vitreous substance and the blood circulating in the retina are such that the slightest pressure of or upon the blood-vessels becomes apparent, especially in the optic disc. It causes visible pulsation of the veins, and, if at all considerable, also of the arteries. The pulsation of the veins can be seen in every retina, and at all ages. Large flat veins, tapering off as they leave the retina, show it best. It consists in an irregular filling of some and emptying of other parts of the vein. The contraction of the vein advances from the centre towards the margin of the optic disc, while the dilatation commences at the margin. A short pause intervenes between the maximum of dilatation and the commencement of contraction. All movements of the eye, all efforts at accommodation for near objects, as well as many changes in the respiration, cause increase of the venous pulse. The blood, thrown into the retinal arteries with every systole, has to pass through the capillaries to reach the veins. The arteries momentarily become dilated, and the quantity of blood in the eye increased. The vitreous substance during this moment is pressed upon, and, in its turn, presses upon the optic disc and its blood-vessels. This pressure becomes perceptible in the veins in the optic disc, in consequence of their becoming dilated before the quantity of arterial blood which causes the increased pressure has reached the veins; and the current of blood in these is impaired, until the pressure of the arterial blood (during the systole) has, through the capillaries, reached the veins.

The systole, after a time, having passed, the pressure of the vitreous substance becomes lessened, the veins become still more dilated, and the blood readily escapes from the eye. This visible movement of blood in the veins is termed the venous pulse.

The circulation in the capillaries is never interrupted.

The pulsation of the retinal arteries in the optic disc is not visible under ordinary circumstances. In rare instances it occurs spontaneously, and appears due to a peculiar arrangement of the vessels. It requires artificial pressure upon the eyeball, or a morbidly increased tension, to render it visible. A brisk filling and emptying of the arteries in the optic disc, synchronous with the pulse, is observed when gentle pressure is made upon the eyeball. The retinal arteries are thus pressed upon the margins of the sclerotic and choroidal apertures, beyond which the visible pulsation does not extend. An entire displacement of the blood from the arteries, with cessation of the pulse, occurs when the pressure is greatly increased.

The minute structure of the retina.—On examining the inner surface of the retina, when fresh, we observe, except over the yellow spot, faint delicate lines radiating from the optic disc, and running side by side. These are the bundles of optic nerve-fibres. Beneath and among these we see the blood-vessels, and also beneath the fibres an agglomeration of granules. When the retina is viewed on the outer surface we perceive the rods and bulbs which occupy this entire surface, and beyond these we see again an agglomeration of granules.

In sections taken from a spot of the retina, near the outer margin of the optic disc, and about midway between it and the yellow spot, and made parallel with the optic nerve-fibres, and through the entire thickness of the retina, we observe a variety of structures, which are piled upon and connected with each other, and which appear arranged in layers. These, commencing at the inner surface, *i. e.* with the optic nerve-fibres, are—

1. The layer of optic nerve-fibres.
2. The layer of ganglionic cells—"the ganglionic layer"—in which is found the larger number of blood-vessels.
3. The granular layer.
4. The inner granule layer.
2. The inter-granule layer.
6. The outer granule layer.
7. The layer of rods and bulbs,—"the bacillary layer" or Jacob's membrane.
8. Intervening between layers 6 and 7 is a transparent line of demarcation, into which many of the rods are inserted, and from which many fibres start, to pass through the retina in a direction perpendicular to that of the optic nerve-fibres; this line by some is termed the "outer membrana limitans," and is described as a separate layer.

A similar line of demarcation (a transparent membrane, when viewed from the surface) is seen upon the layer of optic nerve-fibres intervening between these and the hyaloid membrane. This is the structure into which the fibres ("radial fibres") which pass across the retina perpendicularly, and connect the different layers and their elements with each other, are inserted. This line—also described by some as a layer—is the inner membrana limitans, and the fibres inserted into it may be regarded as the connective tissue of the retina.

1. *The layer of optic nerve-fibres.*—These fibres, having arrived at the upper margin of the retinal aperture, pass from the optic disc upon the inner surface of the retina. The layer is thickest at that part, and gradually becomes thinner the nearer it approaches the periphery of the retina. Those fibres which lie nearest the surface of the retina enter into it, and, judging from the examination of numerous sections, anastomose with the ganglion-cells.

2. *The layer of ganglion-cells.*—The cells are large, vary in size, are multipolar, with several nuclei, and are placed at certain distances from each other. Some proportion seems to exist between their number and the thickness of the retina. The thinner the retina, the more distant are they from each other. They are level with the large blood-vessels. The centre of the yellow spot, where it is level with this layer, is occupied by a cluster of nucleated cells, which are somewhat larger than the granules.

3. *The granular layer* around and beneath the ganglion-cells is an exceedingly delicate layer, well supplied by capillaries. It is, however, little known as regards its minute structure.

4. *The inner granule layer* consists of cells, which on the application of a weak solution of chromic acid, show two or three nuclei. The granules are round, and separated from each other by connective tissue; they are piled upon each other in a certain order, and decrease in number towards the periphery of the retina.

5. *The inter-granule layer* consists of delicate fibres, many of which belong to the connective tissue, and pass through the retina from one layer of granules to the other. Numerous very fine fibres, running at right angles to the former, *i. e.* parallel with the layer of nerve-fibres, are found in this layer, especially at and near the retinal aperture.

6. *The outer granule layer.*—The granules of this layer are, when fresh, transparent; some are round, others oval, resembling pus-cells after having been exposed to chromic acid. Many appear, when fresh, striped transversely, one or two dark parallel lines being perceptible on their surface. The granules are piled upon each other, and lie free in the connective tissue, decreasing in number from near the centre towards the periphery of the retina. Each granule is separated from its neighbour by one filament of "connective tissue."

7. *The layer of rods and bulbs.*—In the region of the yellow spot are elements occupying this layer, larger than the rods, and smaller than the bulbs in other parts of the retina. They are of equal size, standing side by side and perpendicularly upon the granules of the hexagonal cells of the choroid. Further from the yellow spot, we find the usual narrow trans-

parent rods, and at intervals which increase from the yellow spot towards the periphery the somewhat pear-shaped bulbs.

There seem to be two kinds of rods—those which cannot be traced beyond the outer membrana limitans, and those which can be traced into the retina, many of which seem to be continuations of the fibres of the connective tissue.

The connective tissue of the retina ("its framework," "the radial fibres").—This layer consists of delicate nucleated fibres, somewhat thicker than nerve-fibres, which run across the retina and anastomose freely with each other through the medium of filaments (termed "glioma,") and enclose within their anastomoses the granules, &c., of the retina. On reaching the layer of nerve-fibres many seem to split up into smaller fibres, which diverge from each other, and after having passed across this layer insert themselves, "open out," into the inner membrana limitans. A similar insertion is found at the so-called outer membrana limitans. The relation of these fibres to the rods and bulbs is not well understood; many of the rods seem to be continuations of these fibres.

The fibres of the connective tissue intervene between the elements of the retina and the optic nerve-fibres at the retinal aperture, and likewise separate these elements from the zonula along the ora serrata.

In the region of the yellow spot the fibres of the connective tissue have a course which is peculiar. Here, after having advanced perpendicularly into the retina for some distance, enclosing the granules of the outer granule layer (in piles of generally four granules), they assume a wavy (undulating) course; those at the centre of the yellow spot pass directly towards the inner membrana limitans, while those adjoining them pass off at an angle, and pass obliquely through the retina. Having for some distance continued their wavy slanting course, they again ascend perpendicularly, to reach the inner membrana limitans.

These wavy fibres intervene between the outer and inner granule layers.

The connective tissue and the outer and inner granule layers are the most durable structures of the retina, while the layer of ganglion-cells and the granular layer seem most susceptible of destruction.

Vision.

All parts of the eye necessary for vision being healthy, to see an object distinctly it must have a certain size, be placed in a good light, and at a distance which allows the rays which come from the object to be brought to a focus in the centre of the yellow spot of the retina, where a well-defined inverted image of the object is formed. This image, through the fibres of the optic nerve, must be conveyed to the brain, there perceived, and again projected in an inverted direction towards the object. The layer of rods and bulbs is supposed to be the perceptive part of the retina. At the yellow spot, where the most distinct vision takes place, is a single layer of small bulbs, with a few ganglion-cells and optic nerve-fibres intervening between them and the vitreous chamber. Thus, the rays of light reach the bulbs more directly than in any other part of the retina. The undulations of the rays of light, through passing into the elements of this layer, produce a change which is the perception of the object from which the rays emanated.

The part played by the other elements of the retina in the act of vision is not understood. The optic nerve-fibres convey the impression received from the retina. They themselves do not possess perception of light in the optic nerve nor upon the retina. This is well known as regards the optic disc or blind spot. That the same applies to the optic nerve-fibres upon the retina is inferred from the fact that the shadows of the retinal vessels in our own retinæ can be made visible to ourselves in a dark room, by turning the cornea inwards towards the nose as much as possible, and by then moving to-and-fro upon the sclerotic the image of the flame of a candle or gaslight, brought to a focus upon the sclerotic by means of a 2″ or 3″ convex lens. The blood-vessels ramify among and beneath the layer of optic nerve-fibres, and, their larger branches being made visible, the perceptive part of the retina is supposed to lie behind the layer of optic nerve-fibres.

To determine *the acuteness of vision* (= V) of an eye, it becomes necessary to ascertain the size of the smallest object which can still be seen distinctly, and to devise a mode of expressing the result obtained, and with it the degree of the acuteness of vision.

The smallest angle under which by the normal eye an object can still be distinctly recognised is one of five degrees ($= 5^\circ$); *e.g.* take the letter I, which we suppose to be one inch high, its width being equal to one fifth of its height; place that letter at 50′ from the normal eye under examination, and it can be recognised. To ascertain under what angle it is seen, we have to draw a straight line from the extreme points, *i. e.* from the upper and lower ends of the letter to the two corresponding points of the small inverted image of the letter in the retina. These lines cross each other within the eye and at the point of crossing form equal angles. If the letter is brought nearer, say, to a distance of 40′ or 30′, the image on the retina is larger, and the letter, therefore, can be better perceived, because its image is spread over a larger number of perceptive elements of the retina. If the letter is held further off than 50′, say at 55′ or 60′, it can no more be perceived distinctly, because the image on the retina is too small. If we measure the angle on either side of the point of crossing which the lines form with each other when drawn from the two ends of the letter to the corresponding point of the image in the retina, the letter being held at 50′, we find that each angle measures 5°, and express this by saying that the letter I is seen under an angle of 5°. If the letter is held nearer the angle becomes larger, if it is held further off it becomes smaller. The further from the eye the letter I is held the smaller is its image on the retina, and the smaller the angle formed by the straight lines drawn from the two ends of the letter to those of its inverted image in the retina, and the greater the acuteness of vision if this image can still be perceived. Therefore, by determining the degrees (*i. e.* the value of the smallest angle which corresponds to the image of an object), which can still be recognised by the eye, we determine the degree of acuteness of vision. This angle is termed *the angle of vision,* or the visual angle. Some persons see objects under a smaller angle, and therefore possess a greater acuteness of vision. But people in general, to see an object distinctly, must see it under an angle of 5°. It is not necessary directly to measure this angle as long as we possess a series of objects the sizes of which are known, and the furthest distances at which they are still recognised by the normal eye have been ascertained by experiment. For practical purposes, as objects with which to ascertain the acuteness of vision, "test types," or series of letters of different determined sizes, have been introduced, of which those of Dr. Snellen, of Utrecht (Holland), are the most perfect.

An explanation of the principles adopted in selecting and arranging the letters, and of the conclusions which may be drawn from the results of examination with the "test types," are given by Dr. Snellen, together with the types. Those who may not be able to obtain the "test types" can arrive at tolerably accurate results by using any other types, as long as the thickness of the strokes of the letters employed is equal to one fifth of their height. The height of the letters should be the same for each series, but vary according to the distance at which they are held from the eye. Experiment has shown that the furthest distance from the eye at which a letter measuring about one fiftieth of an inch ($=\frac{1}{50}''$) in height can be distinctly recognised by a normal eye, is one foot ($=1'$). That, when measuring about $\frac{2}{50}''$ the furthest distance at which it can still be recognised is $2'$; when measuring about $\frac{5}{50}''$ or $\frac{1}{10}''$, it is $5'$; when measuring about $\frac{10}{50}''$ or $\frac{1}{5}''$, it is $10'$; for letters $\frac{20''}{50}$ in height $20'$; for those of $\frac{100''}{50}$ it is $100'$, &c., provided always that the thickness of the stroke of each letter be one-fifth of its height. We may thus arrange a series of letters, numbering nine each series, so that the letters of the series No. 1, or of series I, measure each $\frac{1}{50}''$ in height, and should be recognised at $1'$, and not further; those marked II, or No. 2, should be recognised at $2'$; those marked at XX., or No. 20, at $20'$, &c. &c. The number above each series, therefore, indicates the distance, in feet, at which the eye under examination, if healthy, should recognise each letter of the series, and the acuteness of vision is normal if No. I can be recognised at $1'$ ($V = \frac{1}{I}$); II at $2'$ ($V = \frac{2}{II}$); XX at $20'$ ($V = \frac{20}{XX}$); C at $100'$ ($V = \frac{100}{C}$).

This method expresses the degree of acuteness of vision by a fraction, of which the number above the horizontal line represents the distance at which the test letters can be recognised by the eye under examination, and the number below the horizontal line the distance at which the test letters can be recognised by the normal eye:—*e. g.* $V = \frac{1}{1}$, or $V = 1$, signifies that at 1′ letters of No. 1 (*i. e.* those which at 1′ should be recognised by a normal eye) can be recognised by the eye under examination, and that therefore that eye is normal as far as acuteness of vision for objects placed at 1′ from the eye goes. Instead of writing $V = \frac{1}{1}$, we may then write $V = 1$. $V = \frac{20}{XX}$ signifies that at 20′ letters of No. 20 (*i. e.* those which at 20′ should be recognised by a normal eye) can be recognised by the eye under examination, and that therefore that eye is normal as far as acuteness of vision for objects placed at 20′ from the eye goes. Instead of writing $V = \frac{20}{XX}$, we may write $V = 1$. If letters which should be recognised at 20′ can be recognised at 10′, we write $V = \frac{10}{XX}$, or $\frac{10}{20}$, or $V = \frac{1}{2}$; if they can only be recognised at 5′, we write $V = \frac{5}{XX}$, or $\frac{5}{20}$, or $V = \frac{1}{4}$, &c. &c. The number above the horizontal line always expresses the distance at which the test letters can be recognised by the eye under examination, and the number below the horizontal line the distance at which the test letters can be recognised by the normal eye, or ought to be recognised by the eye under examination if normal. The acuteness of vision is somewhat influenced by the size of the pupil. It is increased in weak light when the pupil is dilated, and in strong or diffused light when it is contracted. We should therefore pay attention to the degree of light employed in the examination. The range of accommodation and the acuteness of vision diminish *as age advances.* The former is shown by the necessity of holding small objects further from the eye in order to see them distinctly, the latter by the eye being no more able to distinguish objects of certain sizes at the same distance at which they could be recognised in youth.

The images formed in and perceived by the retina and the functions of the optic nerve-fibres become less perfect. Up to the age of forty, vision remains about normal. At the age of sixty it has already much decreased, so that, for instance, letters of No. 20 can only be recognised at from 15″ to 16″. At the age of eighty it is about half as good as it was at forty.

The field of vision (F).—We distinguish between direct and indirect vision. In direct vision an object is perceived which lies opposite the yellow spot of the retina, one or both the visual lines being directed to the same point of the object. In indirect vision objects are perceived which lie opposite other parts of the retina than those used in direct vision. The images from objects so placed are formed on more excentric parts of the retina; they are perceived indirectly, and the retina refers—"projects"—the impressions towards the objects which produced them. The place occupied by the image in the retina determines the part of the field of vision upon which the image is projected. Any object perceived indirectly, while one or both visual lines are directed to one point of an object, lies in the field of vision. Having determined V, or the acuteness of direct vision, we proceed to ascertain F. The limits or boundaries of the field of vision F are given by the most excentrically placed point of an object which can still be perceived while one or both visual lines are directed to the central point of the object. We must distinguish between F, or field of vision, and field of fixation; the latter comprises those points in F, or the field of vision, which, without altering the position of head or body, can still be seen directly.

The fields of vision (F) of one eye and of both eyes together have been measured. That of one eye is sharply divided into lateral halves, a temporal and a nasal half (see Hemiopia). A line drawn through the points which limit the field of vision (F) forms an ellipse. Vertically, the field (F) measures about 160°; horizontally, about 170°. When ascertaining F (the field of vision) for one eye (see p. 559), we must keep the fellow-eye closed. Great prominence of the margin of the orbit and of the bridge of the nose limit the boundaries of the field.

The percipient elements of the retina decrease in a certain proportion from the centre, or yellow spot, towards the periphery, and more rapidly in the vertical than in the horizontal direction. Having ascertained the extent or boundaries of the field of vision, we may wish to determine the acuteness of the retina as regards the perception of objects situated in different parts of the field (for this see pp. 559, 560).

Modes of ascertaining the extent of the sensitive part of a *retina, or the field of vision of one eye* (*extent of F with one eye*).—A record of the extent of the sensitive portion of the retina, to enable us to compare the course of retinal and other changes, the effect of treatment, &c., may be made in the following manner :—A sheet of dark blue dull paper, size two feet square, with a small white cross traced in the centre, and a vertical and horizontal line intersecting each other at the centre of the cross and dividing the paper into quarters, is fixed upon a wall. The patient stands at the distance of 12″ from the paper, facing the white cross, which should be on a level with the eye we propose to examine, while the other eye is kept closed.

The patient is directed to look steadily and continually at the cross while we move a piece of white chalk fixed on a black handle from the margin of the blue paper towards the cross. A mark is made at the spot where the white disc of chalk becomes first visible. Repeating this movement from different parts of the margin of the paper towards the cross, and not from the latter towards the margin, we obtain a series of points which correspond to the most peripheral sensitive portions of retina. Through these we draw a line, and the figure thus obtained represents the extent of the sensitive portion of retina, or, in other words, the limits of the field of vision of the eye under examination. This record, if taken from an impaired retina, should be compared with tracings of the limits of the field of vision of the retina of a corresponding healthy eye.

The field of vision (F) thus obtained is termed the quantitative F, while the so-called qualitative F refers to the perception of letters at varying distances from the white cross. The limits of both kinds can be marked on the same sheet of paper, and, measuring horizontally and vertically from the cross, we state that the quantitative and qualitative perception horizontally outwards and inwards, and vertically upwards and downwards, amounts to so many inches.

A useful mode of ascertaining the sensibility of the more excentric parts of the retina, and with it the limits of F, is the following :—Suppose we wish to examine the retina of a patient's right eye; we place ourselves at a distance of two feet from and facing the patient, and then direct him to look steadily at our left eye, which during the examination is continually directed to his right eye. If, by moving our hand opposite the excentric parts of the patient's retina, we find that he perceives these movements at the same distances as we do with our healthy retina, we suppose the peripheral portions of his retina sensitive. The patient has to look at our right eye, while his left eye is under examination.

Each part of F refers to the retina opposite that part; suppose *e.g.* that, moving the hand in the outer and upper quarter of the field, its movements are not perceived by the patient, we infer that an alteration of sensibility of the inner and lower quadrant of the retina exists.

Whatever modes of ascertaining the sensibility of the excentric parts of the retina be adopted, care should be taken that the position of the patient's eye and its distance from our own eye remain unaltered during the examination, and that they are the same whenever the examination is repeated.

In *the formation of ideas of the sizes, forms, and distances of objects*, we are guided by the sizes, &c., of the images of these objects in the retina, and by the degree of effort of the power of accommodation required to produce distinct images. For the same purpose we make use of certain movements of the eyes, of the head, and of the body.

If we have a knowledge of the exact sizes, forms, and distances of a few objects, we possess a guide in forming our judgment of the sizes, &c., of others. Thus, by a continuous repetition or modification of the muscular efforts which are necessary to see in succession different objects and their details, we acquire experience as regards the muscular power which, each time, is required to recognise the size, &c., of any particular object, and to this experience we appeal when examining new objects. The size of the image of any object on the retina varies with the distance of the object; and knowing the sizes of some objects and those of their retinal images, we possess a second means by which we improve our ideas as to the sizes, distances, &c., of other objects.

The idea of bodily form of any object is chiefly developed through the dissimilarity of the retinal images of the two eyes. While looking at one point of an object this point appears single, and all others appear double. By then viewing in succession other points of the same object, we each time alter the convergence of the eyes as well as the sizes of the two retinal images, and with these the distances of those points of the object which are seen double. We thus gradually acquire an idea of what is termed the form or solidity of an object.

We must more particularly discuss the mode in which the idea of size is developed. Suppose we distinctly see a line of unknown length at a known distance, and are required to state what the length of the line amounts to. To do this we direct the visual line first to one end of the line, then along the line to the other end. The visual line has thus to describe a certain angle, or to travel over a certain distance, and upon the estimation of this angle or this distance (that is, upon the amount of muscular power required to describe the angle) depends our statement as regards the length of the line; *e. g.* if we look from one end of a line which is one foot long to the other end, our visual lines have to travel a shorter distance than when looking in the same way at a line which is five or ten feet long, or more. Less muscular power is required to look over the first than over the other lines, and it is from the experience acquired by repeating the experiment unconsciously upon the objects which surround us that we finally possess the power of rapidly stating the size of any object—its height by estimating the distance of its two extreme points from each other in a vertical direction, its width by estimating the two extreme points in a horizontal direction.

Our estimation of the *rate of motion of an object* depends upon the muscular power which is required to maintain a distinct view of the object in motion. The sensation of the movement of our muscles gives rise to the idea of motion. To form an idea of the rate at which other objects move, we must either be at rest, or we must know our own rate of motion.

Perception of colours, and its anomalies.—Colours, to be recognised as such, must occupy a certain amount of surface and be illuminated by a certain quantity of light. These conditions vary with different colours, *e. g.* a red square figure of a certain size, and on a white ground, if badly illuminated or very small, may still be recognised as regards its square shape, but no longer as regards its colour, which appears black. A blue square figure appears black at a shorter distance from the eyes than a red one.

The power of distinguishing colours is greatest in the region of the yellow spot, and decreases towards the periphery of the retina, though not with equal rapidity in all directions. A square figure of any colour painted on a white background, when carried slowly from opposite the yellow spot to opposite the more peripheral parts of the retina, gradually becomes indistinct, and finally appears black. It at first appears smaller, and becomes at last ill-defined. The colour of the figure becomes indistinct sooner than its shape. A yellow, green, blue, or red square on a black background gradually becomes white.

The larger the coloured surface the further from the yellow spot can its colour be recognised. No exact relation exists, however, between the rapidity with which the power of recognising a colour decreases and the size of the coloured surface. The power of recognising colour, is limited by the time during which it is exposed to view,. and this varies for different colours. Any colour, when looked at for a certain time, gradually fades. We distinguish, as regards *anomalies in the perception of colours,* between those cases in which in the course of a gradually increasing amblyopia, the perception of tints, and finally that of pure colours, is lost; and those in which vision, as regards acuteness, is not at all or but slightly impaired. To the latter group the term *Daltonism,* or *colour blindness,* is applied. There may be complete loss of perception of colours, or only loss of perception of some colours. Complete colour blindness—*Achromatopsia*—in which condition only black and white can be recognised, is rare. Generally the defect is confined to a few colours, while varieties in the intensity of those colours which can be recognised are often more rapidly perceived than by a healthy eye. Red or yellow are distinguished with difficulty, or not at all, from gray, blue, and other colours. Frequently red and orange cannot be recognised, rarely green. Some eyes can only distinguish tints of yellow or blue; few can only distinguish yellow.

In the slightest degrees much light is required to recognise the colour for which the sensibility is diminished. Daltonism is congenital, and is observed in children from parents of blood relationship; it is frequently hereditary; it is incurable and continues for life.

Chromato-dysopia—means the mistaking of colours which in the solar spectrum are very distant from each other. More frequently colours which are related to each other, or shades of the same colour, particularly violet and lilac, are mistaken.

Coloured vision (*Chromopsia or Chrupsia*).—Persons suffering from this affection complain that all objects appear of one colour—yellow, green, or red. Coloured vision may be intermittent or change from one colour to another. It occurs in the course of cerebral diseases, after undue exertion of the eyes, and after operations for cataract. To persons who have taken a large dose of santonin gray objects appear greenish-yellow. Pure white santonin, if exposed to light, as in the media of the eye, turns yellow.

Treatment.—In some cases benefit has been derived from prescribing spectacles with tinted glasses of the colour complementary to the one under which objects appear to the patient. Cases of coloured vision have occurred in which, in a word, or in a series of figures, different letters or figures appeared of different colours.

Photopsia (*Spintherismus, Phosphenes, flashes of light*).—

Photopsia is observed in blind as well as in seeing persons. "Fiery wheels," "balls," "stars of yellow, green, and red colours," "flashes of lightning," "rain of fiery sparks," "a mass of fire floating in the eye," "shining clouds moving about in the eye," "an appearance as if the eye were lighted up with moonlight," and similar photopsiæ are observed during sudden movements of the eyes, after blows on the eyes, after undue exertion, especially in bright light; during inflammatory changes affecting the relations of the roots and bulbs to the adjoining parts, during extra-ocular changes causing hyperæmia of or pressure upon the optic nerve-fibres, after poisoning by various gases, by alcohol, &c.

To test the power of the retina for perception of light, we use artificial light. A record of the degree of this power becomes necessary if the patient has only perception of shadows, or can merely distinguish light from dark, as in advanced cataract or in amblyopia.

The patient being placed as for ophthalmoscopic examination, we throw the light of the ophthalmoscope upon the different parts of the sclerotic, iris, and pupil, and vary the quantity of reflected light by decreasing the intensity of the flame or by holding the instrument further from the eye.

The experience obtained from the comparison of different eyes, with or without cataract, but having healthy retinæ, examined in this way, teaches us the quantity of light which should be perceived by a healthy retina. If no light at all can be perceived, *e.g.* when thrown from above upon the upper half of the sclerotic, so that it should reach the lower half of the retina, while it is readily recognised when coming from other directions, we may rest satisfied that the lower half of the retina does not perceive light from having been displaced or destroyed.

Duration; persistence of retinal impressions.—Many of us must have observed that after looking for a short time at the red disc of the setting sun the disc often remains visible for a few minutes, though the eyelids be closed; the retinal impression has remained persistent beyond the usual time. One eighth of a second is the average time during which a retinal impression at the yellow spot persists before another one can take its place. The time of persistence can by practice (by looking at a clear line on a dark back ground for a certain time) be increased to half a minute. At the yellow spot a more persistent retinal impression fades away gradually in some, with intermission in others, while in the more peripheral parts of the retina it always disappears by intermissions, *i. e.* it disappears, then suddenly reappears somewhat fainter, and so on.

In some morbid changes of the visual apparatus the persistence of retinal impressions has been known to continue for days and weeks, and to return whenever the patient's thoughts have reverted to the objects of the impressions.

Binocular vision.—By binocular vision, *i. e.* by vision with both eyes at the same time, we obtain—

1. A larger field of vision. The inner (nasal) half of one retina perceives objects which are not seen simultaneously by the inner half of the fellow-retina.

2. A better idea of the bodily forms of objects—each eye viewing the same object from different sides—and of the rate

of motion, especially of those objects which directly recede from or approach the eyes.

A difference in the shape and with it a difference in the refraction of the two eyes.—If exceeding certain limits disturbs binocular vision, and that eye is used with which at the required distance vision is most acute; for superficial observation of objects, especially if the power of converging the eyes is well developed, binocular vision may exist even with great differences of refraction.

Protrusion of one eye, or differences of size between the two eyes.—The effect within certain limits of these differences of position and size is that an object placed at the same distance from both eyes appears smaller to one, *i. e.* to the less prominent eye, because the crossing point of the rays in the eye lies nearer the retina; or larger to the other, *i.e.* to the more prominent eye, through the crossing point being situated further from the retina. With both eyes open the object appears of a size which is the medium between that which it would appear to one eye and that which it would appear to the other. Binocular vision is lost as a consequence of all changes which impair the vision of one eye only, and also through strabismus.

The existence of binocular vision is established by alternately covering each eye during reading, when the uncovered eye should continue to read; or by placing a prism before one eye with the refracting angle upwards, when diplopia should appear with the double image, possessing the same distinctness as the true one. If binocular vision is absent we should never neglect examining the defective eye, and by practice we should increase the acuteness of vision.

Combined vision—though it be no more binocular—exists if the two eyes assist each other, *e. g.* in judging of the shape, distance, &c., of an object, as is the case if a cataract has been removed from one eye; though there be differences in the distinctness and sizes of the images formed in each retina, yet the field of vision remains undisturbed and the functions of both retinæ are maintained in greater perfection. In myopes operated upon for cataract it occasionally happens that the eye operated upon is used for viewing distant objects, and the other for reading.

Vision with one eye.—The sensation of muscular action experienced during accommodation, and during convergence (especially if the other eye is not destroyed), is the only means by which one eye can judge of the sizes, distances, and shapes of objects. Assistance is afforded in this by the movements of the head and body.

Anomalies of vision.—The anomalies of vision, independent of those arising from disturbances caused by the "appendages of the eyeball," the eyelids, lachrymal apparatus, and conjunctiva, are at present subdivided into those which are the result of anomalies—1, of the refracting surfaces; 2, of the transparent media; 3, of the parts concerned in accommodation; 4, of the retina; or 5, of the optic nerve or brain.

A short recapitulation of the numerous terms which were in use to designate the different kinds of impaired vision, previously to the adoption of this classification, is necessary, together with an indication as to where the explanation of such terms may be found. Many of these terms are still employed, others are used in different or in more or less restricted senses.

(*a*) *Lesions of the power of recognising the positions, distances, sizes, forms, and rates of motion of objects.*

The patient states—

1. "That with both eyes open he sees double" (this is diplopia).

2. "That with one eye alone he sees double, treble, &c." (this is polyopia).

3. "That objects seen with one or with both eyes appear too small" (this is termed mikropia). This is frequently observed during anomalies of accommodation or sudden changes in the position of the crystalline lens.

4. "That objects seen with one or with both eyes appear too large" (this condition is termed megalopia). The causes are similar to those just stated, but act in an opposite direction.

5. "That an object can only be recognised by rolling the eye about or holding it sideways." Corneal opacities and lesions of the yellow spot have been observed among other causes.

6. "That objects appear distorted, bent, &c." (= metamorphosia). Anomalies of the light-refracting parts and lesions of the retina or brain may be the causes.

7. "That only half or part of an object can be seen." (See Hemiopia and Scotoma.)

8. "That objects which are at rest appear in motion." (See Paralysis and Paresis.)

9. "That the distance of an object is judged wrongly, or cannot be determined at all."

The usual causes are—sudden loss of one eye; the use of spectacles for going about with by persons advanced in age who have not worn any before, and the use of spectacles by those who have been operated upon for cataract, paresis and paralysis of the muscles which come into play during accommodations.

10. "That he is obliged to look for some time before he can recognise an object" (=slow vision.) This symptom is connected with lesions of the retina, optic nerve, or brain.

11. "That objects can be seen standing before the eyes for some time after they are closed" (= persistent vision. See Duration of Retinal Impressions.)

12. "That the images of objects just perceived, vanish and return, the eyes being kept closed" (= intermittent vision. See Duration of Retinal Impressions.)

13. "That after looking, especially at small objects, for some time, they become misty, but that on closing the eyes for a moment, or rubbing them, the objects can again be seen distinctly for a short time" (= weak sight, impaired vision, &c. See Asthenopia and Circles of Diffusion.)

(*b*) *Lesions of the power of recognising colour.*

The patient states—

1. "That all objects appear of the same colour" (= coloured vision—chromopsia). This symptom is observed occasionally in persons who have been operated upon for cataract and in those suffering from changes of colour of the transparent media, as may occur, *e. g.*, during jaundice.

2. "That objects, the flame of a candle, &c., seem surrounded by rainbow colours." (See Glaucoma, Conjunctiva, and Cornea.)

3. "That colours cannot be perceived at all, or that only some can be recognised, or that colours are mistaken" (= colour blindness. See Perception of Colours.)

(*c*) *Lesions as regards the sensibility to light.*

The patient states—

1. "That the light dazzles the eyes." (See Photophobia, or intolerance of light, which is observed congenitally in albinos and in persons without irides.) The usual causes of this condition are corneal opacities and other superficial changes of the cornea. It is also observed during hyperæmia of the optic nerve, as in many cases of myopia and in commencing glaucoma.

2. "That much time is required before he can see when coming from a light into a dark room." (See Night Blindness.)

3. "That the ordinary amount of light is no more sufficient to see small objects." (See Presbyopia.) The functions of the retina and brain should be examined.

4. "That on closing the eyes, or in the dark, he sees flashes of light, fiery circles, falling stars, &c." (= photopsia, phosphenes. See Myopia and Morbid Changes of the Optic Nerve.)

The diagnosis and treatment of the anomalies of vision are most facilitated by ascertaining in succession the functions of the parts necessary for vision. These are:—

1. The light-refracting portions of the eye, comprising the cornea, the aqueous humour, the crystalline lens, the vitreous substance, together with the retina (the screen upon which an inverted image of an object is to be formed), as far as regards its distance from the nodal point, and with it the shape of the eyeball. Anomalies of the curvature of one or several of these structures give rise to impairment of vision through disturbing the refraction. To this group belong myopia, hypermetropia, and astigmatism, &c.

2. The media of the eye, *i. e.* the parts which in health are transparent when viewed with the unaided eye, excepting the retina and the optic disc. The kind of disturbance of vision caused through impaired transparency of the media is described under Opacities of the Cornea, Closed Pupil, Cataract, Opacities of the Vitreous Substance. The light, instead of reaching the retina in the usual way, is diffused over it, and objects appear misty, and the contrast between light and shade is diminished.

3. The parts concerned directly or indirectly in the accommodation of the eye. These are the crystalline lens, the ciliary muscle, the parts immediately adjoining these, and the

external muscles of the eye. The number of persons whose vision is disturbed through anomalies in these parts is very great. The anomalies of refraction often make themselves felt by disturbances of the accommodation. Most cases of what by some is termed painful vision belong to this group. Senile changes affecting the contractility of the iris and ciliary muscle, and the elasticity and consistence of the crystalline lens, are a fertile source of derangements of vision.

4. The retina, optic disc, and the tunics adjoining them. The impairment of vision through lesions of these parts, whether the lesion has originated in the part or the latter has become impaired secondarily, is termed *Amblyopia* (ἀμβλυς = obtuse; and ὢψ = eye) as long as there is perception of light, Amaurosis (ἀμαυρος = blind) if the vision is entirely destroyed. We may thus have amblyopia or amaurosis from choroidal or retinal changes (inflammation, displacement, &c.); from orbital changes (as tumours in the orbit, impairing the function of the optic nerve, &c.).

5. The brain and those other parts of the nervous system which are essential to vision. Previously to ascertaining the state of the parts just mentioned, we must not neglect examining the appendages of the eyeball, the eyelids, the conjunctiva, the lachrymal apparatus, since but slightly perceptible changes of these parts may give rise to changes of vision which much distress the patient; *e. g.* the mucus of slight catarrhal ophthalmia may give rise to muscæ, or to chromopsia or ptosis, and so to the idea that vision is lost, &c. &c.

The mode of ascertaining the refraction and its anomalies, the conditions of the media of the eye, the accommodation and its anomalie, the functions and the disturbances of the external muscles of the eyes, not belonging essentially to the functions of the retina, may be found in books on ophthalmic surgery; the functions of the retina and its morbid changes are treated of in the subsequent pages of this paper.

Anomalies of Vision—the result of extra-ocular changes, and especially of morbid changes of the brain, and of those other parts of the nervous system which are essential to vision.

The term amaurosis is employed if vision is completely lost (= "cerebral amaurosis").

The term amblyopia, if there is perception of light (= "cerebral amblyopia"). The degrees of amblyopia, as long as letters can be recognised, are expressed as shown on pp. 556-7.

Impairment or destruction of the functions of certain portions of the retina or of the optic nerve-fibres often give rise to anomalies of vision, which in themselves are characteristic of the morbid changes of which they are symptoms.

The following are some of the forms of amaurosis and amblyopia frequently observed:

Amblyopia or amaurosis of circumscribed portions of retina. —An amblyopic or amaurotic and circumscribed portion of the retina is termed a "*scotoma.*" It gives rise to interruption of the field of vision.

(1) *Scotoma in the region of the yellow spot (central scotoma —central interruption of the field of vision—impairment of direct vision).*

A scotoma in this region has been observed after looking into strong light or after working too much at the microscope. It may appear in both eyes if both are used in succession. In the eye thus used a yellowish mist, whose place, if the work be continued, becomes occupied by a deep brown one, is noticed. Objects placed opposite this region can no more be perceived. Hours may elapse before the mist has cleared away and vision has become normal again.

The boundary of the impaired portion of retina is ill-defined, while the function of the more peripheral parts may continue undisturbed. With the ophthalmoscope an increased grayish haze surrounding the reddish centre of the yellow spot is observed.

From the scotoma which may recur whenever cause is given we distinguish the amblyopia aud amaurosis which appear more gradually, without any apparent cause, often in both eyes, and remain permanently.

On ophthalmoscopic examination we may find choroido-retinitis, loss of transparency, followed by local pigmentation and atrophy.

In these cases more peripheral parts of the retina sometimes arrive at a degree of acuteness which is not possessed by equally eccentric parts of the healthy retina.

Another form of central scotoma is the one caused by cere- ·tra-ocular changes in which the ophthalmoscopic examination is often negative, and the boundaries of the impaired portions of retina are very irregular. The arrest of the function of a certain number of optic nerve-fibres, *e.g.* from cerebral changes, gives rise to impairment of the function of the retina to which those fibres belong, and to a scotoma which lies opposite the impaired part of the retina, while the rest of the retina remains normal. But if a portion of the retina and of the entire adjoining layer of optic nerve-fibres is destroyed through intra-ocular changes, then the function of the more eccentric part of the retina is abolished, as far as the nerve-fibres are concerned which have to pass from other parts through the destroyed portion.

The following mode of ascertaining the extent of small central scotomata, the fellow-eye being healthy, has been proposed.

The patient is directed to look with both eyes at a white spot on a black dull paper. To healthy eyes the white spot, viewed through a blue-tinted glass held before one, and through a yellow-tinted glass held before the fellow-eye, appears of a dirty green colour; while if the region of the yellow spot of *e.g.* the left eye, is impaired, the healthy right eye looking through the yellow- and the left through the blue-tinted glass, at a piece of white chalk held at the side of the white spot, the spot and piece of chalk appear yellow. Now, on moving the piece of chalk along the black paper, away from the white spot (both eyes fixed on the latter), the chalk appears of a dirty green colour the moment a point is reached where both retinæ participate in the act of vision. The shape of the impaired portion of retina can be found by ascertaining a certain number of these points.

(2) *An amblyopic or amaurotic portion, i.e. a scotoma, adjoining the optic disc, and most frequently the portion next the yellow spot. "Enlargement of the blind spot."*

Small blind or impaired portions of the retina adjoining the optic disc are generally not noticed by the patient. Large ones, if encroaching upon the region of the yellow spot, betray themselves in consequence of portions of an object situated outwards from the point of the object directly looked at appearing indistinct or missing. The impaired portion is rarely irregular, generally transversely oval. Atrophic changes of the choroido-retinal aperture following inflammation or simple distension, as in extreme myopia, are the usual cause.

These changes near the optic disc are the more troublesome the nearer they are to the yellow spot, and those to the right of the spot are more troublesome than those to the left.

If recent and appearing rapidly they for a time impair ("dazzle") vision of the fellow-eye.

To make them more strikingly perceptible, we may cause the patient to look with the affected eye through a small opening at a strongly illuminated white surface, upon which the impaired portions of retina appear as dark spots.

(3) *Amblyopia or amaurosis of other parts of the retina; excentric scotomata; excentric interruptions of the field of vision.*

If eccentric portions are not destroyed, but only more or less impaired, they appear as differently shaped gray, black, or coloured spots, either entirely obscuring portions of an object, or causing them to appear indistinct, misty, or distorted, or smaller or larger. Small impaired spots are often overlooked, especially if confined to one eye, and are best perceived if the other eye is closed. They are generally the result of choroido-retinitis.

4. *Amblyopia or amaurosis progressing from the periphery (margin) of the retina towards the yellow spot; contraction or limitation of the field of vision.*

The impairment of vision—

1. May be confined to one half of the retina ("hemiopic limitation").

2. It may advance to an equal extent from all points of the periphery of the retina towards the yellow spot ("concentric limitation"), as frequently occurs in both eyes of the same patient, a zone of amblyopic retina joining the amaurotic one.

3. It may advance more rapidly from one side ("irregular limitation").

Of this latter, different varieties occur, *e.g.* it may progress more rapidly from the outer margin of the retina, as in cupping of the optic disc; or more rapidly from above and below, as in anæmia and atrophy of the optic disc; or from extra-ocular causes; or irregularly from all sides, as in the different forms of choroido-retinitis.

The "amaurotic" portions of the retina may join healthy portions, but more frequently they are contiguous with "amblyopic" ones.

Cerebral changes affecting vision lead simultaneously to eccentric limitation and to impairment of direct vision; not so morbid changes in the retina.

The prognosis is better if, from impeded innervation or from non-use, direct vision is much impaired while the eccentric parts of the retina remain sensitive throughout, than if direct vision is good, but the eccentric parts are much or irregularly impaired.

For the mode of keeping a record of the field of vision of a retina thus impaired, see p. 559.

5. *Hemiopia; half-vision through paralysis of half of one or of each retina.*—The right halves of the retinæ of the right and the left eyes (the outer or temporal half of the right and the inner or nasal half of the left retina) are supplied by the right optic nerve-tract, and the left halves (the outer half of the left and the inner half of the right retina) by the left optic nerve-tract. The right and left optic nerve-tracts meet at the chiasma. The fibres of the outer half of each tract go, those of the right tract to the optic nerve of the right eye and thence to the outer half of the retina of the right eye, those of the left tract to the optic nerve of the left eye and thence to the outer half of the retina of the left eye; while the fibres of the inner half of each tract cross each other at the chiasma, those of the right tract crossing over to become the inner half of the left optic nerve and to go to the inner half of the left retina, those of the left to become the inner half of the right optic nerve and to go to the inner half of the right retina.

Non-crossing of the tracts of the optic nerves, or absence of the chiasma, with and without impairment of vision, have been observed by several anatomists.

There occur in the chiasma also fibres which go from eye to eye, and others which pass from one side of the brain to the other.

The varieties of hemiopia are, in order of frequency—

1. Loss of vision of the outer half of the right and of the inner half of the left retina, from paralysis of the right optic

nerve-tract; and loss of vision of the outer half of the left and of the inner half of the right retina, from paralysis of the left optic nerve-tract. The line of demarcation between the sensitive and blind part of the retina is vertical and sharply defined. On examination with the opthalmoscope we may find both optic discs healthy or hyperæmic, but never any morbid change confined to one half of the disc only. These two forms are most frequent, and are observed in the course of tumours, of apoplexy with hemiplegia, of diabetes, &c. The morbid changes which cause the hemiopia are situated on one side of the brain. We infer, if loss of vision of the entire retina follows, that these changes are no longer confined to one side of the brain. Hemiopia after apoplexy remains stationary in many cases, and blindness need not be feared, provided apoplexy does not appear elsewhere.

Tumours and periostitis, at or near the chiasma, may give rise to any of the above or subsequently to be mentioned forms of hemiopia. The hemiopia caused by changes about the chiasma is often followed by loss of vision, and not infrequently by recovery of the functions of one optic nerve, the hemiopia remaining stationary for years.

2. Loss of vision of the outer half of each retina (of the right half of the right, of the left half of the left eye). This form, as regards vision, is the least troublesome.

3. A very rare form is paralysis of the inner half of each retina. It has been observed in the course of intracranial tumours.

6. *Night blindness* (*Hemeralopia*) is an anomaly characterised by a sudden impairment, amounting in rare cases to loss of vision, as soon as the quantity of light decreases beyond a certain point, while for the function of the healthy eye that amount of light is still sufficient. Persons suffering from this affection, in full daylight often enjoy normal, or nearly normal, vision. Night blindness varies in degree, and frequently in the two eyes of the same person.

The dilated and sluggish pupil, and the want of contractility of the ciliary muscle, with insufficiency of the internal recti muscles, observed in many cases, seems not to be owing to paresis of the third nerve, since the application of tincture of opium to the conjunctiva causes active contraction of these parts. In rare instances only the region of the yellow spot is affected, a dark brown cloud obscuring the object, which, when held opposite lateral parts of the retina, is distinctly recognised. Red, blue, or violet light is not perceived so readily as green, yellow, or white light. If this affection appears suddenly, following exposure of the retina to bright light, it may continue for months, *e.g.* until winter, and return again in spring.

In fresh cases, or after a good night's rest, it does not appear in the forenoon, though the day may be dull; while after mid-day, with the same degree of light it becomes felt. The result of an ophthalmoscopic examination is negative in many cases. Choroido-retinitis and its effects, or hyperæmia of the optic disc and retina, may be found.

As causes are mentioned—1. Various forms of choroido-retinitis and intra- and extra-ocular morbid changes, affecting first the periphery of the retina. In these cases the night blindness is a forerunner of amblyopia or amaurosis.

2. Scurvy, of which it may be the only symptom; bad food, ague.

3. Exposure, especially if frequent, of the retina to direct or reflected sun- or moon-light. It has appeared as an epidemic among sailors and soldiers, though not all exposed to the same causes may have suffered from it. It is more frequently observed in spring.

Treatment (see treatment of the various forms of choroido-retinitis, pp. 590, 591).—If occurring during or after ague, one-grain doses of camphor, three times daily, have removed it.

Complete exclusion from light (if following exposure to this agent), for from one to five days, and good nourishment, though not preventing a relapse, do cure it for a time.

7. *Snow blindness* is observed at the beginning of winter in many persons, and in animals exposed to the white reflection from the snow. It may reduce vision to perception of light. It soon subsides spontaneously, and has been known to act beneficially in cases of anæmia of the retina.

8. *Amaurosis or amblyopia, from overuse of the eyes*, through fine work, microscope or telescope work, or *from sudden exposure to strong light* (*lightning*). The region of the yellow spot is the part mostly impaired.

With the ophthalmoscope we may find choroido-retinitis. Exposure to strong light has even given rise to ophthalmitis of the exposed eye. In other cases no changes are discoverable within the eye with the ophthalmoscope.

Anomalies of vision from extra-ocular, and especially from cerebral, causes, often present no marked objective symptoms beyond the impairment of vision. The eye whose vision is disturbed may appear quite healthy, or there may be but slight alteration of colour of the optic disc. For the prognosis and treatment of such cases it becomes necessary to examine the other functions of the body, so as to obtain a clue to the seat, &c., of the lesion which has implicated the optic nerve-fibres.

The following are the symptoms which most frequently precede or accompany cerebral lesions which give rise to impairment or loss of vision.

Headache.—In itself is no guide as to the cause and seat of the lesion which may have given rise to impaired vision. It may precede the latter for years, and is attributed to morbid changes in the dura mater.

It is a symptom which frequently appears during or previous to impairment of vision, as the result of disturbed menstruation, and also during congestion of the cerebellum.

Vomiting.—We should ascertain what kind of vomiting there has been, whether of bile (the age of the patient attacked by this kind of vomiting, as a rule, varies between eighteen and thirty), or of blood (the age of the patient varies between forty and fifty) whether there has been obvious cause for it; whether it has appeared after meals, or at any other time. It may be a reflex action, which ceases as the ocular changes progress, or it may be connected with morbid changes about the medulla oblongata or at the roots of the eighth nerve.

Vomiting and headache with amblyopia or amaurosis, as a rule, are signs of cerebral disease.

Giddiness is often complained of for a long period before any lesions of vision occur, especially by persons suffering from heart disease, with intermittent pulse, from rigid arteries, and from derangement of circulation by cerebral changes.

Convulsions, "epileptic fits" of otherwise healthy or of paralysed parts.—Neither their character nor their frequency is a guide as regards the nature of the lesion which may have given rise to defective vision. If confined to one side, we may suspect the cause of impaired vision to lie near or at the corpus striatum. The fit, sometimes, is preceded by complete blindness, the latter being attributed to temporary anæmia of the retina, from spasm of the coats of its blood-vessels.

Hemiplegia is a frequent forerunner, or companion, of disturbances of vision. If the two appear simultaneously, they are attributable to apoplexy near or in the corpus striatum or thalamus opticus. If the hemiplegia precedes the amaurosis for a long time, then we may attribute the latter to secondary cerebral lesions, and to fresh apoplexy if it occurs suddenly.

In amaurosis, with hemiplegia of the right side, and with loss of speech, both hemispheres have repeatedly been found diseased.

Amaurosis, when occurring on the same side as the hemiplegia, is attributed to embolism. Amaurosis from inflammation of the optic discs of both eyes, without anomalies of locomotion, is generally complicated with or caused by lesions of the cerebellum or of the hemispheres.

Ataxy of locomotion.—Of patients suffering from this anomaly, fourteen have come under observation within the last four years. They were middle-aged persons, and complained at first of great general weakness, including that of the genital organs; of a sensation of "pins and needles" in the lower extremities, preceding numbness, accompanied by a painful feeling of constriction of the chest. Then followed cramps in the feet and trembling of the hands, especially when attempting to seize any object. Two months after the first symptoms of ataxy, vision became impaired, a mist "appearing before the eyes," and getting gradually thicker, rapidly reducing vision, in some, to perception of light, in others destroying it completely.

The blood-supply to the retina and choroid remained normal, but the optic disc rapidly became anæmic, and remained so even in the cases where vision rose from one eighteenth to one fifth.

Causes of amblyopia and of amaurosis.—The cause may be—

I. Ocular (retinal, choroidal, or in the optic nerve).

II. Extra-ocular (orbital).

III. Cerebral or spinal.

In cerebral changes accompanied by great disturbance of the circulation within the head, as observed during rapidly growing tumours, and during meningitis, hyperæmia of both the retinæ, rupture of the blood-vessels, swelling of the optic discs, are among the frequently observed ocular affections. In chronic cerebral changes, anæmia and atrophy of the optic nerves are the usual changes observed with the ophthalmoscope.

The changes may be far away from the optic nerve-fibres, and yet, through pressure, impair their functions. There may be extensive disease of the hemispheres with perfect vision. Morbid changes of the right hemisphere are more often complicated by amaurosis.

Changes confined to one side of the brain may cause hemiopia, but never complete amaurosis, unless the latter appears secondarily.

Amaurosis or amblyopia—(a) from various kinds of intracranial and cerebral tumours.—The usual seat of such tumours is the basis cranii, especially the sella turcica and the neighbourhood of the cerebellum.

They produce loss of vision, either mechanically, by pressure, impeding the functions of the optic nerves or the circulation, or by becoming complicated with inflammation of the optic nerves, with meningitis, &c. The latter complication frequently prevents our drawing conclusions from the nature of the impairment of vision as to the seat of the tumour. The most frequent changes observed with the ophthalmoscope in the course of cerebral tumours are—hyperæmia or œdema, or inflammation of the optic disc and retina, and anæmia, or anæmia with atrophy of the optic disc. Slight protrusion of the eyeball, with fulness of the veins about the eyelids, is an occasional complication of tumour at the sella turcica or at other parts of the basis cranii.

(*b*) *From morbid changes at the base of the brain.*—The most frequent is meningitis (tubercular, pyæmic, typhoid). In some cases the impairment of vision appears at the time of the meningitis; in many, however, long after it has passed. Both optic nerves, or one or only part of an optic nerve, may be affected. Amaurosis of both eyes in these cases is generally complicated with paralysis of other cerebral nerves.

(*c*) *Following apoplexy, softening, tuberculosis, abscess of the brain.*—The lesion of vision is often complicated with paralysis of separate spinal nerves or of other cerebral nerves with hemi- or paraplegia. Apoplexy is a frequent cause. It may appear simultaneously in the eye and brain. (See Retina.)

(*d*) *Amaurosis* through constriction of both optic nerves, causing indentation (partial) or total destruction through the two arteriæ communicantes posticæ.

(*e*) *Following injuries,* especially blows or falls, causing concussion of the brain; pain in the head, fits, peculiar anomalies of locomotion, frequently accompaning the impairment of vision. All hope of recovery of vision is lost if once atrophy of the optic disc and retina has set in.

(*f*) *Hereditary amaurosis or amblyopia,* from incomplete development of the brain or eye, microphthalmus, hydrophthalmus (coloboma), or from morbid changes appearing in the brain (hydrocephalus) or eyes during growth or in advanced life.—One or several members of the same family may suffer from this affection for several generations. In hydrocephalus the tracts of the optic nerves which run below the lower surface of the ventricles are unduly stretched during the distension of the latter by fluid, and the thalami optici and crura cerebri are pressed asunder. The floor of the third ventricle, the tuber cinereum, the chiasma, the sella turcica, and the adjoining part of the sphenoid bone, suffer likewise from the distension. Atrophic changes following choroiditis, and anæmia and atrophy of the optic disc and retina, are the changes most frequently observed simultaneously.

Simulation of amaurosis or of amblyopia—(1) *of both eyes,* may occur in insane persons, or with a view of obtaining a certificate of blindness. We may suspect simulation if both the pupils of the patient are active, and if he states that he cannot perceive light. If he says that he can see light, but cannot recognise objects, we may be unable to express an opinion.

(2) *Of one eye, generally of the right.*—If the pupil of the supposed blind eye (provided it be not under the influence of a mydriatic, and the fellow-eye be thoroughly excluded from light) does not contract when suddenly exposed to strong light, but does do so when both eyes are open, we can pronounce the first eye to be blind. We should, if any doubt remains, after having carefully examined the "supposed" blind eye, place a strong prism, *e. g.* one of 12°, with the refracting angle downwards, before the patient's good eye, causing him to look at the flame of a candle, when the simulation at once becomes apparent if the patient states that he sees two flames (attributing them to the action of the prism on the sound eye).

Amaurosis or amblyopia, with disease of the spinal cord.—Tenderness on pressure in the region of the first cervical vertebra, with impairment of vision, has been observed to precede the amaurosis.

The ophthalmoscopic symptoms in advanced cases are anæmia and atrophy of the optic discs and retina. The atrophy of the optic discs is in some cases the first of a series of symptoms arising from spinal changes.

Much benefit has been derived in some cases by repeated blisters applied to the cervical region.

The post-mortem examination of some cases has shown atrophy of the trunks of the optic nerves, and morbid changes *e. g.* atrophy, in the thalami optici.

As causes, have been observed injuries, inflammation, atrophy.

Reflex amaurosis, from "irritation," originating in one of the sensitive nerves, or from "irritation" of other parts of the nervous system.—Cases of this group occur not infrequently without any apparent changes in the eye (the optic disc, among other parts, appearing healthy) or in the functions of the cerebrum or cerebellum.

The amblyopia or amaurosis is attributed to alterations in the tubercula quadrigemina, which are supposed to be the result of "irritation" elsewhere, *e. g.* of injuries to the spinal cord, of irritation of the frontal nerve, of neuralgia of the face, of gastralgia, of irritation caused by worms.

The appearance of the disturbance of vision after such irritation, its increase or decrease keeping step with the irritation, and its removal or relief on cessation of the supposed cause, justify the assumption of a reflex amaurosis.

IV. General or constitutional causes.

Amaurosis or amblyopia appears—

(*a*) *Among other symptoms of albuminuria* (see p. 593).

(*b*) *During syphilis* (see p. 589).

(*c*) *During diabetes mellitus.*

(*d*) *Through embolism* (see p. 598).

The morbid changes comprised under *a, b, c,* and *d,* when localizing themselves in the eyeball itself, assume certain characteristic forms, *e. g.* that of effusion of lymph in syphilis, that of peculiar infiltrations with rupture of blood-vessels in albuminuria, &c. &c. If on the appearance of amaurosis or amblyopia we find the eyes intact, and after examination of other organs discover albuminuria, syphilis, &c., we adopt the same general treatment which would have been carried out if the eyes had been attacked.

(*e*) *During irregularities of menstruation, during pregnancy, parturition, or lactation.*

Sudden amaurosis, appearing about the time when menstruation was expected, has been observed in two cases to subside again after the reappearance of that function. Vision returned gradually in both cases, and has remained normal.

Cases of recurrent amblyopia or amaurosis, commencing at some period of pregnancy and subsiding after parturition, have repeatedly been recorded.

A frequent cause of amblyopia or amaurosis after sudden arrest of menstruation is the occurrence at that period of intra-ocular or of cerebral hæmorrhage, which has been preceded in several cases by severe pain in the head, with unconsciousness.

The morbid changes which give rise to the lesion of vision during the above changes in the body in many respects resemble those observed during albuminuria, and may be divided into two groups—into those whose chief symptom is hæmorrhage, with œdema of the parts adjoining the seat of hæmorrhage; and those which appear with infiltration, inflammation, and the subsequent changes of texture in the eye (generally at and near the optic disc) or in some other part of the body.

(*f*) *Through general anæmia* (*ischæmia*).

1. From general causes, diabetes, diarrhœa, spermatorrhœa, loss of blood, *e.g.* by vomiting. Vision in these cases is lost, in some gradually, in others suddenly. Much good may be done, if the impairment is not considerable, by improving the general health; while if the ophthalmoscopic signs of atrophy of the optic nerve and retina have appeared (and the sooner the worse) little hope remains, though the general health may have been restored.

Vomiting of blood in several patients from forty to fifty years of age (of whom a few suffered from ulceration with sloughing of the mucous membrane of the stomach) has been accompanied or followed by sudden loss of vision of both eyes, or of one, the other eye becoming impaired from one to six months later. Vision in such cases, if lost suddenly, often remains lost, though the general quantity of blood may have been restored. The anæmia and atrophy of the retina and of the optic disc only appear at a later period. There may have been amaurosis for some time, while with the ophthalmoscope we find slight anæmia, or no changes at all. The quantity of blood lost, or anæmia of the brain, does not explain the loss of vision, since other cerebral functions return as the quantity of blood increases.

The amaurosis sometimes appears while the patient is recovering from the loss. In some cases the loss of blood was so slight that the amaurosis could not be attributed to it.

2. Through anæmia, from local causes. Gradual compression of the artery of the retina is followed, at first, by impairment of the functions of the retina in the region of the yellow spot; sudden obstruction, by sudden amaurosis. The less arterial blood enters the retina, the more vision is impaired.

(*g*) *Produced by tobacco, lead, quinine, or alcohol.*

These and other substances often produce spasm, or paresis, or paralysis of the accommodation as well. We should guard ourselves against mistaking the disturbances of vision due to these latter conditions for amblyopia or amaurosis.

By tobacco.—The patients generally are of middle age, thin, pale; rarely complain of pain, and often state that the impairment of vision progressed slowly. Vision may, however, within six months be reduced to perception of light. In the majority, if the disease is progressing, it reaches this degree in from six to eighteen months. It always affects both eyes, though it varies in degree; one eye may become blind without the patient being aware of it. Night blindness and decrease of the acuteness of vision for distance are often its earliest objective symptoms. Photopsia frequently appears, and sometimes after all vision is lost. With the ophthalmoscope we observe anæmia, and finally atrophy, of the optic disc and retina. The greater the diminution of the number of arteries in the retina, the greater the impairment of vision.

By lead.—Impairment of vision has followed medicinal application of lead in two cases under my observation, and working in the metal in several cases. In the former cases protracted headache was followed by sudden loss of vision in the right, and twenty-four hours later in the left eye, with paralysis of the ciliary muscle and iris, and with some increase of temperature. In a similar case vision returned after the use of mercury (to salivation), followed by that of iodide of potassium. In one of the latter cases, on post-mortem examination, except extreme anæmia, no morbid changes were discovered.

By quinine.—Deafness or noises in the ears are usual complications. With the ophthalmoscope we find a tortuous condition, or at least an overfulness, of the veins of the retina in the otherwise healthy eye. Much benefit has been derived from repeated bleeding.

By alcohol.—In a case in which vision was reduced to perception of light, during prolonged abuse of alcohol, the optic disc appeared healthy; vision, after discontinuing drinking and applying leeches to the temples, rapidly returned, so as to allow the patient to follow his employment.

Amblyopia through non-use, after prolonged exclusion, of one eye.—This form of amblyopia reaches a high degree only if it be congenital or of very long standing. It may remain undiscovered for years.

The functions of the retina of one eye, interfering with those of the fellow-eye, may finally lead to "suppression" of direct vision. The development of higher degrees of amblyopia sometimes seems to be prevented through the pupil becoming closed or cataract developed.

Paralysis or paresis of the iris, of the accommodation, opacities of the cornea, and especially strabismus of one eye, are usual complications. Signs that the functions of the retina are nowhere interrupted are the integrity of the field of vision, and the proportionate decrease of sensibility of the retina from the periphery towards the centre.

Development of the Retina.

Among the peculiarities of the fœtal retina must be noticed—

1. The presence of numerous folds.

2. The fœtal fissure, extending from near the lower margin of the crystalline lens to the nearest point of the optic disc.

3. Peculiarities at the retinal aperture and at the ora serrata.

1. The retina is thrown into folds, which are found to be most numerous about the end of the first month; they project into what is to be the vitreous chamber, the larger ones occupying the region of the yellow spot. No optic nerve-fibres are found upon the folded parts of the retina. The folds disappear as the eye increases in size, and the retina gradually flattens itself out upon the choroid. They disappear first along the ora serrata, with which they are somewhat parallel. About the ninth month some folds may still be found near the lower margin of the optic disc.

2. About the beginning of the third month the margins of the fœtal fissure are inverted towards the vitreous chamber; the fissure is broadest near the optic disc, and is closed about the commencement of the fourth month. A narrow seam extending from the optic disc to the ciliary process, and slightly projecting into the vitreous chamber, indicates the direction occupied by the fissure.

3. The retinal aperture (round the optic disc) is very small about the end of the third month. It is not circular, but irregular, some parts of the retina projecting more towards the optic disc than others. These prominences disappear about the fifth month, when the aperture becomes more circular.

About the end of the third month pointed processes appear upon the folded margin of the retina which lies nearest the crystalline lens. These grow forward towards the margin of the lens and form part of its suspensory ligament, while the vitreous substance increases in bulk between the margin of the lens and that of the retina.

The bulk of the retina about the second month consists microscopically of nucleated cells. About the third month the fibres of its connective tissue, and about the fourth month the optic nerve-fibres, can be recognised.

Anomalies of development.—Absence of all the retinal blood-vessels, with a white atrophic optic disc, or incomplete development as regards number, have occurred repeatedly, there being amblyopia in the latter and amaurosis in the former case.

Very thin retinal vessels with an anæmic optic disc, frequently altered in shape, are often observed in children suffering from inherited syphilis.

An opaque white colour of the connective tissue of the optic disc, extending as a pearly white and opaque band over a limited portion of retina, has repeatedly been observed.

A case has been recorded of abnormal position of the optic disc, it being too far distant from the posterior pole of the eyeball.

For other anomalies, see Coloboma of the Choroid, and also Hypermetropia and Myopia.

Incongruence of the retina.—Of this anomaly two varieties have been described. In the one an excentric part of the retina, *i. e.* one outwards or inwards from the usual position of the yellow spot, is used when looking at an object directly, the acuteness of vision of that excentric part being greater than that possessed by the region of the yellow spot of the same eye. Vision of the fellow-eye and the relative positions of the different parts of its retina are normal. In the other variety the yellow spot is stated to be situated on the inner or nasal side of the optic disc.

Hyperæmia of the Retina.

The examination of numerous healthy eyes, the comparison of the two eyes (if this condition is confined to one eye), and the general appearance of the patient, are in slight cases the guides as to the existence of hyperæmia. An increase in the number both of arteries and veins—"arterial hyperæmia"—with an unusual brilliancy of the retina and hyperæmia of the optic disc, are the symptoms observed in hypermetropic and myopic patients suffering, the former from asthenopia, the latter from signs of irritation, in severe inflammation of other parts of the eye, and in some cases from signs of protrusion of the eye from extra-ocular causes.

A different and frequent form of hyperæmia—venous hyperæmia—is the one observed in persons suffering from syphilis, albuminuria, or preceding inflammation of the optic disc, or whenever the return of blood from the retina is impeded through pressure upon the optic disc or upon the optic nerve inside or outside the eye.

The arteries appear generally fewer in number, while the veins are large, tortuous, and numerous. The changes are most conspicuous in and near the optic disc. Venous hyperæmia also precedes and accompanies inflammation of portions of retina which are remote from the optic disc.

Retinitis (Inflammation of the Retina).

Retinitis, as a rule, appears as one of the symptoms of general morbid changes, as albuminuria, secondary syphilis, cerebral tumours, &c. It appears very frequently in both eyes simultaneously, though it may vary in degree; its most frequent seat is the part of the retina adjoining the optic disc and the portion occupying the region of the yellow spot. Peculiarities as regards loss of transparency, shape or situation of the inflamed portion, vascularity, pigmentation, colour, &c., have given rise to the distinction of various forms of retinitis, which, if fully developed, are sufficiently characteristic to allow the general morbid change of which the retinitis is a symptom to be recognised.

Various forms of retinitis.—To facilitate reference we will number the different forms.

No. 1. Retinitis, with uniform loss of transparency, extending from the optic disc over a varying area of the retina, the latter appearing hazy, gray or gray-white, and opaque. A similar change is often found simultaneously in the region of the yellow spot; the arteries are thin, fewer in number, while the veins are tortuous, gorged, and pale red in the leucæmic, dark red in the form complicating cerebral tumours.

No. 2. Retinitis apoplectica.—In the turbid retina are visible, especially near or at the optic disc, an unusually large number of blood-spots, many of which lie next to gorged tortuous blood-vessels (see p. 602).

No. 3. Retinitis, with yellow or buff or rust coloured spots, patches, or nodules, appearing by preference in the region of the yellow spot and round the optic disc. There is generally a marked decrease of the blood supply to the retina (see p. 579).

No. 4. Retinitis pigmentosa (see p. 600).

No. 5. Retinitis, with one or several large gray and opaque patches, occurring more frequently at some distance from the optic disc or near the ora serrata, with small blood-spots and with an increased number of arteries and gorged veins, which are particularly conspicuous in the optic disc, the retina adjoining the inflamed portion being œdematous. The vessels in the further course of the disease gradually resume their natural calibre, though an overfulness of the veins often continues long after the retina has resumed its transparency. The blood-spots and the opaque patches disappear, and the choroid becomes visible as the retina clears. The latter often becomes atrophic, though it may resume its transparency; slight anæmia of the optic disc often remains.

No. 6. Suppuration of the retina.—The retina rapidly (in from twelve to thirty-six hours) becomes yellowish, white, opaque, and swollen, especially round the optic disc and in the region of the yellow spot, where its thickness may be three or four times that of the retina in health. This swelling and the loss of transparency are caused by the presence of pus-cells, exudation-corpuscles, &c. The pus-cells and exudation-corpuscles are supposed to originate in the nuclei of the fibres of the framework.

The outer surface of the yellow and opaque retina in many instances has a uniform red colour, from the extreme capillary vascularity developed in it.

Hypertrophy and excrescences of the connective tissue and of the membrana limitans appear in the further course of the affection, and assist in the destruction of the nerve-elements, ganglion-cells, granules, &c.; the latter become paler and finally disappear. The retina, in from six weeks to three months, is reduced to a semi-opaque or opaque membrane, which, thrown into folds, extends from the optic disc to the ora serrata, or (as occurs very rarely after suppuration) it remains in apposition with the choroid; in any case it becomes atrophic.

Causes and general remarks.—1. Retinitis No. 5 has been observed after sudden changes of light, as sunlight reflected on to, or directly and suddenly striking, the retina; after undue exertion of the retina, especially if persevered in and preceded by hyperæmia. The retinitis in these cases is often confined to the injured eye, and more often to the region of the yellow spot.

2. Retinitis No. 5 and No. 6 occur after injuries and operations, appearing in these cases as one of the changes described under Ophthalmitis, and generally implicating the entire retina of the injured eye.

3. Retinitis No. 5 and No. 6 may often appear after general illness, scarlatina, measles, smallpox, &c., and during pregnancy, suckling, &c.

4. Embolism, albuminuria, diabetes, diseases of the heart and arteries, frequently give rise to retinitis Nos. 1, 2, or 3.

5. Syphilis gives rise to retinitis No. 1 or No. 3; as a rule, appearing in both eyes.

6. Among intra-ocular causes, we meet with retinitis following choroiditis, or accompanying inflammation of the optic disc, or caused by cyclitis or by rupture of blood-vessels and hæmorrhage ("retinitis apoplectica"), or by tumours or entozoa. These causes generally lead to retinitis Nos. 5 or 6.

Retinitis, with much loss of transparency, when of long duration (two or three months), is often followed by atrophy. It may remain stationary for a time or improve, according to the habits, occupation, &c., of the patient; or it may progress rapidly to a certain point and then remain chronic. The longer the duration, the less favorable is the prognosis. It is frequently accompanied by choroiditis.

Symptoms, common to all or several of the above forms of retinitis:

1. Loss of transparency, varying from slight haziness to a gray and opaque colour.

In the opaque portion we often find "blood-spots" or yellow and opaque spots (as in albuminuria), or rusty coloured or yellowish-white nodules and spots (as in syphilis).

The lesions of transparency are most conspicuous in the region of the yellow spot and round the optic disc, and, together with anomalies in the circulation, are characteristic of retinitis.

2. Alterations in the appearance of the blood-vessels. In most forms we find an apparent decrease in the number and size of the arteries, and a gorged, tortuous condition of the veins. Portions of these latter are often entirely or more or less hidden in the opaque retina; the more so, the severer the retinitis.

Groups of enlarged capillaries, appearing to the naked eye as blood-spots, are particularly frequent in the retinitis Nos. 5 and 6, covering the outer (choroidal) surface of the retina. Blood-spots, the results of rupture of blood-vessels, occur in most forms, but more particularly in Nos. 1 and 2.

3. Photophobia, photopsia, chromopsia, with headache, pain in the eye, and with lachrymation, may appear in the beginning or in the further course of the disease, or be entirely missing; or only one, or several, of these symptoms may be present.

4. The exterior of the eye (the conjunctiva, cornea, eyelids, &c.) present nothing characteristic except in retinitis No. 6, in which form the changes observed in ophthalmitis (as chemosis, swelling of the eyelids, protrusion of the eye from œdema or inflammation of the parts within the orbit) often appear.

5. Vision.—No impairment may be observed by the patient if the inflammation occupies only peripheral parts of the retina, or if it is confined to one eye only, or if the occupation of the patient does not require acute sight.

We often find entire loss of transparency of the retina with very slight impairment of vision, probably because the changes are in that stage confined chiefly to the connective tissue of the retina.

In slight degrees, or at first, when the disease occupies the region of the yellow spot or of the optic disc, objects appear to tremble, or glitter, or seem distorted or surrounded by a gray-white, brownish, yellowish, or red mist. More light is required for vision, and objects have to be held closer.

In higher degrees, interruption or limitation of the field of vision is observed. Vision may become reduced to bare perception of light rather suddenly, and then decrease or improve gradually.

The impaired portions of retina are generally not well defined, and on moving the eye retain the same position in relation to the visual line, and to each other in the field of vision. They are by the patient described as black or gray patches when he is looking at a sheet of white paper held close to the eyes.

The prognosis, as regards recovery of vision, is less favorable if there is interruption or limitation of the sensibility of the retina than if the entire retina is more uniformly impaired. The longer the duration of the retinitis, the worse the prognosis. Dark spots in the field may clear up in some parts more than in others, or may disappear entirely.

For the treatment of the forms of retinitis No. 1, No. 2, No. 3, and No. 4, see articles on the subject.

Treatment of form No. 5.—Besides what has been stated respecting the treatment of the similar form of choroiditis in books, we must insist upon the patient keeping both eyes absolutely at rest until all hyperæmia has disappeared from the retina and optic disc. This is effected—1st. By keeping the lids of both eyes closed during the day if there is intolerance of light; if not, by simply prohibiting the use of the eyes for near work.

2nd. By ordering atropia to be applied twice daily, and the wearing of tinted spectacles if the atropia should give rise to intolerance.

3rd.—By avoiding all that might derange the circulation in the retina, as travelling in a carriage, stooping, and occupation causing excitement.

4th.—By bathing the closed eyelids with cold or warm water, according to the liking of the patient, and as often and long as it is pleasant.

One or two leeches to the temple on the side of the affected eye applied at bed-time relieve pain if it is accompanied by much hyperæmia of the inflamed part.

The retina and optic disc should be examined with the ophthalmoscope twice weekly. A glance will suffice to ascertain their condition. When once the retina has became transparent and the choroid again visible, but little further improvement of vision can be expected.

The general hygienic and medical treatment must be directed according to the cause of the retinitis and the general health of the patient.

Mercury has been found of use in retinitis occurring during pregnancy; the tonic treatment, when it has appeared during or after smallpox, measles, and similar weakening diseases. This form in weak persons often goes on into suppuration.

Atrophy of the Retina.

The cause of the atrophy in a great measure determines the kind; and, again, the different kinds point to different causes. With the ophthalmoscope, and on minute examination, we can distinguish four different forms.

No. 1. No morbid changes are perceptible in the choroid or in the transparent retina, beyond an extreme anæmia, either of the two or of the retina only.

Anæmia of the choroid, conspicuous through the groups of stellate pigment-cells being visible throughout the choroid, may be mistaken for atrophy of that tunic by those who are not familiar with the colour and shape of these groups in health.

The retinal arteries and veins are thinner the further they are from the optic disc. They are not tortuous nor unequally dilated. The optic disc is anæmic or anæmic and atrophic. It has a waxy-white and opaque colour.

No. 2. This form is described as inflammation of the optic disc, and descriptions of it are to be found in articles on Glaucoma and Embolism. It is the conjoined result of an impeded blood-supply to the retina and of impaired nutrition through arrest of the function of the optic nerve.

No. 3. Atrophy following retinitis or choroido-retinitis. The optic disc may be anæmic, or atrophic and white, or highly hyperæmic. Its margin, according to the changes in the choroid and retina, is either well defined or it merges into those of the tunics.

In addition to the decrease in the number and calibre of the arteries and veins, we meet with changes in transparency of the retina and with morbid pigmentation. Large arteries, though empty, can often be traced into the retina as whitish opaque lines. Some of the remaining veins appear varicose. The blood-vessels, according to the degree of atrophy, become thinner the further they are from the optic disc. Many can be traced to a short distance into the retina, others disappear in it close to the optic disc. In high degrees, none or a very few thin ones may be visible in the optic disc, extending but a little into the retina. The retina is semi-opaque in some (especially round the optic disc), transparent in other parts. The opaque portions shade off into transparent ones. The black and brown groups of pigment are the result of choroidal changes; the retina, *i.e.* the tissue occupying its place may be so transparent that it appears to be wanting; and the light and dark brown pigmentation of the anæmic choroid is unusually well seen, and presents a very striking appearance when contrasted with that of health.

Causes and general remarks.—Atrophy of portions of or of the entire retina is observed very rarely in aged persons to such an extent as to be regarded as morbid.

The few cases which have occurred resembled, as to ophthalmoscopic appearances, the form described as "retinitis pigmentosa."

The following changes lead to atrophy of the retina:—
1. Any of the lesions which during a long time prevent retinal impressions from reaching the brain, such as diseases of the brain or of the optic nerves. The atropic changes (form No. 1) in these cases appear late and progress very slowly. A certain amount of atrophy may again disappear if the extra-ocular lesion subsides.

2. All changes which mechanically cause anæmia of the retina, as embolism, inflammation of the optic disc, cupping of the optic disc, increase of tension, these lead to form No. 2.

3. A large series of intra-ocular changes, which may be separated into—(*a*) those which commence in the choroid, leading to atrophy of that tunic, and secondarily to atrophy

of the retina; or which during choroiditis invade the retina, destroying it from its outer surface (form No. 3); and (*b*) those which commence in the retina following retinitis (form No. 3), or are caused by pressure of the contents of the vitreous chamber upon the retina, destroying the latter from its inner surface (form No. 2).

Retinitis during Albuminuria.

This form of retinitis may appear whenever albumen is present in the urine; but it is most frequently observed in patients suffering from Bright's disease.

In the latter cases the retinitis has been considered a forerunner of the albuminuria. On careful examination, however, it will be found that alterations of the kidneys have existed long before; though the symptoms may have been so slight, or progressing so slowly, that the retinitis has raised the first suspicion of the presence of kidney disease. When meeting with this form of retinitis, we must be prepared to see other uræmic symptoms make their appearance in our patient, though they need not necessarily follow.

"Granular kidney," with dilatation of the cavities of the heart, and with hypertrophy of the left ventricle, have been found, not in all, but in most cases in which post-mortem examinations could be obtained.

Vision.—From the analysis of thirty-eight cases it appears that, in albuminuria, two chief causes of impairment of vision can be traced. The rarer one is "uræmia," the more frequent the form of retinitis which is to be described.

A combination of the two has occurred. Out of the thirty eight cases, thirty-two suffered from retinitis.

The loss of vision in those presenting symptoms of uræmia has been sudden, the patients becoming blind suddenly, remaining so for from a few minutes to a few hours, and then quickly recovering vision again. Vision, though for a time remaining normal after the first attack, has been observed to fail again gradually in those cases in which pain in the head, dizziness, convulsions, or paralysis, appeared as complications.

The impairment caused by retinitis is not so sudden as in uræmia. It increases gradually for a time, varies in degree, and the region of the yellow spot being generally implicated, is perceived at once. Yellow spots impair it more than the opaque gray and white ones. The former often intervene between the rods and the rest of the retina, and lead to destruction of that part of the retina. The patients complain of a mist intervening between the eyes and objects; of not being able to see so far; of perceiving objects more distinctly when held on one side, &c. Any other form of retinitis, affecting similar portions of the retina, gives rise to similar modes of impairment. By the aid of the ophthalmoscope we can decide whether the impairment is due to changes within the eye, and to what extent this may be the case.

We distinguish between those changes which occur in the course also of other forms of retinitis, and those which are characteristic of this form. The former are hyperæmia followed by swelling and loss of transparency of the retina. The characteristic symptoms are, brilliant yellowish-white and opaque spots round the optic disc, and in the region of the yellow spot. The hyperæmia shows itself by a fulness and tortuous course of the numerous, sometimes distinctly pulsating veins of the retina, with comparatively few and thin arteries, and by a red optic disc. This stage may subside, and no retinitis follow. If retinitis appears, it does so first round the optic disc.

The retina round the optic disc becomes swollen and opaque, or semi-opaque, and sprinkled with blood spots. The opaque portions shade off gradually into more healthy ones.

The optic disc when participating in the swelling and loss of transparency loses its defined margin. Its position can often only be inferred from the vessels of the retina converging towards it. These vessels generally cannot be traced into the disc, and appear much diminished in number. In the retina, round the yellow spot, a similar impairment of transparency is observed; the yellow spot often appearing as a red, or brownish-red, ill-defined dot in the hazy retina.

Round these foci of inflammation we find, solitary or in groups, spots of inflamed retina, varying in size from a minute dot to the size of a hemp seed, or larger. The course of the blood-vessels in the inflamed portions of the retina is more or less obscured. The blood spots may be so numerous or so large as to hide a considerable portion of the retina from view. The blood, by passing through the retina, may escape into the vitreous chamber, and give rise to proliferation of the cells in the vitreous and to floating opacities, or it may accumulate between the choroid and retina, and (as occurred in three cases) lead to displacement of the retina. Some of the blood spots are round, others are oval or streaked. The latter generally appear first, and are situate immediately beneath the optic nerve-fibres. This stage of the retinitis (the swelling, loss of transparency, and blood spots) having continued for some time, the characteristic brilliant yellowish-white, or buff-coloured, and opaque spots appear in the gray swollen retina. In the region of the yellow spot they generally appear in groups, having a somewhat linear arrangement, and giving this part of the retina a striated appearance; while round the optic disc they soon run together into one or several larger brilliant yellow patches, sometimes surrounding the disc like a wall, or striking it on one side, or separated from it by a narrow gray and opaque band of retina. Their number varies, one only or a few may be found round the optic disc, or in the region of the yellow spot; or the parts round the optic disc may be thickly sprinkled with them. The margins of the larger ones often remain surrounded by blood spots for some time, the larger blood spots of the retina passing over or at the side of the spots.

The opacity and swelling of the retina, and the tortuosity and the enlargement of its veins disappear first. The outlines of the optic disc become visible again, together with the vessels in it, and we may find the spots which appear in the later stage of the retinitis alone or mixed with blood spots, while the rest of the retina has regained its transparency.

The spots may become obscured through fresh hæmorrhage. They are easily distinguished from atrophic portions of choroid, by their yellow colour, by the frequent simultaneous presence of blood spots, and by the absence of the pigment patches which invariably adjoin completely atrophied portions of choroid.

The yellow spots disappear gradually in from three to six weeks, leaving the retina transparent. We are then able to judge of the changes of the choroid in which yellow patches similar to those in the retina occur, and which after having disappeared leave the hexagonal cells disturbed, giving rise to groups of brownish dots and patches upon more or less atrophic portions of the choroid. These, together with some anæmia of the optic disc and of the retina, are often the only vestiges left of the retinitis.

Dissections of eyes, in the various stages of retinitis, of persons suffering from albuminuria, have disclosed the following morbid changes.

The blood spots.—These have been met with in, upon, and beneath the retina. In the retina—especially in the inter-granule layer—the blood has been found accumulated in cavities formed by displacement of the granules and of the connective tissue. These structures having become soaked with probably highly albuminous fluid, and with blood-corpuscles, have undergone the secondary changes — loss of transparency, fatty degeneration, thickening of the connective tissue, &c.,—independent of those caused by the blood. The blood itself, the colouring matter having disappeared, had formed structureless, semi-transparent, or yellowish-gray and opaque coagula.

Upon the retina, and among the optic nerve-fibres, such coagula have been found side by side with fresh red clots of blood.

Similar yellowish-white and opaque and red clots have been met with in and upon the choroid. The fluid which escapes from the blood-vessels has probably a great share in the formation of the yellow patches.

Peculiar changes, by some described as "sclerosis," have been found in the coats of the blood-vessels, especially in those of the small arteries, and of the capillaries of the retina and of the choroid. These vessels in many places have appeared somewhat flattened, and more tortuous, with the walls thickened through a homogeneous, strongly light-reflecting "amyloid," not quite transparent, substance. The thickening sometimes has been so considerable as to narrow, and in some cases completely to occlude the lumen of the vessels. This probably has assisted in the formation of small aneurismata, found in many parts of the capillaries.

The retina or choroid adjoining the thickened portions has appeared hypertrophied.

The yellow or buff-coloured spots when situated in the retina itself have been found amongst its ganglion-cells, but more frequently in the granule layers. They have been most numerous round the yellow spot, but have occurred throughout the retina, the thickness of which, as could be seen in sections taken from these parts, has been considerably increased.

The yellow spots, if examined by reflected light and with a low magnifying power, appear yellowish-white, opaque, and roundish; by transmitted light, black and opaque. Examined in sections, and with a high magnifying power, they appear composed of granule cells, of fat-globules, of fibrin, and of altered fatty connective tissue.

The granule cells have exactly the shape and nearly the size of the granules of the granular layer, with the difference that they are less transparent, contain a larger number of minute granules, and are mixed up with free granules and fat-molecules. It is probable that, soaked with albuminous fluid, they, together with the surrounding parts, lost their transparency and underwent fatty degeneration, under the peculiar appearance of yellow spots.

The striated appearance of some of the yellow and other opaque spots is readily explained by the arrangement of the connective tissue of the optic nerve-fibres and of the retina; which tissue, besides losing its transparency, and undergoing fatty degeneration among the yellow patches of the granular layer, presents peculiar changes, which, like those in the capillaries and small arteries, have been described as hypertrophy and sclerosis; and consist in thickening of the nuclei and of those portions of the fibres which join the inner membrana limitans, and which are found passing across the layer of optic nerve-fibres. The former, instead of being transparent, are strongly light-reflecting, and if present in groups produce (seen with the naked eye) an appearance of brilliant white or grayish-white streaks, spots, and patches in the retina, or among the optic nerve-fibres, sometimes marking the blood-vessels. Many of the brilliant grayish-white spots seen during life with the ophthalmoscope are caused by the thickened ("sclerosed") connective tissue of the retina.

The grayish-opaque or semi-opaque appearance of the margin of the optic disc, and of the retina, observed in the commencement of retinitis, is the result of infiltration "with serum," causing swelling of the retina, and an increase of cells and nuclei derived from the connective tissue—"glioma." This swelling round the optic disc, especially if considerable, naturally impedes the passage of blood into and through the disc, and favours the rupture of blood-vessels.

Treatment.—The general medical treatment is that of albuminuria. Much benefit is derived from large doses of strychnine combined with steel. No advantage has as yet been gained from surgical treatment, as iridectomy, &c. Rather abundant local depletion (*e. g.* by applying from six to eight leeches to the corresponding temple) is found of great use only in the commencement of the retinitis, but is injurious when once the yellow patches have appeared. If these spots and patches are numerous in the retina, or if blood has been effused into the vitreous chamber, atropia, applied twice daily, will be found of use. The eyelids should be kept closed by a bandage if one eye is affected, or a shade should be worn if both eyes are affected. The local treatment is discontinued as soon as the yellow patches have disappeared. Displacement of the retina following extensive hæmorrhage or destruction of the retina through atrophy, &c., is the frequent cause of permanently impaired vision. The prognosis, as regards recovery of vision, is good if there are but few spots of the retina affected. An improvement may be expected as long as yellow or blood spots are visible.

Embolism or blocking up of the Blood-vessels of the Retina by Coagula, &c.

Loss of vision from this cause is of frequent occurrence. It has been observed almost exclusively in persons who have suffered from disease of the heart, or aneurism of the aorta, or from other diseases (*e. g.* pyæmia, albuminuria) which give rise to embolism.

Choroiditis, ophthalmitis, cerebral embolism, have occurred as complications.

In a young man who died suddenly of aneurism of the aorta, embolism occurred in both eyes, in all other cases on record only in one eye.

The fact of sudden loss of vision, together with the changes perceived with the ophthalmoscope, are very characteristic.

The ages of the patients in the cases on record varied between twenty-six and seventy.

Vision.—"A coloured cloud or gray mist suddenly intervenes between objects and the affected eye." This, within a few seconds or minutes, is followed by complete blindness, or by bare perception of light. In several cases vision was lost during sleep; in none has any useful amount been regained.

The changes observed with the ophthalmoscope in the vessels of the retina, in the optic disc, and in the retina at the region of the yellow spot, are particularly striking. The optic disc at first either remains pink or appears anæmic. It gradually (in from two to four weeks) becomes white, and at last slightly cupped, and atrophic. No arteries (no blood in the arteries) or only one or two very thin ones are visible in the optic disc, or in the retina near it. Clots of blood may, however, be seen in some of the arteries, especially in those coming from the region of the yellow spot. These clots finally disappear, and grayish opaque lines indicate the places occupied by the arteries. The veins are thin in the optic disc, and somewhat fuller and unequally dilated in the retina, especially at its equatorial region. A peculiar undulating movement, or a momentary advance of a portion of blood from one part of a vein towards another part nearer the optic disc, is observed soon after the embolism has occurred, and again if some circulation returns.

The retina adjoining the optic disc becomes slightly opaque; and in one case this was the only change observed, besides the absence of blood from the arteries. In all the other cases the retina in the region of the yellow spot lost its transparency and assumed a grayish-white and opaque colour, which gradually shaded off into the transparent part, and in extent corresponded about to the portions occupied by the oblique radial fibres. In the middle of this opaque portion (the centre of the yellow spot) a small red spot (attributable to the colour of the choroid shining through this very thin part of the retina) is observed. Minute shining opaque spots appear as the general opacity of this region vanishes, and finally the retina resumes its transparency.

The opacity of the retina appears in from twenty-four to sixty hours after the embolism.

Treatment.—No success has as yet been obtained by local means. The transparency of the retina returned unusually rapidly in the cases in which iridectomy had been performed, but without any favorable results as regards vision.

In several patients, embolism of a retinal artery has been the means of leading to the discovery of lesions in other parts of the body, and this in its turn has led to appropriate general medical treatment.

Retinitis pigmentosa.

This term has been introduced into practice, although the pigment changes so characteristic of this form of retinitis are chiefly due to alterations of the choroid.

Retinitis pigmentosa is often observed in children; in many instances such children are deaf and dumb, and are the offspring of parents who are blood relations. And it has sometimes occurred in several children of the same family. It has also been observed in persons who have suffered severely from ague, and in their children.

In some cases the first symptoms have appeared as late as the age of fifty, the patients still retaining perception of light at the age of seventy. It has hitherto always been found in both eyes of the same person, though it may vary in degree in the two eyes.

Vision.—Two peculiarities of this form of retinitis are, first, the comparatively good direct vision, and secondly, the early appearance of night-blindness. The latter is not only marked at dark, but also whenever in the course of the day the light is unusually dull.

The loss of vision steadily, though slowly, advances from the periphery towards the centre of the retina. The vision, if examined in broad daylight, may appear normal, while in artificial light (which should always be adopted for the investigation) the periphery of the retina is already found impaired.

Such patients may for ten or fifteen years be able to read small type, especially with the aid of convex glasses, and yet all the time may not be able to walk about safely, in consequence of the more peripheral parts of the retina having been destroyed. For the same reason large letters and large objects are less easily recognised in advanced cases, parts of their images being formed in damaged portions of the retina. Exceptionally, a blind zone of retina may intervene between a sensitive, more peripheral, and a more central one, also sensitive; or the central portion may become blind before a more peripheral one.

Slight increase of tension; a pupil of medium dilatation, sluggish or fixed; and often, especially in elderly people of dark complexions, a peculiar greenish reflection from its area, when viewed with focal light, have been noticed. In the advanced stages of the disease, we find in many of the cases opaque white dots or streaks on the surface of the lens, or a chalky cataract, which latter in several instances has been found to be more or less dislocated.

On ophthalmoscopic examination, we observe the characteristic signs of this disease, which are peculiar dark-brown or black patches, many of which are star-shaped, somewhat resembling bone-corpuscles. These gradually become more abundant. Many anastomose one with another, forming a black web. They appear first at or near the ora serrata, and we should never neglect inspecting this part by making the patient look in various directions. They at last appear upon the choroid, near and around the optic disc. Their shape, their great number near the ora serrata and at the equator of the choroid, their rarely being mixed up with atrophic transparent patches, and their being situated upon somewhat anæmic portions of choroid, distinguish them from pigment spots, following other lesions of the choroid. The groups of stellate pigment cells are unusually well seen in the anæmic choroid (deprived of its hexagonal pigment cells), and give it a honeycomb appearance. The anæmia of the choroid, the atrophic changes of its capillaries, and its "epithelium," and of the retina, advance from the ora serrata towards the region of the yellow spot, which latter part longest retains a more natural appearance.

The optic disc, as soon as the amblyopia is well marked, appears pale pink, and somewhat ill defined. In advanced cases it assumes a dull waxy-white colour; its nerve-fibres can be recognised long after the retina has been destroyed. The retinal arteries and veins, in proportion as the atrophy increases, become thinner. The lumen of the arteries becomes narrowed, through thickening of their walls. They appear less numerous. In some cases only one or two thin vessels can be seen passing through the optic disc, and traced a short distance into the retina. The retina undergoes atrophy, and becomes thinner. At first it is transparent, but it gradually assumes a dull translucent aspect, and again becomes transparent in advanced atrophy.

The appearance of the black pigment spots is invariably accompanied by destruction of the rods and bulbs; but the atrophy of the retina stands in no relation to the number and size of these black spots. There may be but a few of these, while all the rods and bulbs are destroyed, the hexagonal cells and their granules disappearing somehow.

Beyond the degree of acuteness of vision, the number and size of the blood-vessels in the optic disc are the best guides as to the state of nutrition of the retina.

In many places, fragments of retina and choroid remain adherent to each other, when on dissection attempts are made to separate the two. The rods and bulbs and layers of the retina adjoining them are destroyed first. The presence of the granules of the "epithelium," in the shape of characteristic black spots and granules, render it probable that they have been pushed into the retina by morbid changes coming from the choroid.

Rupture of Retinal Blood-vessels—Effusion of Blood into the Retina—"Apoplexy of the Retina."

General remarks and causes.—Effusion of blood into the retina from rupture of its blood-vessels has been observed—

1. After injuries, through concussion or direct wounds.

2. In the course of those changes which tend to impede the passage of blood through the optic disc (as glaucoma, inflammation of the optic disc, and of the adjoining retina), or through the orbit, especially when the cause is situated close behind the eyeball, or at the fissura orbitalis superior.

Sudden closure of the jugular veins on both sides has occasioned this affection.

3. In the course of changes within the eye (as tumours, retinitis, myopia), which give rise to hyperæmia of the retina, and choroid, with atrophy.

Among the general diseases which are frequently complicated by bleeding into the retina, are albuminuria, diabetes, atheromatous changes of the arteries, the lesions which give rise to embolism, especially in the sinus cavernosus and at or near the fissura orbitalis superior, diseases of the heart, disturbances of the circulation in the brain from inflammation, tumours, hydrocephalus, &c.

Other frequent causes in young, weak, pale persons are found in anomalies of menstruation and disturbances of the circulation during pregnancy, parturition, and suckling. General symptoms preceding the effusion of blood may be entirely wanting. Severe headache, dizziness, impairment of vision on stooping, frequent bleeding from the nose, extreme nervousness, are among those usually complained of. Rupture of blood-vessels in other parts, the occurrence of apoplexy in other members of the patient's family, are frequently observed. The left eye is the one most frequently attacked, and the greater number of cases occur in the spring. Very frequently rupture of the blood-vessels of the retina is the first symptom of advanced morbid changes of the entire vascular system.

Our attention should at once be directed to the functions of the brain, heart, &c., and our advice and treatment should anticipate the changes and accidents which we have reason to dread when meeting with ruptured retinal vessels.

We shall confine our remarks to those cases in which the blood effusion is the sole or most prominent change, and in which this change is not preceded by glaucomatous symptoms. As regards the effusion of blood occurring during the different forms of retinitis, glaucoma, and myopia, we refer the reader to articles on these subjects.

The diagnosis of blood effusion is made by the aid of the ophthalmoscope. We sometimes are prevented from seeing the retina in consequence of the blood having entered the vitreous chamber and diffused itself through it, or in consequence of its having spread itself over the retina. This occurs more frequently after rupture of retinal vessels at the ora serrata, or near the yellow spot. Or the blood may perforate the retina towards the choroid, and give rise to one or several large, flat, dark red, well-defined patches, obscuring the uniform red colour of the choroid, and often overrun by retinal vessels, or displacing the adjoining retina. Extravasations of blood into the choroid are easily recognised if they are situated in the spaces between the large choroidal veins.

A varicose condition of the unusually dark red vessels of the retina, with some loss of transparency of the latter, obscuring the outline of the optic disc, and with small spots and streaks of blood in the retina, have been observed in cases of hæmorrhage between the choroid and sclerotic.

By the shape of the blood spots we can frequently tell what part of the retina they occupy. Blood effusions among or immediately beneath the layer of optic nerve-fibres appear as red streaks; those in the retina generally appear as small roundish spots, occurring on either side of a vessel or parallel to one of its walls. Or one large roundish clot, as a little smear of blood, may be seen close to the side of a vessel, indicating the spot from which the effusion came. One or several of the large vessels of the retina may be seen ending abruptly at the blood spot, or becoming thin suddenly, while on the other side of the spot they appear large. Thus it may happen that the effusion from one vessel may interfere with the circulation in another. To many of the blood spots no vessels can be traced.

Colour of the effusion and its absorption.—Fresh blood spots have a bright red colour. They mostly proceed from ruptured capillaries, their colour soon changes into a dark, then into a brown red, and at last disappears entirely, leaving the retina transparent, or grayish and opaque. A blood spot of the size of the optic disc, when entirely situated in the retina, requires from two to ten weeks for absorption. It is often accompanied by temporary swelling, and loss of transparency of the surrounding retina. It decreases from the margin towards the centre. We frequently find blood spots which do not pass through the usual changes of colour which healthy blood manifests when undergoing absorption, but which rapidly, sometimes within a few hours, assume a buff or yellowish, or white and opaque colour. It is this rapid change of colour which so frequently is observed in persons suffering from albuminuria.

The retinal vessels may become obliterated or narrowed, or may retain a tortuous course on either side of the seat of the effusions. Cicatrices on the retina and displacement of circumscribed portions are the changes frequently observed after absorption. The optic disc, at first hyperæmic, resumes its normal colour, or becomes somewhat anæmic. It is not infrequently the seat of blood effusions. The tension of the eye in profuse hæmorrhage into the retina is increased, and slight cupping of the optic disc remains after absorption is completed. It decreases, however, and the eyeball becomes soft, if the absorption of blood is incomplete and new effusion occurs.

Vision.—The obscuration of vision is sudden, and varies in kind and degree with the extent and localisation of the effused blood.

The manner in which vision further becomes impaired depends upon the part of the retina occupied by the effusion, upon its quantity, and upon the changes which follow in the retina and in the adjoining tunics. The nearer to the yellow spot the effusion, the more does it affect vision, and the sooner is it noticed by the patient; *e. g.*, a patient suddenly observes a dark streak or cloud, with a reddish halo round it, "straight in front of one eye," and on ophthalmoscopic examination we find a small, dark red and opaque clot of blood in the retina, and in the vitreous chamber adjoining it. The same patient also states that when looking at his hand with the affected eye, he only sees the tips of the fingers and the wrist.

Another patient only sees the tops or lower parts of objects; and, with the ophthalmoscope, we find blood spots also on the more eccentric parts of the retina. Or a patient can just see the shadow of objects, and we find the whole of the retina sprinkled with numerous small blood spots. Another sees objects, straight lines, &c., crooked; and we find effusions of blood beneath the retina (from a retinal vessel), with displacement of small portions of retina.

Muscæ caused by clots close in front of the retina are often complained of. Photopsiæ are rarely met with in the beginning or in the further course of the hæmorrhage.

A rapid improvement of vision is generally observed, unless the blood effusion has damaged the texture of the optic nerve or of the yellow spot. The prognosis is rendered unfavorable chiefly by the age and state of health of the patient, and by the tendency to relapse in the eye or elsewhere. The portions of retina at the margin of the effusion recover first. Patients at such periods often state that objects appear distorted or crooked, and surrounded by a mist, &c. The effects on vision of solitary effusions further distant from the yellow spot frequently disappear entirely, while those near or at that spot generally leave vision impaired, but much less so than we might expect at the outset of the attack.

The slight permanent impairment of vision, compared with the extent of the effusion at first, is probably due to the blood passing through the retina and spreading over it more frequently than it actually disturbs a large part.

One patient, in whom an effusion of blood, of the size of a fourpenny-piece, was observed upon the region of the yellow spot and of the adjoining retina, and who could not recognise a hand when held in front of the eye, was able to read small type a year afterwards, though there remained a bluish, opaque, and "blind" patch across the region of the yellow spot.

Better vision is often recovered after absorption of one large effusion than after that of numerous smaller ones.

None of the changes in the exterior of the eye indicate the presence of hæmorrhage into the retina. Some hyperæmia of the conjunctiva, with slight increase of tension, is often observed at the outset. The pupil, generally of medium size, acts in proportion to the sensibility of the retina.

Treatment.—Locally the treatment consists in the application of a slight pressure bandage over the closed eyelids of the affected eye. The bandage has to be worn for from two to three weeks, after which time, if there is no intolerance of light, the eyes may be used with moderation for "near work."

Atropia, applied twice daily until all blood has become absorbed, can be recommended in most cases. No surgical treatment has been found of use, provided the tension is normal.

We must, as regards the recovery of vision, be careful when expressing an opinion :—partly, because the bleeding upon the retina and into the vitreous chamber may mislead us, and make us suppose a large portion of retina to have become implicated, so that after holding out little hope of recovering useful vision, we may find the retina, after absorption of the blood in the vitreous chamber, but little damaged and vision normal, or nearly so:—and partly, because the tendency to fresh effusions prevents our being able to assure the patient of permanent recovery.

The general medical treatment entirely depends upon the general health of the patient, and upon the cause of the effusion. We look upon the latter as a general warning as regards the state of the patient's blood-vessels.

DISPLACEMENT OF THE RETINA. *Detached retina; subretinal Dropsy.*

General remarks and causes.

The displacement of portions of the retina or of the entire retina into the vitreous chamber is a frequent occurrence, and (injuries excepted) is generally observed in persons beyond the middle age. It is, as a rule, readily recognised by the aid of the ophthalmoscope, and is always accompanied by marked disturbance of vision. The vitreous substance, which in health is of considerable consistence, is remarkable for the rapidity with which it diminishes by pressure whether from without or within; tumours of the choroid, or accumulations of fluid between the choroid and retina, readily cause it to become less, and the retina thus often becomes detached from the choroid and pushed into the vitreous chamber. The beginning of the displacement often escapes the patients' notice at first, unless it comprises the region of the yellow spot.

Causes.—1. Injuries causing concussion and subsequent cicatrization or absorption of the vitreous substance. Months may pass before the displacement makes itself perceptible to the patient, unless it be complicated by effusion of blood into the vitreous chamber. Large perforating wounds are the more likely, sooner or later (in some instances six or seven years after the injury), to be followed by displacement of the retina, the more the wound encroaches from the ciliary region upon the area of the retina.

2. Solid or liquid effusions upon the inner surface of the choroid, occurring spontaneously, or tumours of the choroid, or effusions of blood between sclerotic and choroid, as observed in glaucoma, during irregularities of menstruation, &c., causing absorption of the vitreous substance by pressure, and, simultaneously, displacement of the retina.

3. Myopia.—The undue distension of highly myopic eyes, with atrophy of the choroid, a great tendency to effusion of blood, and a liquid state of the vitreous substance, seem to be the causes of the displacement being more common in myopia (and even in both eyes of the same person) than in any other group of cases. We should, in every case of displacement of retina, investigate the refraction of the fellow-eye in order to see whether myopia is present.

4. Changes of morbid products (pus, lymph, &c.) which may appear in the vitreous substance during choroido-retinitis. These and the altered vitreous substance gradually shrink and drag the retina away from the choroid. Foreign bodies, cicatrices, suppuration of the vitreous substance, often destroy vision in this way.

Spontaneous effusions of blood into the vitreous chamber, followed by morbid changes of the vitreous substance round the effused blood, also belong to this group.

Diagnosis and course.—An examination with the ophthalmoscope, the pupil being dilated by atropia, is necessary in order to recognise the displacement. The exterior of the eye offers, in most cases, no peculiarities, though there may be some ophthalmia, or sluggishness of the pupil, or slight divergent strabismus. In many advanced cases chronic iritis, followed by posterior synechiæ, and frequently by cataract, occur as secondary changes. The eye gradually becomes softer, with very little pain or none at all, and with slight outward vascularity. Sometimes we meet with increased tension and other glaucomatous symptoms, as fulness of the vessels emerging from the sclerotic near the cornea, and anæsthesia of the latter, and severe pain in and around the eye. In every case of this kind in which the eye has been removed, a tumour generally springing from the choroid in the region of the yellow spot, has been found, besides the displacement of the retina; the latter being one of the symptoms of the tumour. In well-marked cases we see on the first glance, close behind the pupil, a dark, floating, well-defined cloud, more or less intercepting the red reflection. This cloud, viewed "directly," is seen in its real place, generally behind the lower margin of the pupil; while examined "indirectly"—"in the inverted image"—it appears to be suspended behind its upper margin. Very small portions of a semi-transparent, or larger portions of a transparent, but only slightly displaced, retina can, by an experienced observer, be recognised by the peculiar dark, uniformly red colour and undulating appearance of the vessels. Larger portions generally are more altered in colour, being semi-opaque, or gray and opaque, and floating in some part (generally in the lower part) of the vitreous chamber. We are, however, not justified in pronouncing a retina displaced as long as we have not succeeded in tracing vessels from the optic nerve into the opaque floating portion, or in recognising the vessels in the floating membrane by their shape, mode of ramification, &c., as retinal vessels. The vessels appear as dark red, or blackish, well-defined lines, branching in, and following the undulating movements of, the opaque floating substance when the patient moves the eye, while the vessels in the optic disc and in the retina in situ have the natural transparent red colour. The displaced retina, next to the vessels, often presents a peculiar silvery white and opaque colour. In many cases only part of the retina is displaced, though, especially by injuries, any part or the entire retina may become displaced.

Varieties.—

1. Displacement of delicate folds, which radiate from the optic disc (as seen in myopia), or in circumscribed patches (as seen in glaucoma), by effusion of blood round the optic disc.

2. Displacement of the lower part of the retina from below the optic disc to the nearest part of the ora serrata. This variety is the most frequent. Its frequency probably depends upon the fact that when other parts of the retina are displaced primarily by fluid, the fluid settles at the most depending part of the eyeball, between the choroid and retina. The displaced retina may be transparent or semi-transparent, so that the

choroid and the colour of the fluid between it and the retina can be recognised.

The retina frequently appears silvery white or grayish and opaque (in old cases yellowish opaque). Its colour in a great measure depends upon the colour of the fluid accumulated between it and the choroid. The extent of the undulating movements of the displaced portion during movements of the eyeball varies. Gray and opaque or brownish-red shreds may be seen floating in the vitreous chamber, near the displaced retina. The retina and the choroid adjoining the displaced portion frequently have a healthy appearance, or occasionally a yellow and opaque line in the retina, with or without small blood-spots on it, indicates the boundary between the displaced and healthy retina.

3. After an injury a fold of displaced retina has been observed to extend from the seat of injury (*e. g.* from the cicatrix caused by a perforating wound through the tunics of the eye near the ora serrata) to the optic disc, while the remainder of the retina has continued in situ for several years.

4. Small folds of any part of the retina may become displaced by tumours of the choroid during extension of the eyeball in myopia, &c. A frequent seat of such folds is the region of the yellow spot.

5. The retina may become separated from the layer of optic nerve-fibres, so that it remains in situ while the optic nerve fibres become alone displaced, through changes of the vitreous substance (see a preparation in the Museum of the Royal London Ophthalmic Hospital, Moorfields).

6. The entire retina may be displaced ("funnel-shaped displacement"), only retaining its attachment round the optic disc, and along the ora serrata. In cases with increase of tension we may also find the zonula detached from the ciliary processes by the fluid between the retina and choroid. The retina may not only be displaced from the choroid, but also torn from its attachment round the optic disc. It then becomes puckered up at the hyaloid fossa; and, viewed through the crystalline lens, appearing yellow and opaque, it has been mistaken for intra-ocular tumour.

The optic nerve-fibres of eyes which have been blind for years, in consequence of the retina having been displaced, but the choroid appearing healthy, presented hardly any morbid changes; while in those eyes in which cartilage or bone has been formed upon the inner surface of the choroid the nerve-fibres have been found wanting, and their connective tissue atrophic.

In the case of an eye with extensive colloid changes in the choroid, and with displacement of the otherwise transparent but swollen retina, all the retinal elements could be recognised. The rods and bulbs, however, appeared swollen, and in many places they were wanting. Many of the capillaries were varicose, others aneurismatic. The changes found in several retinæ, which had been displaced for years, through hæmorrhage into the vitreous chamber and between the retina and choroid, were the following:

The retinæ appeared as semi-opaque membranes, the outer surfaces of which were sprinkled with colloid globules, blood-spots, granules of lime, and with patches of hexagonal cells. In the retinæ, near their inner surfaces, were numerous groups of brown and gray pigment-molecules, many of which appeared enclosed within shrivelled blood-vessels.

The vitreous substance undergoes various changes; sometimes it becomes fluid, but remains transparent, or the portion within the area of the ora serrata retains its normal consistence, while that in the area of the retina becomes fluid, as has been observed in some myopic eyes. Fibrous, cartilaginous, and connective tissues are frequently developed in the vitreous chamber after acute inflammation, and by their contraction materially assist in the displacement of the retina.

The fluid occupying the space between the choroid and displaced retina is often transparent or yellowish, and always more or less albuminous. Sometimes it is mixed with blood, often with an abundance of granule-corpuscles or with cholesterine-crystals (which may be visible through the retina); or traversed by bands of fibrin or of connective tissue, extending from the inner surface of the choroid to the outer surface of the retina.

An accumulation of lime-particles lying loosely upon or attached to the choroid, plates of bone of varying thickness, and colloid formations, are the products most frequently found upon the inner (retinal) surface of the choroid.

Vision.—The displacement, if it commence without pain or outward inflammation, may escape notice for some time, until the patient accidentally discovers that the sight of the eye is failing. Some patients have stated that they suddenly found they could only see the halves of objects, or that a black, well defined cloud (sometimes appearing at first red, then yellow) obscured some, and generally the upper parts of objects, when viewed with the defective eye only. The complaint, for example, of seeing only the body, and not the head of a person, is almost characteristic of displacement of the lower part of the retina. Objects at the outset of the displacement often appear crooked, bent, or multiplied, or as if surrounded by a red haze. These changes in many cases, especially if following an injury of the tunics occupying the ciliary region, may for years be preceded by muscæ or by some "dimness." In some cases the muscæ appear suddenly, and the displacement shortly follows.

In some cases severe throbbing pain in the eye and temple, fiery circles, stars suddenly appearing and disappearing, precede the displacement for weeks.

Some patients state that after bodily exertion a flash of light passing across the eye has been followed by sudden impairment of vision.

Owing to changes in the retina adjoining the displaced portions, the impairment of vision is often more considerable than might be expected from the extent of the displacement, especially in fresh cases. The transition from the blind to the normal parts of the retina is gradual, if the displacement is small; it is sudden, if large portions of retina are displaced.

The greater part of the retina may be displaced, and the patient may still be able to read small type, if the region of the yellow spot has remained intact; or all perception of objects may be abolished if the retina round the optic disc has been displaced, though there be a large excentric portion in its proper position. Only the upper or lower portion of an object is perceived if the upper or the lower half of the retina only is displaced. In a few cases of complete displacement of the retina the power of perceiving light has been retained.

Secondary changes often destroy the sensibility of portions of retina which are not displaced.

Vision may become much improved after the changes in the retina next to the displaced portion and those in the vitreous chamber have subsided. The fluid often becomes less or alters its position, and the sensitive parts of the retina are thus less excluded from vision. It not infrequently happens that the displacement is complicated with effusion of blood into the vitreous chamber or with cataract, and the retina may not be accessible to ophthalmoscopic examination. It is particularly necessary in such cases to recognise the presence of displacement by testing the sensibility of the retina by artificial light. For this purpose the patient is placed in a dark room, and the flame of a candle is moved before the suspected eye, the healthy eye being closed; the flame can be perceived only by sensitive portions of the retina. The lower part of the retina being the one which is generally displaced, we find that if the flame be held opposite it (that is, above the level of the pupil) it cannot be perceived. In cases of cataract this is conclusive. In effusion of blood into the vitreous chamber a doubt remains as long as the blood is accumulated at the most dependent part, thus preventing the light from reaching the retina. In such a case the diagnosis remains uncertain for a time.

Treatment.—The chief object is the speedy removal of the fluid from between the retina and choroid. The spontaneous "absorption" of this fluid, in part or *in toto*, has frequently been observed, though the retina, returning to its former position, has never resumed its function. If the displacement is limited and the displaced retina is transparent, or nearly so, and the patient otherwise in good health, we order from six to twelve leeches to be applied to the temple of the affected side, at bedtime, after which both eyes are kept closed for two or three days, and the patient is confined to bed. The improvement after repeated leeching is in some cases very great. Patients who had bare perception of shadows have been able, after the second leeching, to read large letters. In some, however, effusion of blood had occurred between the choroid and retina; in others no improvement followed.

If the entire retina is displaced, no treatment restores even perception of light.

An important local remedy, which, though it may not effect improvement of vision, seems to retard or prevent further displacement, is the application of atropia. This should be repeated three times weekly, so as to keep the ciliary muscle of the affected eye at rest. No treatment tending to restore vision, or to arrest the displacement, need be adopted—

1. If the entire retina is displaced, or if the displaced retina has lost its transparency, or if there exists atrophy of the rest of the retina and of the optic disc.

2. If the displacement occurs in the course of ophthalmitis or of suppuration of the retina, or if suppuration of the vitreous substance within the area of the retina has set in.

3. If the displacement is a complication of intra-ocular tumours.

In the two latter cases the excision of the eyeball may have to be performed.

Myopic persons are the only subjects in whom the displacement has repeatedly been observed in both eyes, though there seems no reason why it should not occur in both eyes of other persons also. The displacement of the retina in one eye does not by sympathy seem to produce a similar change in the fellow-eye. The observation that the displaced retina occasionally becomes ruptured spontaneously, causing the fluid between it and the choroid to escape into the vitreous chamber, while the displaced portion of retina resumes its position upon the choroid, has led to the treatment of perforating the displaced retina by operation. No serious accidents have followed the operation in any case.

The earlier the operation is performed, the greater is the improvement of vision that may be obtained. This sometimes is almost immediate, or appears a few days later.

The disappearing or the diminution of the displacement, a turbidity of the previously transparent vitreous substance, improved sensibility of the peripheral parts of the retina, and especially our being able to perceive the rent in the retina with the ophthalmoscope, are signs of the success of the operation. Immediately before performing the operation we ascertain the degree of impairment of the vision, and the extent and position of the displacement. The patient being seated as for the ophthalmoscopic examination, the eyelids kept open by the wire speculum, and the eyeball fixed by an assistant, the operator with the left hand uses the ophthalmoscope, and having by "direct" examination obtained a view of the most prominent portion of the displacement, thrusts a cataract needle of sufficient length through the tunics into the vitreous chamber at the spot which seems most suited for reaching the displacement, watching the passage of the needle across the vitreous chamber, and through the displaced retina into the space between it and the choroid. Having by its aid inserted the first needle, he gives the ophthalmoscope to an assistant, who throws light upon the eyeball, while the operator passes a second needle through the tunics, about a third of an inch from the first one, carrying it as nearly as possible to the spot where the first one has passed the displaced retina. After having brought the second in contact with the first needle, he thrusts it on still a little further (about to the same extent as the first one). The points of the two needles are now supposed to be in the space between the choroid and displaced retina, their stems crossing each other.

The object of the further manœuvre is, to tear a hole in the displaced retina. This is accomplished by approaching the handles of the needles towards each other (without withdrawing them or pushing them further into the eye, and without making the movements so extensive as to cause the points to touch the choroid), thus causing the stems to recede.

Having manipulated with the needles in such a manner as to make the existence of a rent in the displacement probable, one needle is withdrawn, and the ophthalmoscope is again used to ascertain, if possible, the effect of the operation.

If the previously transparent vitreous substance is now turbid, or a rent is visible in the retina, or flocculi of retina are seen projecting from the displacement, the second needle is also withdrawn. The manipulation with the two needles should be repeated if no alteration has occurred in the appearance of the displacement. The patient is put to bed and the eyelids of both eyes are kept closed for a few days. In six cases thus operated upon no evil results followed the operation itself. Our experience as regards the kind of cases in which the operation should be recommended, and the final result, is too limited to enable us to establish any rules.

The prognosis is under all circumstances very gloomy, and justifies our recommending an operative treatment, which in the few cases in which it has been adopted has had some favorable and no unfavorable results.

In one case a small portion of the retina, between the yellow spot and the optic disc, was found displaced. A divergent strabismus which existed simultaneously was remedied by operation. A few weeks after the operation the vessels in the displaced portion of the retina resumed their natural course, and vision became much improved.

CLINICAL REMARKS

ON PERFORATIONS

AND

SOME OTHER MORBID CONDITIONS

OF

THE MEMBRANA TYMPANI.

BY JAMES HINTON.

Owing chiefly to the labours of Mr. Toynbee, our knowledge of the diseases to which the membrana tympani is subject has been of late years greatly increased. Of some of these diseases it is my aim in the following paper to give a brief account.

Before proceeding, however, to describe its morbid conditions, a few points in respect to the healthy membrane may be referred to. It is composed of six more or less distinctly separable layers, the epidermoid, the dermoid, the radiate fibrous, the circular fibrous, the mucous, and the epithelial.[1] Of these laminæ the dermoid and the mucous are the least complete, and each of them seems to be chiefly made up of a thin layer of connective tissue, in which the nerves and vessels of the membrane are distributed. Essentially the membrane consists of a thin plate composed of characteristic elastic fibres, arranged in rays on the outer, and in circles on the inner aspect, and covered externally by skin and internally by mucous membrane. It contains no gland structures, but on its inner surface, near the border, there exist minute projections of the mucous membrane,

[1] Mr. Toynbee, "On the Structure and Functions of the Membrana Tympani," 'Philos. Trans.,' 1851.

which contain vessels but no nerves, and are some of them sessile, others attached by pedicles.[1] The radiate fibres are most numerous at the centre, very few at the upper part; the circular are scanty towards the centre, and are most numerous near the circumference, which, however, they scarcely reach.[2]

Subsequent investigations have added little of moment to Mr. Toynbee's description. Dr. Von Troeltsch[3] has pointed out that the cellular corpuscles which lie among the fibres of the fibrous laminæ are provided with numerous ramifying and anastomosing processes, and has assigned the names of "anterior and posterior pockets of the membrana tympani" to two small membranous folds which may be seen on the internal surface of the membrane at its superior part. The posterior and larger of these "pockets" is bounded by the chorda tympani nerve, and contains some of the peculiar fibres of the fibrous laminæ, the anterior is a fold of connective tissue merely, attached to the structures which pass through the glaserian fissure.

The membrane is supplied with blood both on its external and its internal surface. The external blood-vessels pass downwards from the roof of the meatus, in the dermoid layer, to the centre of the membrane, whence they radiate towards the cir-

[1] Gerlach, Von. Troeltsch, 'Anatomie des Ohres,' p. 38.

[2] The varied distribution and length of these fibres have given rise to many speculations as to the functions of the membrane. Mr. Pilcher considered that each radiate fibre vibrated to its own note, conceiving that there existed also corresponding variations in thickness, density, and tension—the same view that has been frequently suggested respecting the cochlea, and recently sustained with so much fresh evidence by Helmholz ('Lehre von der Tonempfindungen,' p. 198). Erhard ('Clinische Otiatrie,' p. 23) holds that the varying density and bulk of the concentric rings of circular fibres give to the membrana tympani the power of resounding to notes of every pitch, which cannot be imparted by varying tension to strictly homogeneous membranes. Bonnafont ('Maladies de l'Oreille,' p. 5) is of opinion that the anterior portion of the membrane responds to higher sounds, and the posterior to lower ones; and that the tensor tympani muscle gives tension to the former, while the stapedius gives tension to the latter; these muscles being therefore destined to regulate the reception, the tensor tympani of acute, the stapedius of grave sounds. My own observation accords with Bonnafont's statement that the posterior portion of the membrane is put on the stretch by traction on the tensor tympani, and the anterior part to a slight extent by traction on the stapedius. But I have not yet been able to confirm his view that loss of the anterior part of the membrane impairs especially the hearing of grave sounds and loss of the posterior part that of high ones.

[3] Loc. cit.

cumference. Here they communicate with the minute internal branches which run in the mucous layer, continuous with the vessels of the tympanum. A few nervous twigs have been traced into the dermoid layer from the N. temporalis superficialis; and some small grey fibres have also been detected by Gerlach in the mucous layer.[1] The membrane is thus both vascular and sensitive; in the healthy state, however, vessels are seldom visible, but if any irritation is present, or sometimes without apparent cause, a red line of vascularity is to be seen extending down from the roof of the meatus along the handle of the malleus. Bonnafont also has noticed that very acute sounds will cause a visible redness of the membrane. When the congestion reaches a higher degree, a ring of vessels becomes visible also near to the circumference, radiating branches uniting it with the vessels around the malleus. (See Fig. 11.)

It is to be remembered, however, that lines of vascularity may be produced by the irritation occasioned by the speculum if kept long in the meatus, and thus may be present towards the end of an examination though absent at its commencement.

In the healthy state the membrane presents a surface of a peculiar glistening transparency, obliquely placed, and of an obvious concavity. Von Troeltsch[2] estimates the angle formed by the membrane with the upper wall of the tympanum at 140° on an average, though it varies much in different individuals. He thinks that this angle bears a constant relation to certain other characters of the cranial development. In an adult cretin he found it as much as 167°, approximating thus to the almost horizontal position of the membrane in the infant. In an examination, however, which by the kindness of Dr. Down I was allowed to make of the ears of the inmates of the Earlswood Asylum for Idiots, I did not notice any instance of unusual obliquity of the membrane.

The points which specially attract the eye and should be first sought out as the starting point of the examination are—(1) the handle of the malleus, and (2) the bright spot. The malleus commences superiorly with the white and prominent short process, and runs downwards and generally somewhat backwards, as

[1] Politzer, 'Beleuchtungsbilder des Trommelfells,' p. 12.
[2] Op. cit., p. 23.

a broad white line, terminating near—but a little above and in front of—the centre of the membrane at its most concave point (the umbo). The bright spot extends in a triangular form from near the termination of the handle of the malleus downwards and forwards. The nature of this bright spot has been first thoroughly explained by Politzer.[1]

"If," he says, "the membrane were a plane surface, no *light-reflex* would be present, inasmuch as from its inclination to the meatus all the light cast upon it would (by the laws of the reflection of light) be reflected upon the anterior and inferior wall of the meatus. But in consequence of the curving of the membrane inwards, by the tension of the malleus, there arises a change in its inclination, of such a kind that its anterior part is placed directly opposite to our optic axis, and the light thrown on this part is accordingly reflected direct to our eye." The process may be rendered visible by stretching a membrane and holding it in the position of the membrana tympani, when no bright spot is seen, but if it be rendered concave by a rod attached to it in the position of the malleus, a bright spot (light-reflex) appears exactly as in the natural membrane.

The extent and form of this bright spot is somewhat variable. Even in healthy membranes it may or may not reach to the circumference, it may be a single broad streak of light, or may be divided across or lengthwise. If the membrane moves outward, as when the tympanum is inflated, or inward as during the act of swallowing, the motion is generally most delicately marked by the changes in the appearance of the bright spot. In diseased conditions it may be wanting altogether, or from variations in its curvature its position may be changed, or several may be present in different parts of the membrane.

The colour of the healthy membrane cannot be exactly described, because it varies with several circumstances which Politzer has pointed out. By virtue of its transparency its hue is modified by the rays of light reflected from the promontory, so that its own colour cannot be exactly estimated. The kind of light employed also affects it; it is more blue by daylight, more yellowish by artificial illumination.[2]

[1] Op. cit., p. 23.

[2] All the drawings appended were made by natural light. The direct rays of the sun were used, whenever practicable, to give distinctness to the view, but the

Fig. 1 represents a healthy membrane drawn shortly after its removal from the body. The blue tinge seems to me to depend upon the dark cavity which exists behind the membrane, and to be analogous to the blue of space. When the internal wall of the tympanum is red, a pinkish tinge may be imparted to the membrane by the light transmitted through it from within. In children it is generally less bright and transparent than in the adult.

The other plates represent some of the more frequent deviations from the healthy appearance of the membrane :—thickening and opacity (No. 2); chalky and other deposits (3, 4 and 5); thinning and collapse of the membrane (5, 6, 8); granulations upon its surface (9); the effects of hereditary and of secondary syphilis (2 and 10). Lastly, a series of them represent various stages and forms of perforation, and the process of healing (11—24.)

There are many other morbid conditions of the membrane besides these, and the present paper constitutes accordingly, but an instalment towards a description of its diseases. It is necessary only to premise a few words on the mode of examining the ear.

For the inspection of the membrane at once the simplest and the most effective method is that lately reintroduced by Von Troeltsch of reflecting the daylight by means of a mirror into the speculum. The reflectors commonly used for examining the larynx or the retina serve well. The patient being placed before a window, the surgeon stands somewhat sideways to the light which is easily reflected into the meatus; daylight moderately clear being amply sufficient. One great advantage of this method is that the reflector may be worn on the forehead, and both hands thus left at liberty.

Inflation of the tympanum with air will be frequently mentioned in the following remarks. In every case, unless the use of the catheter is expressly mentioned, this was done on the method introduced by Politzer. It consists in passing a stream of air with moderate force into the pharynx, through the nose, at the moment when the patient swallows; advantage being thus taken of the opening of the Eustachian tube by the tensor

appearance represented is that obtained by the reflection upon the membrane of diffused daylight.

palati which (as shown by Mr. Toynbee) takes place during that act.[1] It is a simple and easy procedure, in very many instances an excellent substitute for catheterism. Politzer used an india-rubber bag provided with a flexible nozzle, which was placed about half an inch within one nostril of the patient and the nose closed over it. The patient was told to swallow at a given signal (holding a little water in the mouth for the purpose), and when he swallowed the bag was compressed, the air generally rushing up the Eustachian tube even in spite of very considerable obstructions. Air may be blown in like manner through a simple india-rubber tube, or very effectively injected by means of the two elastic balls introduced by Dr. A. Clark for the atomizer. My own preference, however, is for blowing by the mouth which may be very exactly regulated and does not introduce cold air.

Perforation of the Membrana Tympani.

This is a very frequent occurrence. Among 1088 cases of aural disease attended at Guy's Hospital in the two years from April 1863, to April 1865, 173 or one in six were cases of this affection. It is met with by far most frequently in children, probably because the diseases of childhood are its most frequent causes. Among these causes scarlatina, as is well known, stands at the head; next, in my experience, comes the common catarrhal inflammation described by the patient as "a cold," with which may probably be included the so-called "teething" discharges of infants, a by no means unfrequent cause of destruction of the membrane; third, "fever;" then measles and hooping cough. Not unfrequently perforation is ascribed to chickenpox, which in this respect would seem to be a more formidable malady than has been supposed.[2] Ten were referred to accidents, five to blows, one to a fall, four to scalds. The penetration of sharp bodies, such as knitting-needles introduced to remove wax or allay itching is also a frequent cause of perforation.

[1] Von Troeltsch has inferred from numerous dissections that the tensor palati alone opens the tube, and that the levator acts, though less powerfully, as a constrictor ('Arch. der Ohrenheilkunde,' vol. i, p. 15).

[2] The statements of patients are too incomplete to render the statistics of the diseases producing perforation of any value.

The foregoing numbers, however, give no true indication of the frequency of the lesion, since they do not include those patients in whom the perforation was already healed at the time of their first application; a number as we shall see in the sequel in all probability very considerable. It cannot, however, at present be estimated with certainty.

In the great majority of cases perforation occurs from an inflammatory affection extending to the membrane either from the meatus or from the tympanic cavity. That it may result from inflammation commencing primarily in the membrane itself is probable, but I believe it has not yet been clearly traced. Inflammation of the tympanum is the most frequent cause, and an observation I had an opportunity of making leads me to think that it occurs in this way more frequently than is imagined.

In May 1864, a girl, æt. 10,, of good constitution, came under my care on account of an intermittent albuminuria that had continued since scarlatina three years previously. The affection was slight, and the health seemed not much affected. Twelve months before there had been, during a cold, an attack of pain, lasting a few hours, in the left ear, which was then examined and seen to have at first a flat and slightly dulled aspect; afterwards red vessels were visible on its surface, both radiating and around the circumference, and it presented also a peculiar appearance chiefly at its upper part, being marked by faint oval outlines, which appeared to me to arise from bubbles in contact with its inner surface; an appearance I have observed in the ears of children three or four times, and I think there can be no doubt as to its origin in the mixture of air and fluid within the tympanum. The hearing was much dulled. In another week the membrane had become less vascular, but it was decidedly concave, the stapes being plainly visible and apparently in contact with its inner surface. The tympanum was gently inflated twice. The membrane lost its vascularity, and the hearing in a few days became good but not perfect (two feet for a watch heard on the right side at six feet.) Just twelve months after, on May 17th, she woke again in the night with pain in the left ear, followed the next day by a watery discharge. There was a little fever, and the hearing was reduced to ten inches on the right side, and four on the left. The left membrane was white and thick. A saline powder was ordered, and a lotion of three grains of borax. Four days after, the ears being quite easy, there was syringed from the left meatus, together with some milky fluid and a few flakes of epidermis, a small mass of soft shreddy discharge, and a circular orifice about the size of a large pin's head was seen at the lower part of the dull white membrane. The next day the orifice was smaller and of an oval form; four days after, it was scarcely to be detected as an orifice, but appeared as a small dark point with somewhat thickened white edges, but air passed through it with a slight dry sound. In three days more no trace of it was visible, and air inblown entered the tympanum with a hollow puff, not escaping. A slight attack of sickness with a little return of the albuminuria now occurred, but soon passed off. The right ear in this instance was involved as well as the left, being indeed the more deaf of the two, and the membrane becoming for a time uneven and of a dull, dark, grey hue, with a slight tinge of pink.

It seems probable that this is an example of a large number of cases which pass almost or entirely unobserved. There was nothing in the symptoms indicative of anything more than an ordinary slight discharge from the ear. The recovery seemed perfect, though the hearing (two years after) is slightly impaired; but it is not impossible that the affection was essentially the same as that insidious and painless process of destruction, which in cachectic, and especially in tuberculous children, leads to an extensive or total loss of the membrane; while the exposed tympanic mucous membrane becomes red and spongy, and the walls of the meatus, often excoriated by the acrid discharge, are swollen and tender, and either brightly pink or livid with congestion, or covered with thick white layers of softened and sodden epidermis. It is interesting to note the presence of an excess of fluid in the tympanum twelve months before the occurrence of the perforation.

As before observed, a certain proportion of the discharges from the ear which occur within the first three or four years of life, and are ascribed to teething, are attended with more or less destruction of the membrane. Whether in these cases the inflammation originates in the meatus or in the tympanum there is no sufficient evidence. I have had the opportunity of examining after death only one such case, a miserable, wasted, syphilitic child eight months old, in whom a purulent discharge from the ears had existed for upwards of three months. In this case both meatus were tolerably healthy, but the tympana were filled with a dark sanguinolent grumous fluid, the membranes almost black, partly from congestion, partly through discoloration from the fluid behind. On each side there was a perforation about a line and a half in diameter at the lower and anterior part; but firm flakes of epidermis covered the entire surface of each membrane and must have hindered the escape of the matter. The tympanic portion of the Eustachian tubes appeared healthy and contained no fluid. The bones were not diseased. Judging from this case, and from the extreme frequency with which accumulations of fluid within the tympanum and congestion of its lining membrane are met with in the post-mortem examinations of children who die from whatever cause,[1] it would seem probable that these "teething" discharges from the ear are mostly of tympanic origin. On the other hand the frequency with which they pass, leaving no discernible consequences (though this, as we have seen, is no proof that the membrane has not given way), and the absence of any detectible sign of disease within the tympanum would lead us rather to suppose that most frequently the meatus alone is implicated. But it is again to be remembered that a discharge from the meatus with unruptured membrane is often an accompaniment of catarrhal disease within the tympanum. On the whole, considering the rapidity with which perforations may heal and the infrequency with which examination of the ear is made in such cases, it must at present remain undecided whether the infantile "otorrhœa" be generally of external or tympanic origin, and also why in some cases, without recognised difference from the cases that recover, it should permanently injure the organ.

Fig. 11 exhibits the appearance of the membrane in a case of perforation, commencing, probably, from without. The history illustrates also the rapidity with which changes occur

[1] I have for several years endeavoured to examine the petrous bone in every case I have met with of death during early life, and I can testify that it is decidedly the exception to find the tympanum healthy. It is, in the majority of cases, the seat of congestion and of excessive secretion. Cases of this kind were reported by me in the 'Med. Chir. Trans.' for 1856. Dr. Von Troeltsch and others have reported a similar result.

in that highly vascular structure. The patient was a healthy girl, aged eight years, who had suffered for four years with repeated attacks of discharge from the right ear, often attended with pain, and brought on first by chewed paper put into the ear by a companion. A watch audible at five or six feet normally was heard at four inches only. On examination the meatus was found somewhat red, swollen, and covered with discharge; on the membrane which was of a dull grey hue with a a purplish tint, besides a general congestion of its vessels, there was, just beneath the end of the malleus, a small red surface, bounded below and in front by a whiter portion of membrane, apparently a thickened patch of epidermis. This red surface appeared raw and slightly depressed, and had the aspect of a superficial ulceration. The membrane was entire as proved by the fact that air passed into the tympanum, through the Eustachian tube, moved the membrane forward but did not pass through it. On the next day, at the anterior extremity of the red patch, a small darker spot was seen, visibly pulsating, and through which, on the inflation of the tympanum, air passed outwards with the usual slight screeching sound. No fluid, however, escaped with the air.

When next seen, eleven days after, the red surface was no longer to be distinguished, the perforation had healed, a slight depressed point alone marking its position; the general aspect of the membrane was much more transparent than before, and less vascular, but it looked too concave and somewhat irregular. It moved outward on inflation, by which the hearing distance also was improved.[1]

That the perforation, in this instance, took place from without I infer partly from the appearance of the membrane, strikingly contrasted as it is with that exhibited in Fig. 12, which represents its aspect in cases of chronic suppuration within the tympanum in early life; partly from the inflammation of the

[1] In every case in which, in this paper, healing of a perforation is affirmed, it is implied that the fact was proved by the introduction of air into the tympanum, the only test on which reliance can be placed. It is Politzer's opinion, however, that the mere contact of the edges of a perforation, when there is no loss of substance, may suffice to prevent the escape of air blown with moderate force into the tympanum, and to allow the membrane to bulge. This view is based upon the rapidity with which such small apertures appear to close and again break open.

meatus preceding the perforation, partly from the red depressed (ulcerated) surface within which the perforation formed, and partly from the absence of any considerable escape of fluid when air was blown through it from within. The speedy healing in a case so chronic confirms this view, since such rapid healing of perforations arising from catarrhal inflammation within the tympanum, so far as I have observed, occurs mostly, if not only, in acute cases.

The patient, who is still under treatment, continues to progress well; the hearing having much improved and the membrane become free from opaeity. The position which was occupied by the red surface is now clearly traceable as a thinner slightly depressed portion of the membrane, distinguished from the rest by its greater transparency and darker hue. No doubt therefore can exist that it was an ulcer of the membrane penetrating at one spot, and healing with loss of the external layers. By the eye, this surface is indistinguishable from the thin scar of a healed perforation, but it appears to yield less on inflation of the tympanum.

Fig. 12 illustrates the occurrence of perforation from chronic purulent inflammation within the tympanum. It exhibits, in a highly developed form, the aspect given to the membrane in childhood by the accumulation of puriform fluid behind it, with thickening of its mucous layer, and congestion and extravasation of blood in the same layer, seen through the transparent and scarcely altered more external laminæ, as through a thin, slightly hazy glass.[1]

In this instance, also, there was presented a condition I have never observed before, and have not seen noticed, viz., a distinct *slough* of the membrane. This slough was of a characteristic aspect, dark, dirty grey, softened and with a distinct line of separation round it, a minute perforation having already formed at its anterior border. It occupied nearly the centre of the membrane for about two lines by one and a half, immediately below the malleus. It is possible that a proper sloughing of the membrane occurs not unfrequently, especially in cachectic

[1] The appearance is very characteristic, but, as seen during life, it is often somewhat dulled by the presence of a softened and imperfectly transparent layer of epidermis. This layer, which was, as usual, thin and soft, had separated in the specimen from which the drawing was taken.

subjects, or in the severer cases of scarlatina. It may have escaped observation hitherto from the presence of other grave disease serving to divert attention from the ear. The specimen from which the drawing was taken was removed from an idiot boy, aged fourteen, who died of phthisis, and in whom no affection of the ear had been observed. The tympanic cavity was full of purulent fluid; the mucous membrane thick and spongy; the Eustachian tube, though its lining membrane was healthy, contained no fluid—doubtless in consequence of the closure of its tympanic orifice by the tumid lining of the tympanum.

Perforations arising from mucous accumulation or suppuration within the tympanum most frequently appear to take place and to extend by a process of ulceration, the diseased and softened texture wasting insensibly away. A general bulging of the membrane or distinct pointing of matter behind it is, I believe, rare. Minute abscesses form in the substance of the membrane itself, distinguishable as small yellow prominences with more or less vascularity around. These break and leave for a time slight depressions, without perforation. I have seen such abscesses follow hooping-cough, and accompany chronic inflammation of the meatus. But they have no constant connection with tympanic suppuration. A distinct bulging of the membrane I have not myself seen. Politzer, however, says[1] he has often seen general or partial bulging, with redness of the surface; but it seems questionable to me, even from his own description, whether this appearance is really that of bulging, since he states that the same appearances occur in accumulations within the tympanum, and in inflammations of the dermis of the membrane. He has frequently observed, however, before perforation, in acute cases, greenish-yellow ill-defined patches on the membrane, apparently from exudation compressing the vessels, and in one case he saw perforation ensue in the position of one of these patches. Dr. Moos mentions that he has seen the *upper* part of the membrane bulge in a case of acute catarrh of the tympanum, and the lower part in one of bleeding into the tympanum, during the course of purpura.[2] But though bulging is rare the membrane is often seen much flatter than is natural, and with a tense and rigid aspect. This condition, however,

[1] Op. cit., p. 47.

[2] 'Klinik der Ohrenkrankheiten,' p. 139.

by no means always coincides with the presence of fluid in the tympanum. Possibly it implies a previous *vis a tergo,* diminishing the natural concavity of the membrane, which then has grown rigid in its new position. But on this point observations are still wanting.

That peculiar hue of the membrane in which the fluid contents of the tympanum show through the still transparent laminæ, is seldom seen except in children. In catarrhal inflammation of the tympanum in adults, the epidermis, if not the other layers also, soon becomes thickened and opaque, and the membrane presents a general dull grey hue, varying from nearly white to a greenish yellow, or with a decided purplish tinge, with more or less redness along the malleus. If perforation has taken place, or the meatus participates in the inflammation, all the parts are bathed in a watery or purulent discharge, or covered with loosened flakes of epidermis, which often so much resemble the diseased membrane in appearance that it is only by estimating their position, or carefully testing by the syringe or the probe, that their nature can be determined. The membrane also may be variously excoriated, or covered with granulations, or wholly or partially thinned and fallen in, to which conditions allusion will be made hereafter.

In regard to the position and form of perforations, Sir Wm, Wilde, and after him Moos, affirm that they are most frequent in the anterior and lower part of the membrane, where the air blown through the Eustachian tube impinges. Politzer speaks doubtfully on this point, and my own experience has presented me with fully as many in the inferior and posterior segments. They seldom extend quite to the periphery, and are rare in the upper part of the membrane. I have seen none confined to, or commencing in, the upper and anterior part, in front of the short process of the malleus; but they occur sometimes in the corresponding region behind, where thin, relaxed, and irregularly sunk-in portions of membrane, probably scars, are also not unfrequent. Once I observed, in an adult of weak constitution, a small perforation immediately above the short process, which after closing for a short time opened again, and after some months nearly the whole membrane was found to have been destroyed. In this case there was a very large formation of thick soft masses of epidermis, which accumulated at the inner extremity of the meatus.

When placed anteriorly perforations may be hidden by the prominent anterior wall of the meatus, and only demonstrable by the passage of air through them. And this may also be the case when situated in visible parts of the membrane if they are very small, or the surrounding membrane is tumid or granular, or they are overlapped by polypi, which very frequently accompany them, or even without any of these conditions, since it is sometimes impossible for the eye alone to distinguish whether the surface which meets it at the bottom of a depression in the membrana tympani is the internal wall of the tympanum, or a scar or granular surface more or less nearly in contact with it. Indeed, Politzer affirms that the swollen mucous membrane of the promontory will sometimes bulge before air blown into the tympanum, even when the membrane is wanting, and that treatment alone can reveal the true nature of the case.

When small, if the membrane is moist, the position of a perforation is very often denoted by a fluid surface, brightly reflecting the light, and pulsating with the beat of the heart. Sir Wm. Wilde, who first drew attention to this appearance, considered it pathognomonic of perforation; but it is not strictly so. Though seldom, cases do occur in which small pulsating spots are seen on unperforated membranes, most often when there are granular dots upon its surface. The motion is evidently imparted by the blood, and implies, not necessarily an aperture, but a thin surface of fluid in contact with a beating vessel.

In size and shape perforations present every variety. An almost total loss of the membrane (Figs. 22, 23) is one of the most frequent forms. (Moos found it second in frequency.) But however complete the destruction, there is (I believe always) a certain portion of the membrane, though it may be extremely narrow, left at the circumference. Sometimes this remaining portion is thick, red, and of a fleshy look, only distinguishable, it may be, from the reddened meatus by its position; at others it is thin and white (see Fig. 24); and by a continuance of treatment, and the use of suitable astringents, it may often be restored from the former to the latter condition. Between these cases and those of simple pin-hole perforations (Fig. 20) there is every gradation of size, the shape being determined very much by the natural divisions of the membrane, the handle of the malleus often forming a kind of boundary line: frequently the lower portion of the handle is embraced, as it were, by a kidney-shaped orifice. Two perforations divided by a bridge of membrane are sometimes seen.

When the membrane is largely destroyed, the characteristic appearances of the exposed internal tympanic wall can generally be clearly distinguished; anteriorly the fossa leading to the Eustachian tube; in the centre the projection of the promontory, and behind it the rounded cave-like depression of the fenestra rotunda; above this the stapes, sometimes with, but more generally without, the long process of the incus attached. (See Figs. 23 and 24.) Even in the case of comparatively small perforations, the handle of the malleus is generally found more or less sunk inwards, the short process being prominent in excess, and the lower end directed strongly towards the promontory, sometimes touching it, sometimes adherent by firm tissue; but when the aperture is of large size the ossicula seldom retain their integrity. The incus is mostly either wanting or its long process more or less destroyed; and unless a strip of membrane is preserved on either side of the handle of the malleus, this process also, I believe invariably, after a time, disappears. Fig. 23 represents the ear of a girl æt. nine; discharge had been present since the age of three, coming on from exposure to cold, shortly after chicken-pox. There was also at first a great discharge from the nose. Twelve months before I saw her the discharge had ceased for a month, after treatment in Paris, but had recurred, and was very profuse and offensive. There was removed from the ear a large quantity of black grumous matter, evidently deposited in consequence of the use of a lotion of green tea, which had been carried on for several months. The membrane was entirely gone, with the exception of a narrow peripheral margin, the malleus, however, being entire, and in contact with the promontory. The mucous membrane of the tympanum was of a livid red, thick and spongy, the fenestra rotunda not distinguishable, the head of the stapes just appearing. The local treatment consisted in the application of finely powered *talc*, mixed with a small quantity of morphia, which was gently blown into the ear, to cover all the exposed portion, every third day for three weeks, the powder being carefully removed by syringing before it was reapplied. Iodine was applied externally, and the ear was occasionally washed out by a weak solution of sulphate of zinc or nitrate of silver, air being also regularly passed through the Eustachian tube. Under this treatment the spongy tympanic membrane assumed a healthy pale and dry appearance, and the discharge ceased. The hearing, which was at first four inches from the watch, continued about the same, but it was greatly improved by the artificial membrane, the use of which, however, was deferred.

In this case the destruction of the membrane had only recently become complete. Twelve months previously the orifice had been comparatively small; and the continued use of the green tea infusion probably contributed to enlarge it by causing an accumulation of irritating matter. Hence it is that the handle of the malleus, although entirely denuded of membrane, was perfectly preserved. It showed no slgns of decay during the period of treatment: but in all probability will disappear in the course of time. In the case of a man who fell, while intoxicated, into a brewing vat of hot fluid, and in whom the entire membrane was destroyed by the scald, the malleus remained for a time in a similar isolated condition, retaining, also, its normal position; but, after the lapse of a few weeks, it had disappeared. In Fig. 24 this process is seen completed, and the condition to which an almost total loss of the membrane tends is shown. It represents the left ear of a girl, aged 18, who had suffered from frequent discharge since childhood; but referred her deafness only to three years back. The cause could not be ascertained. The membrane was entirely wanting except a slight ring round the margin; nothing was left of the malleus below the short process, which, also, was somewhat wasted. The depression of the fenestra rotunda was clearly seen, as also were the head and posterior crus of the stapes. The tympanic mucous membrane was dry, though somewhat thickened, and the Eustachian tube pervious.

The hearing in this case was almost annihilated, evidently from disease which implicated the labyrinth, and the artificial membrane did not produce benefit. On the opposite side a smaller perforation exists, with less destruction of the hearing power, and the artificial membrane is of some advantage.

Left to themselves perforations appear frequently to heal. I have seen several instances in patients who have been under my observation at distant intervals. The fact is evidenced also by the large number of persons who present themselves for the cure of deafness, in whom the membrana tympani presents appearances which exactly correspond with those known to belong to scars, and whose history indicates a previous perforation. It is impossible, however, to say in what proportion of cases spontaneous healing occurs; or how much destruction of the membrane usually precedes it; and the more because there sometimes occurs in chronic tympanic affections a peculiar atrophy of the membrane, leading to appearances not distinguishable by any known marks from those of scars; so that, in almost every case of apparent scar, unless the history is known, it is impossible to say whether perforation has existed.

In a large number of cases, however, whether the loss of substance have been large or small, healing does not take place. An irritable condition, with constant or intermittent discharge, may continue indefinitely (it is no very uncommon thing to meet with it of more than thirty years' duration); or the secretion may cease, and the exposed tympanic wall become dry, often smooth and pale, the remaining portion of the membrane being generally of an opaque white aspect, and flattened, or irregular, or altogether fallen in. Less frequently it is thinned, unnaturally transparent and glistening, presenting a surface a good deal like that of a thin sheet of mica, the malleus being often quite indistinguishable, even when the orifice is but small. Sometimes, and especially in cases in which the Eustachian tube is obstructed, the membrane appears solid, presenting a dry, smooth, and uneven pinkish surface, the orifice having a sharp round edge, as if cut out with a die.

Fig. 13 exhibits the usual appearance of a perforated membrane when the discharge has ceased and the parts have assumed a quiet state:—the white flat membrane, the malleus obscurely marked, but its position indicated by a slight fringe of vascularity, the edges of the perforation thin, the membrane ceasing as it were abruptly. The history of the case is interesting, as illustrating the very frequent occurrence of a nervous lesion with a previous structural disease. The patient was a woman aged eighteen, who had married at sixteen, and had a child the subsequent year, which she suckled for nine months, appearing to be in perfect health. About the end of the ninth month she became suddenly totally deaf. She was sitting at rest, when there came, without warning, a rushing sound as of rain in her ears, and she found she could not hear. For a day or two there was a slight improvement, but it did not last. Her health has not suffered; but a tinnitus (described as extremely severe) has continued ever since, and she has been subject to occasional giddiness. She continued to suckle for two months, when she applied to me, just able to hear a few loud and piercing sounds. There was no loss of muscular power or control, the eyes were normal except a high degree of hypermetropia; the other functions perfect; urine free from albumen and sugar; no relatives had been deaf. On examination, it appeared that she had been somewhat deaf at times ever since scarlatina, which occurred when she was a child, and was followed by a discharge from both ears, which, however, had long ceased. The right membrane presented the appearance shown in Fig. 13; the left was thin and somewhat sunk in, with a small white patch, apparently a scar, anteriorly. Air was easily passed, but without effect upon the hearing, into the left tympanum, and *through* the right. Treatment, which comprised a variety of tonic measures, suckling being discontinued, was of no avail. The precise cause of the sudden loss of hearing in this case is a matter of conjecture. It evidently links itself with those forms of *paresis* which Dr. Handfield Jones has done so much to elucidate; and the loss of function may reasonably be referred to the early pregnancy and lactation; the general exhaustion expressing itself in the failure of a weakened organ. In this respect the case seems to me to be typical of a large number of cases in which the function of the auditory nerve is impaired; examination detecting also proofs of local injury.

The appearance of the border of the perforation in this instance is characteristic of the absence of a tendency to heal. In such cases the edges of the wound are, in fact, cicatrized, and though sometimes they may be induced to heal by the application of caustics, these do not usually succeed. Moos reports a case in which an old perforation, involving most of the posterior portion of the membrane and probably of twelve years' standing, healed in eighteen days after the access of an attack of inflammation in the remaining portion of the membrane, a slight improvement of hearing ensuing. Mr. Harvey also mentions the case of a man in whom inflammation of the tympanum, with abscess at the back of the pharynx, followed syringing, a perforation which had existed many years closing up thereafter.[1] The appearance of a perforation which is beginning to heal is shown in Fig. 14. It is characterised by a slight pale swelling of the edges of the orifice, contrasted alike with the smooth thinness of a perforation which has cicatrized unhealed, and with the red and excavated border, or granular and spongy tumidity, of one in which inflammation still exists, or ulceration is progressing.

Figs. 14 to 17 exhibit the process of healing of a perforation in successive stages. The patient was a youth, aged fourteen, delicate, and with the tonsils so much enlarged as nearly to meet. He applied to me in October, 1865, for a painful discharge from the right ear, in which, for two or three years, he had been subject, during frequent colds, to deafness. About six weeks ago while at the seaside there had come on in it first a buzzing noise, and then a slight aching, followed by a free discharge with a little bleeding. Three weeks ago he had brought up from his throat a portion of the husk of a grain of oats, which he had swallowed a few days after the discharge commenced. It was now expected that the ear would recover, but it did not. Has lately had a series of boils upon the neck. Watch heard, right ear, on contact; left ear, which is of healthy appearance, twenty-four inches. The right membrana tympani was anteriorly thick, and of a dull grey hue, uneven, and the malleus hidden; posteriorly, close to the meatus was a small mass of fungoid granulations, and just in front of these, and partly hidden by them, an orifice in the membrane the size of which could not be determined on account of the thickened and granular condition of the surrounding parts. He could not inflate the tympanum, but air inblown passed readily through the perforation, bringiug with it bubbles and mucus, and improving the hearing to two inches. The treatment adopted consisted of quinine and iron internally; nitrate of silver, and afterwards tannin, to the tonsils, and finally a solution of alum by means of Dr. A. Clarke's atomizer; moderate stimu-

[1] 'The Ear in Health and Disease,' p. 57. In this case, however, it might be doubted, from the history given, whether a previously healed perforation had not been freshly ruptured.

lus in the form of light claret; under which the general condition and the throat gradually improved. For the ear various cleansing and astringent lotions were applied; carbolic acid, nitrate of silver, sulphate of zinc, borax and opium; the granulations were touched two or three times with caustics, and air was once or twice a week blown through the tympanum to clear it, while the meatus was filled with an astringent lotion. Improvement to a certain extent soon followed, the granulations disappeared and the orifice contracted, but it did not heal and showed a continually returning tendency to increase again, and to re-assume an irritable character, the fungoid condition of the meatus recurring more than once after the caustic applications had removed it.

On the 26th of January, upon examining the ear after it had been apparently thoroughly cleansed not only by syringing, but by the use of a lotion of sulphate of zinc and by inflation, I was struck by the continued presence of viscid mucus within the orifice. To remove it I placed the patient in a recumbent position, filled the meatus with a warm solution of sulphate of zinc (gr. iv ad ℥j), and, causing him to swallow some water, passed air into the tympanum (on Politzer's plan) causing it to bubble up freely through the lotion in the meatus. Then on syringing the ear, a mass of coagulated mucus the size of a grain of wheat was removed; but still, upon examining, more viscid secretion was perceived within the orifice. The same process was accordingly repeated, and with the same result, *six times*; until, at last, on so washing out the tympanum nothing was removed. The effect was decided; the hearing rose to eight inches, and from that time the perforation progressed uninterruptedly to a perfect healing, the ear being washed out daily in a similar manner by the patient. Fig. 14 shows the appearance of the membrane ten days after the removal of the mucus; it presents, I believe, the characteristic aspect of a healing perforation as described above. The fungoid condition had entirely disappeared; the internal wall of the tympanum, just discernible, was of a red colour and moist; there was no vascularity of the membrana tympani, but its external layers were opaque and thickened, hiding the handle of the malleus entirely, and presenting an uneven flattened surface instead of the usual delicate concavity. Fig. 15 shows the membrane four days later. The orifice had already greatly contracted, and the membrane had become much clearer, so that the malleus could be traced. Posteriorly the epidermis had become thickened, and overlapped the posterior border of the orifice (at a later period a thick hard mass of epidermis was removed from this spot). There was very little discharge. The attempt to blow air through the tympanum was gradually discontinued, and ten days afterwards the orifice was entirely healed, presenting the aspect of a thinned and slightly depressed surface, as in Fig. 16. This scar gradually contracted, the edges becoming slightly thickened in the process, giving it a crater-like appearance, as seen in Fig. 17, taken at the end of three months, in May, 1866. When last seen, July 21st, the appearance was that of a minute dark speck surrounded by a comparatively broad white rim, and on using a lens the dark central spot was seen to be a very thin convex portion of membrane surrounded by a raised and thickened ring. The thinned portion, the new scar tissue, bulged slightly when the tympanum was inflated. The hearing was, for the watch, five inches (after inflation 9); the voice was heard moderately well, though not acutely. The hearing had therefore very considerably improved; but during the early stages of the healing it had for a time diminished. On the left side the watch was heard at fifty inches, or double what it had been.

This case leads to the consideration of the treatment of perforations, whether recent or chronic, in which discharge is present. The result of my experience is that the chief object should be to remove entirely the morbid secretion accumulated within the tympanum, and to keep the cavity cleansed. Wounds of the membrana tympani appear to have as strong a tendency to heal as wounds of any other part; nor is there any lack of power to supply, by an efficient substitute, even the most considerable losses of substance. But when the tympanum becomes congested the swelling of its mucous lining soon closes the Eustachian tube, and its secretions having no other exit than through the membrane, perforations are continually kept open, or, after closing, are reopened, by the pressure of discharge within. It is evident, also, how apt the tenacious mucus which the congested membrane pours forth must be to cling about the recesses of so irregular a cavity, and by its very presence to maintain the irritation which led to its production; or if the tendency to an excess of secretion within the tympanum should cease, as it often does, spontaneously, the mucus already collected in the tympanum most probably dries up into dense and rigid layers, agglutinating together the ossicula, the membrane and the tympanic walls, and destroying the capacity of the organ to respond to sound. That such drying up of mucus does take place in the tympanum during life, I am strongly persuaded alike from the examination of *post-mortem* specimens, and from the observation of disease, especially of cases of long-standing perforation. In these the assiduous employment of lotions, caused by inflation of the tympanum to enter all its crevices, will often bring about the softening, and allow the removal by syringing, of large masses of semi-solidified mucus, of the presence of which the eye could discern no trace. Such mucus is often collected in the upper part of the cavity of the tympanum (probably surrounding the articulation of the malleus and incus), and from thence large masses may with patience be removed. It may be seen sometimes gradually descending below the upper border of the perforation, when the membrane is largely destroyed, until at last it becomes loose and separates, or may be watched unwinding itself, as it were, from around the buried head of the stapes. In some recent cases, too, of destruction of the membrane (as in that from which Fig. 23 was taken), fluid mucus may be seen trickling down from this hidden part of the tympanum over the exposed and healthy promontory; nor can the treatment be considered complete until this part also is cleansed and restored to health. An envelopment of hard dry mucus around the heads of the malleus and incus appears to me a not improbable cause of some obscure cases of deafness, and may perhaps help to account for the occasional benefit obtained by injection of liquids into the tympanic cavity.[1] Dr. Jago speaks of the drying up of mucus within the tympanum, after an attack of common catarrh, as a matter of his own

[1] This appears to be a perfectly safe procedure so far as any serious results are concerned, though I have known a healthy ear weakened for a long time by the injection of warm water into the tympanum. It is easily accomplished through the catheter, and is extensively used and recommended by many competent men in Germany. See the writings of Politzer, Moos, Lucae, Schwartze, &c. Von Troeltsch, who was opposed to it, has recently spoken of it more favorably. That it does good in some cases is certain, whether in any cases not otherwise to be benefited seems to me not yet proved.

personal experience;[1] and a case at present under my care appears to be an instance of it. A boy, aged seven, healthy, but beginning to practice self-abuse, and somewhat weak, had had an offensive discharge from the left ear for three years, ascribed to measles. There was a perforation, about a line and a half in diameter, near the anterior border of the membrane, with a small polypoid growth (as is frequently the case) springing from the meatus close by. He could blow through the orifice; the discharge was milky, mingled with flakes of epidermis. Watch heard one inch. Carb. iron was given, the polypus touched with nitrate of silver, and a lotion of sulphate of zinc gr. iij morphia gr. j to the ounce, freely used. The perforation soon became smaller, and the hearing improved. In one month the discharge had ceased; air was still blown through the membrane; but the orifice was a mere point, scarcely visible. The membrane had a healthy look; watch heard six inches. Hoping that healing was on the point of taking place I did not advise a speedy return, and six weeks passed before the patient was seen again. The perforation was then much larger, again about a line and a half in breadth; and it was situated within a red, depressed, rough surface, of oval shape, and about twice its own size—apparently a superficial ulceration. Hearing diminished to three inches. There had been no discharge, and though he blew through the membrane none escaped, nor did any come away on syringing. The ear was then freely washed several times, in the manner before described, with a solution of sulphate of zinc (gr. iij ℥j), and at length air would no longer pass. On examination a white mass was found occupying the orifice, which neither the most forcible inflation nor the syringe would remove. A small pair of ring forceps being introduced, a dense pellet of thickened mucus was drawn out of the cavity of the tympanum, and on repeating the process a second also was removed of a like kind, again requiring the forceps to bring it through the perforation. The hearing improved, and the orifice assumed a healthy aspect; but though the membrane appeared clear, and the ear perfectly dry, a free washing with the lotion on two subsequent occasions brought out of the tympanum shreds of dense mucus, which closed up the orifice while they traversed it.

[1] 'The closure of the Eustachian Tube,' &c., 1854.

For the suggestion of this method of treating perforations of the membrane we are indebted, as for so many other resources in aural surgery, to Dr. Politzer. In adding my testimony to its efficiency I would merely insist upon the necessity of carrying it out to a sufficient extent. It is only by the greatest perseverance that the tympanum can be thoroughly cleansed. There are a few cases which as yet appear to set this treatment at defiance, but they are rare exceptions; nor are they cases specially characterised by obvious constitutional disorder. The first object to be obtained is to coagulate the discharge, and so to render it less tenacious. This I have found best effected by the sulphate of zinc (gr. iij—v ad ℥j), with which I usually combine from one to two grains of hydrochlorate of morphia, which appears to me often very useful. Politzer gives a caution against using lotions (such as liq. plumbi), which form insoluble precipitates with the secretion, when the orifice is small, such precipitates being, of course, very difficult of removal. Nitrate of silver, chloride of zinc, chloride of iron, alum, are all of service; but the sulphate of zinc appears to surpass them all in its cleansing qualities. Tannin in solution, like vegetable astringents in general, is of less value; but applied in powder to granulations, and even to the softer kind of polypi, it is often very efficient, as also is alum and other astringent powders. But as a rule for the treatment of inflamed and discharging surfaces within the ear—either the meatus, the surface of the membrana tympani, or the tympanic cavity—I have found the most effective remedy to be powders not of an astringent, but of a simply absorbent character; and among those I have hitherto tried I have found powdered talc the best. This also I usually combine (1 grain in 20) with morphia. It should be removed by syringing, and reapplied frequently—daily if possible—and it is rare for it to fail to bring the discharging and tumid surface, however chronic may be its evil habit, into a healthy state. By means of it the formerly tedious and unsatisfactory treatment of an exposed and suppurating tympanic cavity is rendered effectual and moderately speedy. But powders also should be carefully used in perforations of small size, lest they should become entangled behind the remaining portion of the membrane. After the tympanum has been thoroughly evacuated, however, they may be applied with advantage to the edges of the orifice; absorbent if these continue granular, and more astringent and stimulating — as alum or sulphate of zinc—if the reparative process appears deficient. They may be blown in through a small glass tube, or applied by a camel's hair brush when their action on the whole surface is not desired. A similar treatment I have found also both less irritating and much more permanently effective, after the removal of polypi, than the use of caustic.

Sometimes, as in Fig. 20, with a small orifice, the puriform secretion within the tympanum is visible through the transparent membrane. Fig. 21 shows the same secretion driven through the orifice by a strong inflation, and forming a drop on the surface of the membrane. In such cases as these, I have sometimes found the injection of warm liquids through the Eustachian act favorably; but perforations of so small a size, if of any standing, seem less prone to heal than larger ones. A free incision of the membrane suggests itself as a reasonable remedy, but I have not yet employed it in any such case. Figs. 20 and 21 are taken from the right ear of a woman aged thirty, who had been deaf, with discharge, since scarlatina at eight years old. In addition to the perforation, the whole structure of the membrane seemed altered; no malleus, or only some slight remains imbedded in old deposits above, was visible; the membrane was lax and flat, and its proper structures atrophied; the meatus, too, was narrowed, by swelling of its walls. On the left side the membrane was almost wanting. The deafness on each side was very great, but was somewhat benefited by cleansing the ear with sulphate of zinc, and on the left side by the use of the artificial membrane. The treatment was cut short by the patient leaving England.

A very troublesome form of perforation, and one which it is often difficult to recognise until its symptoms have become familiar, is a small orifice situated at the lower part, nearly or quite on a level with the floor of the meatus. In this position (as when situated quite in the anterior part), a perforation is very apt to be hidden by the curved wall of the meatus, so that its presence may pass undetected, unless special attention is directed to it, and may go on for a long while to produce extreme annoyance. I have seen two or three such cases, the symptoms being quite unlike those attending perforation of any other part of the membrane, even of the inferior part if not absolutely involving the lower border. Apparently the meatus is irritated by the secretion, perhaps confined in the angle between its ascending floor and the obliquely situated membrane, and the patient complains of a constant irritation in the ear, of slight pain on moving the jaw, with sounds as of the squeezing of liquid, which he can also sometimes produce by pressing beneath the meatus. This action gives a temporary relief, and by it matter can sometimes be pressed out of the ear, though there may be scarcely any habitual discharge: the membrana tympani is thick and slightly red, and the meatus throws off flakes of softened epidermis. In the case which first drew my attention to this form of affection, there existed, besides the symptoms above described, frequent and severe attacks of giddiness, and an inability for any kind of mental exertion, which were relieved sometimes by an increased discharge from the ear, and disappeared when the tympanic irritation was removed. The patient was a youth of sixteen of healthy constitution, but suffering when first seen from a slight pustular eruption on the neck. He had had a discharge from the ear in childhood, but this had left no permanent effect. Since measles, however, at eleven, there had been a discharge, never ceasing for many weeks together, from the right ear, sometimes very profuse. This had been aggravated by a blow on the head shortly after the measles, and again by "gastric fever," fourteen months since, when there had been much pain in the head preceding the increased discharge. The symptoms complained of were those described above, which had existed, gradually increasing, for more than a year, and prevented him from pursuing his studies. Shaking the head also gave pain. Watch heard four inches. The meatus was lined with softened epidermis, but after syringing, the membrane was seen of a dull grey tumid look, and vascular along the malleus; no perforation was seen; he could not inflate the tympanum, but air blown into it passed through the membrane with a slight squeaking sound. Warm glycerine was dropped into the ear every night and a small blister applied. In a few days the swelling of the meatus had abated sufficiently to allow the upper edge of a small perforation situated at the inferior border of the membrane to be seen. He could blow air through it. There was scarcely any discharge, but a bubble seemed often to break in the ear. Chloroform and R. Iodin. were applied daily behind the ear. He was taking bark and ammonia. Subsequently the position of the orifice, which was only just visible on a line with the floor of the meatus, was touched with a ten-grain solution of nitrate of silver, a weaker solution being dropped into the meatus. The Eustachian catheter was used once or twice (a proceeding which was probably not necessary), and when the secretion seemed entirely removed, alum in powder was blown into the meatus (this also being of doubtful benefit). In two months the orifice appeared to have healed; a small white patch occupied its position, and when air was passed into the tympanum the membrane yielded before it, none escaping. All symptoms of local irritation and giddiness had ceased. Watch heard at eighteen inches. In six months the patient called on me again on account of pain in the other ear (the left). The right continued free from discomfort, and the hearing sufficiently good (watch thirteen inches). The perforation, however, had opened again and he blew through it with a dry whistling sound. There was a calcareous deposit in the upper part of the membrane, both in front of and behind the malleus, which had not been perceptible before. A small mass of hardened epidermis was removed from the meatus, which was tender. The solution of nitrate of silver was again applied, and the hearing on the next visit was twenty inches. Again, six months after, I saw the patient. The discharge had returned slightly after a cold. The orifice was about the same size as at first, and its edges were red. Hearing as before. The tympanum was now thoroughly washed out with sulphate of zinc solution, and much mucus removed. This was twice repeated, on the last occasion very little coming away. Chloroform and iodine were again applied daily behind the ear. Watch twenty-four inches; air blown through the orifice with a dry sound. Nine months afterwards the ear was again examined, and the perforation found to be healed. The tympanum was freely inflated by the patient. He complained only of an occasional bubbling sound in the ear; was able to attend well to all his duties. The membrane was in parts very thin, in parts thickened by chalky deposits. Watch heard twelve inches.

In this case the perforation, after having healed, at least so far as that the membrane bulged before the inblown air, recurred. I think, however, it may be expected, the tympanum having been afterwards thoroughly cleared, that the present healing will be permanent. The hearing is amply sufficient for practical purposes.

The opposite membrane in this case exhibited a marked condition of disease, unattended with noticeable deafness; the greater portion of its extent, especially at the upper part, was occupied by calcareous deposits, the rest of the membrane being transparent. The short process of the malleus projected strongly, the membrane falling somewhat more than usual inwards. While the Eustachian tube was pervious, however, the hearing was very good. On one occasion, after bathing in the sea, and feeling water enter this ear, it became slightly painful, and in a fortnight the tube was found obstructed and the hearing reduced to two inches. On inflating the tympanum it rose at once to thirty-six inches, and, with the use of remedies addressed to the throat, continued good.

In the tissue by which losses of substance in the membrana tympani are repaired, the proper fibrous structure of the membrane is not found. Scars consist here, as elsewhere, of connective tissue merely, covered externally and internally by a layer of cells. For the most part they are much thinner than the natural structure and are easily recognised by their darker, more transparent hue, and by being slightly depressed, the surrounding border of membrane also being generally thickened. They bulge out on inflation of the tympanum; often, if they are extensive, forming large bladdery protrusions, which gradually recede again on the cessation of the pressure from within; sometimes also they move visibly to and fro during swallowing or respiration. These scars, however, as before remarked, cannot be distinguished from thinned portions of the membrane arising from any other cause, such as superficial ulceration (as in Fig. 11), or from a mere wasting of the membrane, which occurs without any other visible change.

Figs. 18, 19, and 22, illustrate different forms of healed perforation, having been taken from patients in whom the process went on more or less under my own observation.

T. J— a healthy man, æt. 31, applied to me, June 27th, 1865, for discharge from each ear and deafness, which had come on without pain or known cause three months before. Each membrane was perforated near the centre; on the right side the orifice was nearly circular and about a line and a half in diameter; on the left, about twice as large. He could blow through both. Watch heard on each side four inches. A blister was applied, and a lotion of alum and Tr. Opii (gr. ij, and ♏v—℥j) ordered, warm, at night. On July 4th he reported himself improved in hearing and less discharge. He did not continue his attendance, but in the following year he came again at my request, and on June 1st I found that the perforation in the right membrane had healed. There was a dark hard mass in the meatus, after the removal of which the membrane presented the appearance given in Fig. 18. The general surface is somewhat opaque, but appears natural in structure; just below and in front of the umbo, is a more transparent portion, which is darker in colour as less intercepting light. It is manifestly thinner than the rest of the membrane; as the patient swallows, it sinks visibly inwards, forming a shallow depression, and bulges outward on his inflating the tympanum. In front it is bounded by a slight thickening of the membrane, but posteriorly its margin is indistinct. It occupies precisely the position of the former orifice. Watch heard twelve inches, voice sufficiently for all purposes. On the left side the orifice continues very little changed, and there is still discharge. Watch heard eight inches.

J. H—, æt. 32, policeman, was knocked down violently in a struggle with a burglar in November, 1864. A discharge followed from each ear, most from the left. He suffered much from severe pains in the head and dizziness, for which, after fourteen months (in January, 1866), he applied to Dr. Wilks. For six weeks three grains of Pot. Iod. were given without much relief, when I was requested to examine the left ear. I found a small polypus on the floor of the meatus, close to the membrane, which was swollen, of a dull grey hue, and traversed by congested vessels. Inferiorly was a roundish perforation, the size of a small pea, through which puriform matter exuded; Eustachian tube not pervious. The hearing was almost abolished, indicating a lesion deeper than the tympanum. A lotion of Sulph. Zinc. and Morph. Hydroc. (aa. gr. 2 — ℥j) was dropped into the left ear each night, and the medical treatment continued. On the 8th of March, when he was next seen by me, the perforation had healed; the upper part of the membrane was opaque, grey, flat, and seeming as if fallen in on the internal tympanic wall. Inferiorly the surface was of a snowy white, and slanted obliquely outwards and downwards. The Eustachian tube still did not yield to the attempt to inflate it. Dried secretion was removed. The pain still continued severe about the occiput. On June 13th air was passed (by Politzer's process) into the tympanum, but with slight relief; the Eustachian tube continued open; the pains in the head, though diminished, were still often severe; the hearing had improved a little, so that words could be distinguished, though with difficulty. The tuning fork, placed on the head, was also heard very imperfectly. The appearance of the membrane had altered considerably, and is shown in Fig. 19. The upper part was transformed into a dense yellowish mass, feeling to the probe like bone; the short process of the malleus projected strongly in its place, and the handle could be faintly traced below. Inferiory was a thin transparent surface of scar-tissue, which projected like a bladder on the inflation of the tympanum. I have employed locally, for the most part, morphia lotions, with occasional blisters and ointments of Pot. Iod. with Hyd. Binoid. or Ext. Aconit. The tympanum, also, has been inflated, but his visits have been rare. There is still a little discharge, and some cerebral irritation, but these are gradually abating. I do not think much improvement of the hearing probable.

The right membrane also, in this case, presents an appearance like that of a scar inferiorly, a thinner portion yielding on inflation and falling back on swallowing; but if there was a perforation it had healed before he was seen by me. On this side the tympanum appears otherwise healthy, and the hearing is much less impaired.

In this case, I think, there has been most probably a slight fracture, involving the left tympanum to some extent, and inducing a slight effusion of blood into the upper part of the cavity. The dense deposit in the upper part of the membrane, the great impairment of hearing, and the serious cerebral symptoms, lead me to this view. In other cases of blows upon the head, and notably in that of a railway guard whose head came in contact with a bridge while travelling, symptoms of a similar kind, attended with analogous lesions of the membrana tympani, have come before me, and have gradually abated, apparently being relieved by means which tended to diminish morbid action within the tympanum.

Fig. 22 exhibits a mode of healing of a perforation which I think is rare; the aperture closing, and the hearing becoming tolerably good, coincidently with the formation of polypoid excrescences in the external meatus. Much more frequently growths of this kind disappear as a part of the general more healthy action, of which the closure of the orifice is a result; and their growth or return is most commonly a sign of aggravated irritation or progressive ulceration.

J. P—, a pallid girl, æt. 19, living in a damp and unhealthy part of Bermondsey, applied to me in May, 1865, for disease of both ears, which she ascribed to colds three or four years before. She had had smallpox the previous summer. There was a small orifice of the lower and posterior part of each membrane, with how much loss of substance it was not possible to judge, the edges of the wound being red and swollen; the rest of the membrane was thick and much fallen in; polypoid granulations grew from the roof of the right meatus. Watch heard on contact on the right side, two inches on the left. Both Eustachian tubes were closed; to the right a catheter was applied, and warm water gently syringed through, with improvement of the hearing to eight inches. On the left Politzer's plan of inflation succeeded, and this was constantly repeated to each ear in the treatment. Quinine and iron were given, and lotions in great variety, laudanum, permanganate of potash, carbolic acid, zinc, were used for several months. The progress, however, seemed unsatisfactory, though the condition improved a good deal, and the artificial membranes greatly aided the hearing. I ascribed the ill-success to her unfavorable local surroundings;

but, after an absence of about two months, during which she stated she had only syringed out the ears once a week with warm water, she presented herself, much better in hearing, the discharge still continuing. On examining, I found that the perforation on each side had healed. Air, passed into the tympanum, impinged freely on the membrane, and moved it slightly outward, but did not pass through. The development of granulations, however, in the meatus had decidedly increased, and, in each ear, the upper and posterior part of the membrane was hidden by a considerable fleshy growth; in front the membrane was moderately bright, but its surface irregular; posteriorly it was of a dark red. Watch heard six inches on each side; voice easily. Fig. 22 shows the condition of the right ear; the appearance of the left is very similar. The hearing still improves, and the membranes continue entire. The portion in which the membrane was wanting is of a dark red colour and rough aspect. The polypus decreases in size slightly. I have thought it best to use no other local application than warm water; but have applied Tr. Iodin. behind the ears. The patient suffers from chronic choroido-retinitis, for which she is taking Pot. Iod. and sarsaparilla. In a younger sister both membranes are destroyed by scarlatina, and a third suffered from chronic catarrh of the tympanum, but has recovered.

It is difficult to state the limit to which a loss of the substance of the membrana tympani may be supplied by new-formed texture. Von Troeltsch and Politzer agree in stating that two thirds of its extent may be thus replaced, a statement which my own observation would lead me to consider moderate. Moos* affirms that he has twice seen the entire membrane destroyed, except the part immediately attached to the handle of the malleus, and that from this the whole aperture has been closed with new tissue. Often, however, we see apparent scars of large size, with history of discharge, in which the handle of the malleus is almost or altogether wanting. I do not know, in this respect, what can be said to be impossible. A case which I most minutely examined in the year 1865 has made it a matter of question with me whether, even when the membrane is entirely destroyed, or at least

[1] Op. cit., p. 133.

reduced to a mere narrow rim, a complete septum may not replace it.

G. F—, æt. 52, a healthy man of dark complexion. When a child, deafness and an eruption on the scalp alternated with each other. Remembers that when the head was better he was more deaf; imagines he was always deaf more or less; was delicate as a child, but had not much earache; he has, however, since had much pain in the left ear, especially about ten years ago. Used to have a discharge, but none for several years; not much tinnitus; more deaf when out of health or tired. About fourteen years ago, after looking over some business papers, he became all at once "as deaf as a post." This happens often. At first my watch was not heard on either side, but on his pushing the right tragus in a peculiar manner somewhat upwards and backwards, it was heard on that side at two inches. He can almost always produce temporary improvement in this way. The right membrane was irregularly thinned in various parts, giving the appearance of numerous small depressions, which bulged out on inflating the tympanum and fell back on swallowing. It was also a good deal fallen inwards, and the short process of the malleus projected much. A hard accumulation of epidermis was removed from the roof of the meatus, and the tympanum freely inflated, by which the hearing was much improved, varying from six to fifteen inches. From the left meatus a great accumulation of soft but adherent epidermis was removed, a pale irregular polypoid growth was then seen anteriorly, touching and partly hiding the membrane, which appeared white, of a soft thick look and irregular surface. The tympanum could not be inflated without the catheter, from which air entered it with a loud harsh blowing sound, afterwards a watch was heard one inch, and he could inflate the ear. The improvement continued the next day. The polypus was then removed by Wilde's snare, when it appeared that in the membranous septum which occupied the position of the membrana tympani there was a small orifice, like a pin-hole, previously closed by the polypus; for now when he inflated the ear the air passed out of this small aperture with a whistling sound, whereas, before, the membrane had bulged before it. The polypus itself appeared to have been developed from, or around, a small projecting spicula of bone, which yielded to a slight pressure and came away. On exploring farther, none of the ossicula could be discerned, but in the position usually occupied by the short process of the malleus the probe came in contact with a flattened bony surface. In contact with this was the thick white membrane (covered in parts with flakes of epidermis) which I had taken for the altered membrana tympani, but which on further examination appeared to have a different character. For around the inferior two thirds of the meatus, in the usual position of the membrana tympani, and just in front of the white septum referred to, there was perceived a rim of thick tough membrane, resembling soaked chamois leather. This membranous rim was about two lines in depth, it was firmly attached externally to the meatus, and its inner margin was free. On applying traction to a part of it by the forceps, the patient complained that I seemed to be "pulling away a part of the real organ," different from the pain given by the removal of the polypus or the adherent epidermis. In short, this membranous rim answered, in all respects, to a remnant of the natural membrane attached to the cartilaginous ring, which was plainly visible. But the septum which closed the meatus appeared to be quite disconnected with this. It was attached, inferiorly, evidently *within* the position occupied by the remnant of the membrane, with which it had no visible connection. It appeared like an entirely new formation of thick, strong, though flabby tissue. The next day the small perforation noticed was no more to be seen, the septum bulged on inflation, which slightly improved the hearing, but pressure on the tragus improved it more, raising the hearing distance for the watch to twelve inches. This action made no visible change in the ear, but the effect seemed to depend, in part, on the closure of the meatus, since the presence of even the smallest speculum entirely prevented it. The vapour of acetic ether was passed warm into the right tympanum with improvement of the hearing, and the patient left me with each ear performing its function moderately well. Tonics, with astringents to the throat, and a lotion of carbolic acid to the left ear were advised, but the hearing again diminished, and in five weeks he returned. Watch, right three inches, left two inches. The left ear had altered considerably; deep in on the floor of the meatus was seen a small polypus, which fell off without bleeding on being touched by a probe. There was a slight milky discharge, on removing which the portions of membrane before described were seen to have become continuous, forming a white soft septum; no orifice existed. Inflation of each tympanum again restored the hearing; right to fifteen, and left to twenty inches. This was repeated for three days with the injection of acetic ether vapour by the catheter. At the end of this time all trace of the polypus was gone, and the ears appeared in a healthy condition. The septum above described had evidently grown into one with the circumferential remnants of the membrana tympani, forming a complete membranous layer occupying nearly the ordinary position. The line of junction was visible as a slight ridge. The membrane was of an opaque white, soft-looking, but smooth and bright, and fell slightly into vertical folds, especially at the upper part. It was quite insensitive when touched, and fell inwards towards the promontory, which was easily felt by the probe. There was no trace of malleus or the other ossicula.

The patient could generally inflate the tympanum, when it yielded slightly, most at the lower part. The hearing was sometimes a little improved by this; but it became much better at times without obvious reason. Sometimes a little rub of the meatus would improve it; at others it was better after the escape of a little fluid from the ear. Believing that these variations depended upon the partial loss of the ossicula, and were determined chiefly by the varying position of the membrane in respect to what remained of them, I introduced the artificial membrana tympani, which had a decidedly good effect, though I did not always succeed in placing it aright. With it the watch was heard often at fifteen or twenty inches, and the voice without difficulty.

I am uncertain what construction to put upon the appearances in this case; whether an almost entirely new septum was formed after loss of the natural membrane, or whether after a partial loss the relaxed central part had sunk back, and so given the appearance of a mere ring of membrane below, with a new membranous formation behind it. The total disappearance of the malleus, and the entire distinctness of the partial ring of membrane from the septum behind—so that even the separation of the former from its attachments did not disturb the latter—seem to me to favour the former view, but it is difficult to believe it possible. I report, merely, what I saw.

The extent to which the hearing is restored when a perforation of the membrane heals, is very variable. Mainly it depends, of course, upon the condition of the deeper situated parts of the organ. But although the hearing may be exceedingly good in spite of most extensive disorganizations of the membrane, and these of very various kinds, it appears that certain accidents of healing exercise a considerable influence upon it. The contraction of the scar, which ensues speedily upon its first formation, has often appeared to me to diminish the hearing power for the time. In a large proportion of the cases of healing I have had an opportunity of closely watching, I have noticed an increase of deafness immediately following the closure of the perforation, which has passed off, or given place to a decided improvement, in the course of some weeks. This effect may be due to an increased *tension* given to the membrane for the time. But in healing, the edges of a perforation, or the new tissues which unite them, frequently contract adhesions with other parts of the tympanic cavity, and, especially if the orifice be large, with the internal wall. A scar may frequently be seen adherent to the promontory; sometimes in its whole extent, almost obliterating the cavity, or dividing it into two, one above and another below.

Sometimes the healing is incomplete, and a mass of scar tissue adheres to the promontory, binding down the malleus and the superior portion of the membrane, while the inferior margin of the perforation remains free, and the lower portion of the tympanum is exposed. Or the adhesions may assume other positions, closing up the anterior or posterior portion only of the tympanum. In the former case air blown through the Eustachian tube may not escape externally, although the membrane is still perforate. Such adhesions are unfavorable to hearing; but cases in which they are very extensive may sometimes be benefited by means of the mechanical pressure of air exerted from within.

The air-prěss, with or without medicated vapours, or simply blowing through the catheter (which a patient may be taught to introduce for himself, and so be made independent of the surgeon to some extent), perseveringly employed, may loosen an adherent scar, and restore to use even a long useless organ.

In April, 1865, I was consulted by a clergyman, æt. 46, on account of a sudden and almost total deafness affecting the right ear, the left having been "quite dull" since an attack of fever fourteen years before, since which time there had been an almost constant slight discharge from that ear. On examination, the right tympanum appeared to be almost free from disease, and the hearing fortunately returned in the course of a fortnight. On the left side the membrane was seen to be thick and dense around the short process of the malleus, which alone was visible; the greater part was dark, thin, irregular, and fallen in upon the tympanic wall, the remnants of natural structure, and scar-tissue being indistinguishable. He could not inflate the ear. On that side he required a very loud voice close to the ear, and a loud crack of the nails was heard only at one inch distance. A tuning fork placed on the head, however, was heard almost as well as the average. It was thought advisable to attempt to improve the left ear, if possible, so far at least as to make it available for the occasional relief of the other. The Eustachian catheter was introduced, and air blown into it passed along the Eustachian tube with a free, harsh rustle, and the hearing was slightly improved. The inflation of the tympanum with air, and with steam mixed with the vapour of iodine, was repeated frequently, and a small curved knife was introduced through the membrane to divide any adhesions that might be binding down the malleus; subsequently the patient was taught to introduce the catheter for himself. By degrees the membrane was seen to be partly raised up from its collapsed position, and small bladdery protrusions formed at the lower part during the inflation of the tympanum. The discharge also ceased under the use of various lotions, and alum and talc in powder. The hearing gradually improved, and in the course of three or four months the ear had become quite effective for quiet and distinct conversation. He is now, after an interval of eighteen months, in about the same condition, though it varies a good deal.

Schwartze[1] reports a somewhat similar case with a more complete success. The patient was a woman aged 67, whose right ear had been deaf from childhood, and who had lost the hearing of the left five years previously after severe pain. She could scarcely hear without an ear trumpet; the whole of the left membrana tympani was fallen in and only yielded to inflation or suction in one small spot anteriorly. On using the catheter, with a moderate stream of air, the hearing returned so that conversation was easily distiuguished; and on examination the membrane was seen less sunken, bladder-like in parts, the stapes no more projecting, and in the centre was a large ecchymosis. A tearing sensation had been felt in the ear. The improvement remained in spite of the renewed sinking inwards of the membrane, and the re-formation of adhesions was guarded against by repeated use of the catheter. A distressing tinnitus also was removed.

For perforations that do not heal the artificial membrana tympani is in most instances a sufficient relief. I find a simple disc of vulcanized India rubber, with a silk thread attached to the centre, very effectual, and, as it seems to me, free from every objection to which the wire, as employed by Mr. Toynbee, might have been open. I have not seen any irritation excited by it, nor have I known it cause tinnitus; if it is used too long the silk may cut itself out, leaving the disc in the meatus, which may alarm the patient, but it is soon removed by the syringe. It might also easily be strengthened at the centre. In this form it is very inexpensive and available for the poor. It is now well known that the relief given by an artificial membrane or plug of cotton wool is not confined to cases in which perforation exists. Erhard,[2] I believe, was the first who called attention to this fact, which he proved upon his own person, and Mr. Toynbee, in a paper read during the last session of the Medical and Chirurgical Society, adduced cases and dissections in support of the view, that in every case of benefit from the artificial membrane there was either a partial loss or some displacement of the ossicula, especially a loosening of the incus from the stapes. Further investigation will decide this point; in the mean time the case from which Figs. 8 & 9 were

[1] 'Archiv für Ohrenheilkunde,' B. II, H. 3, p. 207.

[2] 'Schwerhörigkeit Heilbar durch Druck.'

taken has a certain interest in relation to it. The patient was a man aged 20, who had been hard of hearing as long as he remembered. When about thirteen the ears discharged and bled; does not remember ever blowing *through* the ear. At sixteen he consulted Mr. Toynbee, under whom he became much better. He ordered the use of the artificial membrane occasionally, but the ear would not bear it. Often hears better for a time on inflating the tympanum, and also after rubbing the tragus (as in the case of G. F—before mentioned). Before consulting Mr. Toynbee had much tinnitus, very little since. Had measles and whooping-cough when a child, never scarlatina. Watch heard on contact each side; voice slightly raised near; tuning fork on head well heard, rendered louder in each ear by closure of the meatus; right membrana tympani covered with red granulations over all its surface, except a small portion above and below; much fallen in, but coming forward on inflating. Left membrane of dull grey hue, and softened, sodden look, irregular surface, its central part fallen in upon the promontory; inferiorly there is a portion especially lax and thin, which falls distinctly inwards on swallowing, and much more if the nose is at the same time closed; on inflating the ear, the whole membrane starts outwards, and the lax portion projects like a bladder.

The ears were inflated on Politzer's plan, improving the hearing greatly, and the tinnitus ceased. A lotion of carbolic acid, 1 part in 300, with Tr. Opii ten drops to the ounce, was used. A slight attack of scarlet fever shortly followed, but did not affect the hearing. On his recovery I found the ears in the same state. The improvement of hearing from *rubbing* them was very striking. It was found to have most effect after inflation; the little finger was introduced into the meatus, and worked about a little while, and then withdrawn with a click. This was seen to cause the distended membrane on the left side to fall much inwards again; and when it was again distended by inflation the watch was not heard so well. On a subsequent occasion it was noticed (and afterwards confirmed), that when he inflated the left ear he heard my (rather low-ticking) watch *worse*, but loud sounds such as carts, and the voice, *better*. During syringing on this side he once or twice had a distinct sensation in the tongue. Large masses of dried secretion and plates of epidermis, clinging about the roof of the meatus and upper part of the membrane, were removed from both ears. To the right membrane powdered alum and talc were applied, and subsequently solid nitrate of silver was gently rubbed over the granulations. After this a minute perforation formed in the centre of the membrane, not visible, but permitting the escape of air. After a fortnight's use of a lotion of Zinci Sulph. and Morphia, and after a little purulent fluid had been forced through it by inflation of the tympanum, this orifice healed. Soon, however, it returned, still very minute. Under the occasional application, by a camel's-hair brush, of a ten-grain solution of nitrate of silver, the granulations almost entirely disappeared. The hearing, always improved by inflation, became permanently much better; watch from six to twenty inches; voice heard well across a moderate-sized room, and sufficiently for all purposes.

The left membrane also gave way slightly. After inflation had been practised several times, the hearing having much advanced, there became visible through the membrane, at its posterior and upper part, a yellowish mass, evidently within the tympanum, and having all the aspect of pus. About the same time I observed that not only did the membrane sink in during swallowing, but that when he breathed forcibly through the nose it flapped in and out, and also even during ordinary breathing after a certain motion of the jaw. This caused a small quantity of purulent matter to exude; when he inflated the tympanum, however, the membrane bulged strongly, but neither air nor fluid escaped. There appeared, therefore, to be a small valvular opening in the membrane, situated just below the position of the stapes. A small oval red surface like an ecchymosis, also, was seen a little above the position of that bone (which was not visible). This red spot had disappeared in three weeks, as also had the fluid within the tympanum and the opening in the membrane; a lotion of Zinci Sulph. and Morphia had been used. The hearing on this side also became and continued good. A ten-grain solution of nitrate of silver was brushed over the membrane for a time with a view of diminishing its relaxation, but apparently without effect.

Rhinoscopy was had recourse to, to examine, if possible, the condition of the Eustachian tube, while the membrane moved during respiration, but no good view could be obtained, and it was found also that the peculiar motion of the jaw by which the tube was rendered patent could not be performed with the mouth open. This motion did not seem at all to affect the position of the soft palate.

In this case the artificial membrana tympani produced a decided, though not very great, improvement in the left ear (without existing perforation). On the right side it produced none. It is worthy of notice that on this latter side inflation of the ear had a simply beneficial effect; on the left side, on the other hand, even when it improved the hearing for the voice and louder sounds, it diminished it for the watch. The pressure, too, on the meatus by which the membrane was forced inwards, always greatly improved the hearing, and after this adjustment inflation as uniformly diminished it, requiring a readjustment of the pressure to restore it.

It appears most probable to me, that on the right side, on which inflation was beneficial and the artificial membrane not, the ossicula were perfect, and that bringing the membrane out from its collapsed position restored the normal relation of the parts. On the left side, in which benefit was procured by pressing the membrane inwards, and by the artificial membrane, while inflation always impaired the hearing of some sounds, and sometimes of all, I should be of opinion either that part of the chain of ossicles was wanting, or that the incus and stapes were disconnected, and that the solid connection of the membrane with the labyrinth was complete only when the membrane was approximated to the internal tympanic wall; being thus probably brought into contact with the head of the stapes, which, however, was never visible.

The contrast on the two sides seems to me to lend support to the view, that the artificial membrane acts on the chain of bones. The view has been propounded that in cases of perforation the amount of hearing greatly depends upon a pervious condition of the Eustachian tube. My experience is entirely discordant with this view; and I believe that the mere perviousness of the tube is by itself of no influence when the membrane is incomplete.[1] The presence of a bougie

[1] Some cases in proof I published in the 'Lancet' for 1865.

in the Eustachian tube, however, will, for the time, render the watch audible at a greater distance in some cases of perforation. This appears to me most probably due to irritation of the tensor tympani, causing the muscle by its more vigorous contraction to brace up for the moment the relaxed ossicula. Washing the ear with a solution of sulphate of zinc will often have a similar effect, lasting for some hours, in cases in which warm water has no such influence.

Mr. Nottingham[1] reports cases of perforation in which great improvement followed the use of a solution of sulphate of copper. A bracing influence on the ossicula seems a probable explanation in these cases; as also in those frequent instances in which, after the use for some months of an artificial membrane, the hearing, though the perforation continues unchanged, remains good without it.

Granulations on the surface of the membrane (as seen in fig. 8) are not very uncommon. Sometimes they extend from the meatus to the membrane, at others they are limited to a portion of the latter. For local treatment, the application, once or twice a week, of Arg. Nit. gr. x—xx ad ℥j has seemed to me to answer well. In the case narrated above, a small perforation followed the application of the solid nitrate. I do not think in consequence of it, but that the secretion within demanded an outlet. After it healed it opened a second time. Even when a large part of the surface of the membrane is covered with granulations the hearing may be, practically, very good.

A thinning and falling inwards, in a more or less collapsed state, of the membrana tympani appears to be a frequent consequence of inflammation within the tympanum. Figs. 6 and 7 illustrate this condition, complicated, however, with obstruction of the Eustachian tube on both sides, and in the left with great tumefaction of the floor of the bony meatus, and inflammation of its lining membrane. The patient was a youth, æt. 17, weak, but of sound constitution, who consulted me in April, 1865. The disease of the ears commenced about the age of 10, with a severe affection of the throat; ever since that time there had been more or less discharge from the left ear, and his deafness had gone on increasing, though it would be

[1] 'Diseases of the Ear,' 1857.

relieved, at rare intervals, for a few hours after loud cracks in the ear (characteristic of deafness from obstruction of the tube). A loud voice was required, quite near either ear; watch not heard; a loud crack of the finger-nails heard only upon contact on the left side, and at 2 inches on the right; the tuning-fork, however, was heard well on the head. The deafness was interfering seriously with his mental development. Throat relaxed and red. Each membrana tympani was very much fallen in. On the right side it had a dry and shrivelled look, the surface irregular, red streaks along the malleus, which stood strongly out. On the left side the membrane had a similar position, but it looked sodden and soft. Inflation on Politzer's plan, which was easily effected, at once restored the hearing. The air entered the left ear with a clap, the right (afterwards listened to) with an obscurer wheezing sound. The patient exclaimed, "I can hear now," and was immediately able, with each ear, to carry on natural conversation at six feet. A low watch was heard on contact on the right side, and at one inch on the left. Astringent gargles and stimulating liniments with iodine were ordered, with the iodide of iron and a small dose of quinine, internally, and hygienic measures. The inflation of the tympanum, the effect of which, at first, was temporary, was practised almost daily for a fortnight; the hearing progressively improving. At the end of this time a perforation formed on the left membrane, not, however, at the spot on which air from the Eustachian tube impinges (anteriorly and below), but in the posterior part about on a line with the umbo. This perforation never exceeded two lines in depth by one in breadth; a little matter escaped from it at various times, and by the middle of June it had healed again. On the right side, the inflation being continued also, there became visible in the posterior portion of the membrane a well-marked yellow surface, which, on narrow inspection, appeared to be due to fluid contained within the tympanum, which the air forced into it thus brought forward into contact with the inner surface of the membrane. The inflation evidently increased the apparent mass of this fluid, and though no visible perforation ever formed, and air never seemed distinctly to pass through the membrane, on more than one occasion a few drops of puriform fluid were found in the meatus afterwards, and the yellow appearance within vanished by degrees. On July 14th the hearing had for some weeks continued satisfactory without treatment, there was no perforation on either side, and the discharge from the left meatus (to which powdered alum had been applied a few times) was very slight, none escaping. At this time the patient took a walking tour in Scotland, continuing well until he contracted a severe cold. On his return in September, I found him again very deaf; each meatus was red, swollen, and discharging; he could not inflate the ears.

Inflation, during which air passed through both membranes, again restored the hearing. A similar treatment was resumed, and the perforations healed. The inflammation of the left meatus, however, tended constantly to recur, and with every cold the hearing again became bad. An India-rubber bag, fitted with a nozzle, was supplied to him, by means of which to inflate occasionally his own tympana,[1] and by this means he was able for many months to maintain the hearing at a satisfactory pitch.

The lax and atrophied condition of the membranes, however, which gives them a constant tendency to fall inwards, renders a thoroughly satisfactory result difficult, though, I think, not impossible to obtain. The drawings were taken on the 30th May, 1866, and represent the aspect of the membranes at a time when the hearing was so far good that deafness could hardly have been suspected by a stranger.

Somewhat less frequent than the collapse of the whole membrane, as in the foregoing case, is a partial collapse such as that represented in Fig. 5. The patient was a healthy girl, aged 18, who came to me at the Hospital in November 1865. She had become gradually deaf, she said, some three or four years since, from colds; there had been discharge till about a year ago. Watch, contact each side. Tuning-fork heard well on the head; closing the ears made no difference to the sound. No tinnitus. On the right side (see fig. 5) all the anterior portion of the membrane, except a narrow margin in front, and a very slight streak separating the deposit from the handle of the malleus, was occupied by a firm white mass of chalky deposit, which visibly increased the thickness of the

[1] For this purpose I think Dr. Andrew Clark's two small India-rubber balls, as used for pulverising fluids, is the best.

membrane. Behind the malleus, the upper part of the membrane was thinned, sunk in, and evidently crumpled, or fallen into loose folds. It lay in contact with the stapes, which was plainly to be discerned through it, the head of the bone forming a well-marked projection. The long process of the incus could not be clearly distinguished. The patient could not inflate her own tympanum, but on my doing so, the hearing was at once improved to four inches for the watch, and very greatly for the voice. On then examining the membrane, the collapsed portion was seen to be blown outwards, away from the stapes, which was no longer visible; the folds or creases were obliterated; and the portion of membrane had a slightly pink hue, from minute vessels on its surface; it seemed to be rendered vascular by the inflation. The improvement lasted only for two or three days. Tonics were given, and astringents applied to the throat, and inflation of the ear practised frequently, always with benefit, sometimes lasting several days; but the permanent improvement was but slight. The patient was therefore taught to inflate her own ears on Politzer's method, or rather (what I find the simpler plan) a relative was taught to blow into her ear during swallowing, by means of a piece of india-rubber tubing. This was done every day; and the result was that the hearing became and remained good. Watch, eight inches; voice well heard at twenty feet; no inconvenience in her profession, that of a teacher of music. The collapsed portion of membrane, however, still lay in contact with the stapes. After treatment had been discontinued for two months, the hearing continued good. In this case, the diseased condition on the two sides was (as it often is) strikingly symmetrical; the left membrane also being collapsed to precisely the same extent, and lying in contact with the stapes. The outline of that bone, however, was thickened and obscure, instead of delicate; and there was no deposit in the membrane anteriorly. On this side inflation at first diminished instead of increasing the hearing distance for the watch; but it did not appear to have this effect in respect to the voice, and its daily repetition had an equally favorable result on each ear. The artificial membrane was at one time tried, and found to improve the hearing on each side.

A younger sister of this patient was seen by me once. The right membrane was generally thin and slightly collapsed, and a small polypus came away on syringing. Watch, four inches. On the left side the membrane generally was healthy, but a white, rigid, somewhat raised band ran from the short process of the malleus to the posterior wall of the meatus. Watch, twenty inches. She also ascribed the disease of the ear to colds.

Whether the partial collapse in this instance proceeded from a perforation which had healed, or from a process of atrophy, preceded or not by inflammation, it is not possible to say. For a long time I had never seen a perforation confined to the upper and posterior part of the membrane; but lately two or three recent instances of it have come under my observation; one of them also in a young woman with a history simply of colds, and in whom there exists an old perforation posteriorly, but lower in its position, in the opposite ear as well. I think it therefore very probable that the collapsed portions of membrane in the preceding case are scars. In this instance, the deposit in the membrane was of a calcareous character, and though very extensive and of very considerable thickness, it can scarcely be supposed to have interfered materially with the hearing, both on account of the further morbid conditions, which were amply sufficient to account for the deafness that existed; and because, on the opposite side, in which there was no deposit, but which was in other respects similar, the hearing was, if anything, still less perfect. Such calcareous deposits, the seat of which is in the fibrous layers (the *Substantia propria*), are very common; and though they frequently accompany other lesions of the tympanum, which give rise to deafness, they often exist where no imperfection of hearing is detectable. Politzer, who has examined this matter very minutely, met with chalky deposits in the membrane very frequently in persons with perfect hearing, and reports, among other cases, that of a student in whom there existed on each side crescentic chalky deposits, both in front of and behind the malleus, the rest of the membrane being healthy. He had never had ear-disease; and not only was his hearing perfect, but he had an acute and delicate musical ear.[1]

[1] 'Trommelfelltrübungen und deren Bedeutung für die Diagnostik der Gehorkrankheiten,' Wien, 1863.

These deposits arise frequently after perforation and discharge from the tympanum, but so far as observation has yet extended, scarcely less often when there has been no discharge and the membrane has not given way. Moos[1] states that he has several times watched their development, and has found that they may form very rapidly. In a girl aged 9, who perforated the membrane with a knitting needle, and suffered from an acute suppurative inflammation of the tympanum, a considerable chalky deposit formed in the anterior portion of the membrane in fourteen days after the perforation had healed, the hearing being as good as could be desired for the voice, though much diminished for a watch. In the case of a woman aged thirty-six, Moos saw a chalky deposit the size of a grain of wheat form in four weeks. She had had chronic catarrh of the tympanum without perforation for eighteen years. The hearing was less impaired on the side in which the deposit formed than on the other.

In the case represented in Fig. 3, the hearing was good; to the voice apparently perfect; for my watch thirty inches (about the same on each side). The drawing was taken from the left ear of a girl aged eleven, who had never had any affection of the ear. Her sister, however, aged fourteen, was under my care for deafness, and very clearly belonged to the class of patients whom Mr. Hutchinson has pointed out as being instances of hereditary syphilis. The taint seemed to be faintly expressed also in the subject of the drawing, in the general form and aspect of the face, but there had been no affection of the eyes, nor other characteristic disease. Besides the chalky deposit (which takes in this instance an oval form, less frequent than that of a crescent, corresponding to the course of the circular fibres), the membrane generally was of a patchy white opacity.

The hereditary disease, if it existed in this case, probably had exercised little or no influence on the condition of the ear; chalky deposits are met with in such cases, but are by no means very frequent. The ordinary aspect of the membrane in cases of deafness from congenitial syphilis is represented in Fig. 2:—a dull, whitish, uniform opacity, the membrane having a flattened and somewhat dried and rigid aspect. The appear-

[1] Op. cit., pp. 101, 120.

ance gives the impression of a thickening of the mucous layer of the membrane, or of a deposit, not improbably of lymph, within the tympanum; but as yet there are, I believe, no post-mortem examinations of these cases which would justify us in speaking positively.

This subject presents many features of great interest, but it cannot now be entered upon. Deafness thus arising is unhappily very common, and is also usually very extreme in degree, as it was in the present instance. The patient, aged thirty, improved very markedly for a time, while taking scruple doses of hydrochlorate of ammonia. Although she had been scarcely able to understand a word, she became so well that she undertook a situation, but its duties proved too much for her strength, and her hearing suffered a relapse, from which, however, she again shows signs of recovering.

In contrast to the preceding case, Fig. 10 exhibits the condition of the membrane in a man suffering from acquired (secondary) syphilis. It was taken from the left ear of a man, æt. 23, who applied to me May 15th, 1866, having had a chancre six months previously, and suffering from ulcerated throat. The membrane seemed in a condition of syphilitic ulceration. But the whole of the disorganization it had undergone was not the effect of the specific affection, which appeared rather to have seized upon a part previously the subject of disease. From a child there had been, at times, a discharge from the left ear, but it had ceased for some time until four weeks before. The watch was heard only on contact. Anteriorly, the membrana tympani was of a dark livid colour; the posterior and upper portion was of a dull brown-red, the lower part presenting a dirty, grey, rough surface, resembling a slough, and bounded superiorly by a hard and swollen edge. Nothing of the malleus could be seen, but near the position of the short process there was a small, very dark oval spot, depressed, and looking like a perforation. The Eustachian tube was obstructed.

Iodide of potassium was given in three-grain doses, and also applied as wash, and in a week, though there was some swelling and excoriation of the meatus (frequent in secondary syphilis), the membrane was much healthier, and the ulcerated surface seemed healing. Air was inflated, distending but not passing through the membrane, and improving the hearing from three inches to six. The patient did not return, but I have recently seen him again. A hard mass of thickened secretion was removed from the meatus, which was red, and, where it bounds the membrane posteriorly, had a swollen and angry margin; the membrane was concave and dirty white, like sodden linen. The malleus was just visible, much drawn in; above the short process there still remained the dark depressed surface. Watch heard three inches, improved by inflation of the tympanum to ten inches. Membrane not perforated.

Another form of deposit in the membrane is represented in fig. 4; it is not chalky, but firm and cartilaginoid, mnch resembling the atheroma of the larger vessels. It has its seat, chiefly, in the fibrous layers, but it more or less involves the rest. The fibres are, in some of such cases, found individually enlarged and thickened; in others, they are broken down and mixed with granular detritus, or with fat-globules.[1]

In the present case, the thickening of the membrane is conjoined with other morbid conditions within the tympanum; probably tense bands uniting the inner surface of the membrane to the tympanic wall, for when the tympanum is inflated which the patient can now do with ease, the posterior and upper part of the membrane bulges outwards before the pressure of the air, but no other part yields. The same result ensues when traction is exerted upon the membrane by rarefaction of the air in the meatus. A similar condition exists to a somewhat less extent on the opposite side.

The patient is a youth, æt. 20, who applied to me in September, 1863. Had been becoming deaf seven or eight years, he thought from colds; no illness except a "brain-fever" five years ago, probably from the sun on his head (he was at the Blue Coat School), but this did not influence the deafness. Lately, he had had pains in the ears, especially in the left; there was a constant whizzing noise in the left ear, and very frequently also in the right. Watch, right half an inch, left contact; heard on the skull, as was also the tuning-fork, fairly well. On treatment, consisting of inflation of the tympanum with the vapor of chloric ether, and the use of an ointment

[1] Politzer, loc. cit.

of iodide of potassium with the biniodide of mercury, the pain soon ceased, the tinnitus left the right ear and became somewhat less in the left, and the hearing improved to a decided extent, especially for the voice, on each side, though very distinct speaking was still necessary. Beyond this point, however, I have not been able to advance the case, in spite of the use of many remedies, including the introduction of a small curved knife behind the malleus with the view of dividing any adhesions that may exist, and so restoring mobility to the membrane and ossicula; a plan first employed, I believe, by Bonnafont,[1] and lately employed with success, at least for the time, by Mr. Toynbee. In the present case it proved neither useful nor harmful. The position at which the knife was introduced may be seen posteriorly, where the deposit seems to divide; but nothing in the form of a scar is visible.

[1] 'Maladies de l'Oreille,' 1861.

EXPLANATION OF THE PLATES.[1]

1.—Healthy membrana tympani, taken shortly after death.
2.—Membrane of a white opacity, except a narrow ring at the circumference; a rigid and prominent white margin at the upper part in front; hereditary syphilis (see p. 664).
3.—Chalky deposit with mottled opacity of the membrane; hearing good (see p. 663).
4.—Crescentic deposit in the course of the circular fibres; membrane concave anteriorly (probably bands of adhesion in tympanum), (see p. 665.)
5.—Membrane thin and collapsed on stapes, posteriorly; anteriorly, large chalky deposit; hearing restored (see p. 660).
6.—Thinning and collapse of membrane; bony meatus enlarged, and its lining inflamed; closure of Eustachian tube, left ear (see p. 658).
7.—Membrane thin and fallen inwards; thickened epidermis partially covering it posteriorly, meatus somewhat enlarged and red; closure of Eustachian tube, right ear (see p. 658).
8.—Granulations on surface of membrane, which is thin and fallen in; stapes projecting; a minute perforation in the granular portion not visible; right ear (see p. 655).
9.—Membrane exceedingly thin, and fallen completely in upon the promontory, with exception of a small thickened portion attached to the short process (which alone remains) of the malleus; a thick white mass, apparently of epidermis and dried discharge, overlies the stapes; hearing moderately good; left ear (see p. 655).
10.—Ulceration of the membrana tympani; secondary syphilis; discharge since childhood (see p. 664).
11.—Chronic inflammation of meatus and membrana tympani; an ulcerated surface below the malleus; small perforation at its anterior part (see p. 625).
12.—Sloughing of membrana tympani; ecchymoses on its internal surface; puriform secretion in the tympanum (see p. 627).
13.—Old perforation of membrana tympani, not healing (see p. 633).
14.—Perforation beginning to heal; membrane thick and uneven (see p. 635).
15.—Ditto, farther advanced; membrane beginning to clear; thickened rim of epidermis.
16.—Ditto, healed.
17.—Ditto, scar contracting; membrane healthy; slight vascularity near the scar.
18.—Perforation healed; scar thin and depressed, bulging on inflation; thickened margin anteriorly (see p. 645).
19.—Perforation healed; hard deposit above, thin bladdery protrusion below; posteriorly a small polypus (see p. 645).
20.—Pinhole perforation in centre of membrane; purulent fluid in tympanum, appearing through; membrane flaccid, dark, slightly vascular; malleus not visible; bony meatus slightly enlarged and lining reddened (see p. 641).
21.—Ditto, a drop of pus forced through the orifice by inflation of the tympanum.
22.—Perforation, posteriorly, healed with formation of an irregular polypoid growth; position of perforation dark and livid (see p. 647).
23.—Membrane destroyed (after chicken-pox), except a thin margin; mucous membrane of tympanum red and spongy; stapes just visible; malleus remaining, but in contact with promontory (see p. 631).
24.—Old destruction of the membrane, a narrow margin only remaining; head of stapes visible; incus and handle of malleus wanting; mucous membrane of tympanum dry (see p. 632).

[1] The reader is requested to notice an accidental error in the arrangement of the plates, whereby the numbers of the series are made to follow from right to left instead of from left to right.

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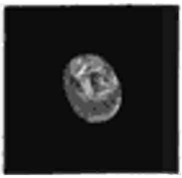

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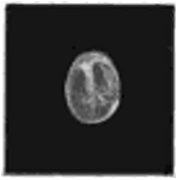

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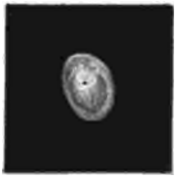

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W West Chromo-lith.r

LIST

OF

GENTLEMEN EDUCATED AT GUY'S HOSPITAL,

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,

&c. &c.

University of Oxford.

SECOND EXAMINATION FOR THE DEGREE OF BACHELOR OF MEDICINE.

Augustus Burke Shepherd, M.A., Bras. Coll.

University of Cambridge.

EXAMINATION FOR THE DEGREE OF DOCTOR OF MEDICINE.

T. D. Welch, M.A., M.B., Cai. Coll.

SECOND EXAMINATION FOR THE DEGREE OF BACHELOR OF MEDICINE.

George Mickley, B.A., Clare Coll.

EXAMINATION FOR THE DEGREE OF MASTER IN SURGERY.

T. D. Welch, M.A., M.D., Cai. Coll.

FIRST EXAMINATION FOR THE DEGREE OF BACHELOR OF MEDICINE.

Oswald Henry Foster, B.A., Cai. Coll.

University of London.

EXAMINATION FOR THE DEGREE OF DOCTOR OF MEDICINE.

Frank Buszard.

SECOND EXAMINATION FOR THE DEGREE OF BACHELOR OF MEDICINE.

First Division.

*Arthur George Mickley. | †Charles Smith.

---

* Gold Medal and number of marks qualifying for the Scholarship in Midwifery.

† First Class Honours in Medicine. First Class Honours in Midwifery.

*Ebenezer Fulham Turner. | †George Henry Savage.
‡Francis Thomas Tayler, B.A.

SECOND EXAMINATION FOR THE DEGREE OF BACHELOR OF SCIENCE.

First Division.

§Stephen Wootton Bushell.

FIRST EXAMINATION FOR THE DEGREE OF BACHELOR OF MEDICINE.

First Division.

||Frederick Taylor.

PHYSIOLOGY ONLY.

First Division.	Second Division.
George Rolph Raine.	Reginald Eager.

PRELIMINARY SCIENTIFIC EXAMINATION FOR THE DEGREE OF BACHELOR OF MEDICINE.

First Division.

**George Bradley.
Johannes De Liefde.
Frederic Durham.
††Richard Clement Lucas.
§§George Henry Percival
George Francis Kirby Smith.
Edward Harry Steele.
Hugh Eccles Walker.

Second Division.

Alfred Ashby. | William Stanger.
John Taylor.

Royal College of Physicians of London.

EXAMINATION FOR MEMBERSHIP.

Philip Henry Pye-Smith, M.D.

---

* Honours in Medicine.

† First Class Honours, and number of marks qualifying for the Scholarship in Medicine. First Class Honours in Midwifery.

‡ First Class Honours in Medicine. First Class Honours, and number of marks qualifying for the Scholarship in Midwifery.

§ Obtained the Scholarship in Biology. Placed first, and First Class Honours in Geology and Palæontology.

|| Obtained Gold Medal in Organic Chemistry and Materia Medica. Gold Medal in Anatomy.

** Honours in Chemistry and Natural Philosophy.

†† Honours in Biology.

§§ Honours in Biology.

EXAMINATION FOR LICENTIATESHIP.

George Henry Savage.
Charles Smith.
Francis Thomas Tayler, B.A.
Alfred Edward Wilmot.
G. J. B. Stevens.
R. W. S. Barraclough.
John Morrill.
F. W. Humphreys.
John Gill.

FIRST EXAMINATION FOR LICENTIATESHIP.

W. P. M. Boyle.
Charles Gôte Gurdon.
E. Noble Edwards.
R. C. Lucas.
William Toulmin.
C. J. Sells.
Benjamin Walker.
George Stokell.
G. F. Trotter.
Henry Cheesman.
J. W. Morris.
W. Betts Giles.
J. Greaves Wiseman.
W. J. Bennett.
William Bevan Lewis.
George Rootes.
G. S. Elliston.
William Stanger.

Royal College of Surgeons of England.

EXAMINATION FOR FELLOWSHIP.

Alexander Mason Mac Dougal.

EXAMINATION FOR MEMBERSHIP.

NOVEMBER, 1865.

A. Chr. Jackson.
John S. S. Perkins.
Charles Smith, M.B.
James Hopkins Walters.
John Augustus Ball.
Richard Bottomley Nowell.
James Mare Taylor.

JANUARY, 1866.

Algernon Ewen.
R. R. S. C. C. Lloyd.
G. John Muriel.
W. Evatt Wright.
Stephen Wootton Bushell, B.Sc.
J. Haslam.
F. E. Manby.
H. Cecil Smith.
Herbert Goldingham Budd.
Fred. Everard Hunt.

APRIL.

E. L. Crowther.
H. K. King.
E. M. Owens.
Henry Warlow.
William John.
William N. Hiron.
Joseph Riley.
Samuel Key Watson.
John Williams.
Arthur Goodwin.
F. W. Fowke.
Henry Maynard.
George Spearman.
Henry Morris, B.A.
William J. Richards.
George Rolph Raine.
Wm. French Thurston.
Arthur Matthews.

May.

John Jenkins.
G. S. Elliston.
William Grimes Palmer.
G. T. Hankins.
George Birch.
Richard Morgan.
C. Handyside Carver.
A. Newsam.
H. W. Fagge.
A. E. Fluder.
Alfred Harwood.
Thomas Franklin Lloyd.
G. J. B. Stevens.

July.

Reginald Eager.
F. Vinay Cardozo.
Alfred Claude Taylor.
J. G. Carruthers.
H. S. Taylor.
E. A. Trimnell.
F. W. Salzmann.
John Lewis W. Ward.
J. C. Chester.
Edward Clarke, M.D.

EXAMINATION FOR LICENTIATESHIP IN MIDWIFERY.

Robert W. S. Barraclough.
Andrew Christopher Jackson.
Frederick Martyn Rickard.
William French Thurston.
William John.
Robert Charles Earle.
Henry Weekes.
Joseph Riley.
William Harris Butler.

FIRST, OR ANATOMICAL AND PHYSIOLOGICAL EXAMINATION.

November, 1865.

Benjamin Duke.

January, 1866.

Ernest Fussell.
J. R. Bosworth.
G. E. Martindale.
T. E. Webb.

April.

J. L. Moseley.
C. J. Sells.
C. Munden.
W. B. Lewis.
George Stokell.
J. W. Barry.
Richard Rendle.
J. G. Wiseman.
Frederick Taylor.
A. H. Buck.
Johannes De Liefde.
Branford Edwards.
Charles Higgens.
J. A. Sharp.
T. J. Burroughs.
J. R. Morgan.
Henry Gould.
J. F. Goodhart.
P. T. Scott.
C. W. Chapman.
Albert Williams.
John Webb.
Alfred Ashby.
W. R. Cortis.
A. H. Baines.
F. S. Daldy.
J. G. Carruthers.
C. J. Worts.
W. J. Bennett.
George Andrews.
R. L. Wilson.
J. B. Saundry.
Edward Sunderland.
T. W. Joy.
F. W. Salzmann.
R. M. Cole.
Edward Colson.
Benjamin Walker.
W. F. Thurston.

Joshua Duke.
Clifford Crewe.
J. F. Codrington.
F. Wallace.
W. A. Hunt.
George Mason.
J. Rigby-Hughes.
J. C. Howell Spencer.

May.

George Vawdrey.
E. S. Pearse.
R. E. Daniel.
John Guy.
E. E. Rastrick.
L. C. A. Carré.
G. W. Brumwell.
G. R. Nunn.
F. Vinay Cardozo.
David Owen Fountaine.
William Turner.
A. C. Taylor.
J. G. Hurford.
J. A. Bevan.
R. Harris.
H. E. Walker.
John Carr.
A. H. Morrill.
Charles R. Brown.
Frederick Knowles.
William Jebson Stothard.
Geo. W. Smith.

July.

Charles Lewis.
A. R. S. Perkins.
T. W. Evans.
M. O. Coleman.
W. Field Flowers, B.A.
J. D. Mason.
Thomas Brockwell.
Lewis Edwardes.
R. J. Shepherd.
A. A. Atkyns.

Apothecaries' Society.

FINAL EXAMINATION FOR LICENTIATESHIP.

September, 1865.

Robert Stuart.

October.

William Harris Butler.

November.

George Rendle.
Algernon Ewen.
William Evatt Wright.

December.

J. S. S. Perkins.
R. W. S. Barraclough.
Henry Denne.
Frederic Edward Manby.

January, 1866.

Benjamin Chasten Gowing.

February.

George Thomas Hankins.
Edward Matthew Owens.

MARCH.

F. Martyn Rickard.
H. Cecil Smith.
Henry Warlow.

APRIL.

James Haslam.

MAY.

James Milward.
Alderson Newsam.

JUNE.

George Spearman.
W. F. Thurston.
E. L. Crowther.
G. J. B. Stevens.
R. R. S. C. C. Lloyd.
E. A. Trimnell.

AUGUST.

Alfred Charlton.
William John
John Henry Croft.
F. W. Salzmann.

FIRST EXAMINATION FOR LICENTIATESHIP.

OCTOBER, 1865.

Frederick Boultbee Lardner.

NOVEMBER.

Herbert William Fagge.

DECEMBER.

Edward Matthew Owens.
William Stanger.
Charles J. W. Meadows.

JANUARY, 1866.

Robert Harman Smith.
William French Thurston.

MARCH.

Benjamin Duke.

APRIL.

E. Noble Edwards.
J. Greaves Wiseman.

MAY.

F. W. Salzmann.

JUNE.

William Bevan Lewis.
George Stokell.
William Jebson Stothard.
Charles Nutt.
J. F. Codrington.
Charles James Worts.

JULY.

Benjamin Walker.
Edward Sunderland.
John Guy.
William James Bennet
John Webb.
Frederick Wallace.

AUGUST.

T. J. Burroughs. | Joshua Duke.
Louis C. A. Carré.

OCTOBER, 1865.

GOLD MEDAL FOR MATERIA MEDICA AND PHARMACEUTICAL CHEMISTRY.

George Rolph Raine.

GOLD MEDAL FOR BOTANY.

Stephen Wootton Bushell, B.Sc.

GUY'S HOSPITAL MEDALLISTS AND PRIZEMEN, 1865-6.

EXAMINATION OF STUDENTS IN MEDICINE, SURGERY, AND THE ALLIED SCIENCES, AUGUST 3RD, 1866.

THIRD YEAR'S STUDENTS.

Benjamin Neale Dalton, South Lambeth, the Treasurer's Gold Medal for Medicine.

Benjamin Neale Dalton, South Lambeth, the Treasurer's Gold Medal for Surgery.

James Gurney Carruthers, Northampton, Proxime Accessit.

Prizes.

Benjamin Neale Dalton, South Lambeth, first Prize, £40.
William John, Haverfordwest, second Prize, £35.
Arthur Bowes Elliott, Richmond, Yorkshire, Honorary Certificate.

SECOND YEAR'S STUDENTS.

William Bevan Lewis, Cardigan, first Prize, £35.
James Frederick Goodhart, Brighton, second Prize, £30.
John F. Codrington, Newcastle, Australia, Honorary Certificate.

FIRST YEAR'S STUDENTS.

William Howard Nicholls, Brighton, first Prize, £30.
William Prior Mallam, Kidlington, Oxon, second Prize, £25.
Augustus Hewitt Aldridge, Shirley House, Dorchester, third Prize, £10 10s. (presented by one of the Governors).
Chas. Dudley Maynard, Hornsey, Honorary Certificate.
Herbert Chabot, Camberwell, Honorary Certificate.

ENTRANCE EXAMINATION IN CLASSICS, MATHEMATICS, &c., OCTOBER, 1865.

Herbert Chabot, Camberwell, first Prize, £25.
Charles Bradley, Liverpool, second prize, £20.
Charles James Oldham, Brighton, third Prize, £15.
William Egerton Saunders, Peckham, Honorary Certificate.

PUPILS' PHYSICAL SOCIETY.

PRIZEMEN.

R. C. Lucas, for his Essay "On Headache," with Reports of Cases, £10.

B. N. Dalton, for his Paper "On Malformations," read before the Society, £10.

Reginald Eager, for his Paper "On Sleep," read before the Society, Special Prize, £5.

GENTLEMEN WHO HAVE HELD HOSPITAL APPOINTMENTS SINCE OCTOBER, 1865.

HOUSE-SURGEONS.

George Eastes.
Edwin Burrell.
G. H. Savage, M.B.
Arthur Taylor.
John Gill.
F. W. Humphreys.

SUPERNUMERARY MEDICAL ASSISTANTS.

OUT-PATIENTS' DEPARTMENT.

Arthur Taylor.
F. W. Humphreys.
A. B. Shepherd, M.A., M.B.
H. G. Howse.

RESIDENT OBSTETRIC CLERKS.

F. W. Humphreys.
Arthur Taylor.
H. C. Hilliard.
Henry Denne.
A. C. Jackson.
F. E. Manby.
F. T. Tayler, B.A., M.B.
J. A. Ball.
H. G. Howse.
S. W. Bushell, B.Sc
R. S. Barraclough.
Clement Palmer.
G. R. Raine.

DRESSERS TO THE SURGEONS.

Reginald Eager.
H. S. Taylor.
S. W. Bushell, B.Sc.
H. G. Howse.
John Gill.
G. J. B. Stevens.
W. G. Palmer.
G. R. Raine.
G. S. Elliston.
E. A. Trimnell.
George Mickley, B.A., M.B.
S. Key Watson.
H. G. Shorter.
Henry Morris, B.A.
R. B. Nowell.
H. G. Budd.
J. S. S. Perkins.
James Riley.
William John.
J. R. Stocker.
Charles Sangster.
A. B. Elliott.
George Rootes.
O. H. Foster, B.A.
Charles Hedley.

CLINICAL CLERKS.

Winter Session, 1865-6.

J. A. Ball.
J. H. Walters.
F. E. Manby.
Henry Morris, B.A.
B. Gowing.
George Mickley, B.A.
Henry Denne.
Algernon Ewen.
W. E. Wright.
J. S. S. Perkins.
Alfred Charlton.
R. B. Nowell.

Summer Session.

G. J. B. Stevens.
A. Harwood.
Reginald Eager.
G. R. Raine.
J. W. Morison.
B. Duke.

DRESSERS IN THE OPHTHALMIC WARDS.

E. F. Turner, M.B.
F. T. Tayler, B.A., M.B.
R. W. S. Barraclough.
H. G. Budd.
A. C. Jackson.
Henry Morris, B.A.
Henry Denne.
John Gill.
H. Taylor.
G. J. Muriel.
W. B. Giles.
George Mickley, B.A., M.B.

ASSISTANT-SURGEONS' DRESSERS, AND DRESSERS IN THE SURGERY.

H. H. Braye.
F. Knowles.
T. A. Buck.
T. B. Dyer.
B. N. Dalton.
A. Newsam.
H. Warlow.
M. O. Coleman.
George Rootes.
O. H. Foster, B.A.
E. N. Edwards.
H. K. King.
J. Gittins.
H. Moon.
E. M. Owens.
R. Morgan.
B. Duke.
F. A. Thomas.
H. Cheesman.
W. Boyle.
William John.
F. W. Fowke.
A. B. Elliott.
H. Maynard.
J. P. Cheetham.
Edward Greaves.
H. W. Fagge.
C. J. W. Meadows.
Charles Nutt.
C. E. Wing.
A. H. Baines.
Edward Colson.
C. W. Chapman.
T. C. H. Spencer.
John Carr.
C. Munden.
B. Walker.
E. Sunderland.
B. Edwards.
C. Crewe.
C. A. Nason.
Inglis Mason.
W. Stanger.
C. Jordison.
J. W. Morison.
W. B. Giles.
W. G. Palmer.
H. S. Taylor.

C. Sangster.
E. Rastrick.
W. F. Thurston.
T. Spurgin.
J. Chester.
W. J. Richards.
W. Greene.
R. Tudge.
C. Eccles.
C. Hedley.
H. Lyne.
T. J. Burroughs.
J. W. Barry.
C. R. Brown.
C. J. Sells.
A. H. Morrill.
J. B. Saundry.
E. L. Crowther
J. Williams.
J. L. Ward.
W. Spratt.
H. P. Banks.
J. Riley.
W. C. Toulmin.
J. Thorne.
J. R. Bosworth.
J. W. Cooke.
B. Norman.
J. Webb.
G. R. Nunn.
F. Wallace.
J. G. Carruthers.
G. Stokell.

AURAL SURGEON'S DRESSERS.

W. Greene.
T. Spurgin.
C. J. W. Meadows.
Charles Nutt.
W. J. Richards.
A. B. Elliott.
W. P. Boyle.
W. J. Bennett.
G. W. Smith.

DENTAL SURGEON'S DRESSERS.

William John.
Benjamin Duke.
H. Moon.
C. Jordison.
H. G. Howse.
F. W. Humphreys.
George Mickley, B.A., M.B.
W. B. Giles.
J. R. Stocker.
J. W. Elliott.
J. A. Ball.
S. W. Bushell, B.Sc.

POST-MORTEM CLERKS.

B. N. Dalton.
W. Spratt.
J. W. Morison.
G. S. Elliston.
Edward Greaves.
Charles Munden.
C. Jordison.
C. Sangster.
C. Higgens.
F. W. Fowke.
O. H. Foster.
G. F. Trotter.
G. Andrews.
R. Harris.
C. E. Wing.
J. W. Barry.
F. Wallace.
A. Williams.

MEDICAL WARD CLERKS.

JUNE—AUGUST.	SEPTEMBER—NOVEMBER.
P. T. Scott.	F. Wallace.
A. Williams.	C. W. Chapman.
R. Rendle.	R. Harris.
W. B. Lewis.	Joshua Duke.
S. S. Brown, B.A.	Johannes De Liefde.
J. A. Bevan.	F. S. Daldy.
H. Airy, B.A.	J. Rigby-Hughes.
B. Edwards.	Arthur Roberts.
J. P. Hartree, B.A.	C. D. Maynard.
J. F. Goodhart.	E. W. Way.
G. T. Willan	W. D. Lovell.
J. G. Hurford.	

SURGICAL WARD CLERKS.

JUNE—AUGUST.	SEPTEMBER—NOVEMBER.
L. C. A. Carré.	A. H. Aldridge.
G. W. Shipman.	A. Roberts.
Joshua Duke.	A. A. Thomas.
J. N. C. D. Colley, B.A.	W. N. Blenkarne.
Samuel Jackson.	Frederic Durham.
D. W. C. Hood.	J. A. Thomson.
A. E. Kynaston.	H. E. Waddy.
Thomas Pink.	E. W. Alabone.
C. D. Maynard.	W. P. Mallam.
Edward Elphick.	Edwin Tipple.
C. T. Brookhouse.	J. A. Bevan.
W. A. D. Fasken.	G. L. Cave.

GUY'S LYING-IN CHARITY.

NUMBER OF CASES OF LABOUR ATTENDED DURING THE YEAR.

1865.	October	155
	November	135
	December	155
1866.	January	132
	February	123
	March	119
	April, May	154
	June	113
	July	123
	August	129
	September	160
	Total	1498

GUY'S HOSPITAL.

1866-67.

THE MEDICAL SESSION

COMMENCES ON THE FIRST OF OCTOBER.

The Introductory Address will be given by the President, The Right Hon. Sir LAURENCE PEEL, on Monday, the First of October, at Two o'clock.

Gentlemen desirous of becoming Students must produce satisfactory testimony as to their Education and Conduct.

Fee for Hospital Practice and Lectures:—First year, £40; second year, £40; and £10 for every succeeding year of attendance. One payment of £100 entitles a Student to a perpetual Ticket. Materials used in practical courses are charged extra.

Ward-Clerks, Post-mortem Clerks, Clinical-Clerks, Dressers, Resident Obstetric-Clerks, and Dressers in the Eye-Wards, are selected from the Students, according to merit. Each Dresser (except those in the Eye-Wards) has the privilege of rooms and commons in the Hospital free of charge for one month of his course. The Obstetric Clerks have the like privileges for two months each—one month as junior, another as senior. Two House-Surgeons are appointed every four months from those Students who have obtained the College Diploma.

The Students are required to conform to the Rules and Regulations for the internal management of the Hospital.

The privileges of a Student will be withdrawn in the event of neglect or misconduct.

Certificates will not be given for Lectures or Practice, unless duly attended.

The Winter Session terminates March 31st.

The Summer Session commences May 1st, and concludes July 31st.

VOLUNTARY EXAMINATIONS

ARE HELD AT FOUR PERIODS OF THE STUDENT'S COURSE, AS FOLLOWS:

FIRST.—At Entrance—in Elementary Classics, Ancient and Modern History, and Mathematics. The Candidate who distinguishes himself most, receives £25; the second Candidate, £20; and the third, £15. The Entrance Examination will commence this year on the 8th October.

SECOND.—At the end of the first Sessional year, in all the Subjects of the first year's Course of Study. Three Prizes, the first of

£30, the second of £25, and a third of £10 10*s*., (presented by one of the Governors) are given according to the respective merits of the first three Candidates.

Third.—At the end of the second Sessional year, in all the Subjects which form the Course of Study up to that time. First Prize, £35; Second Prize, £30.

Fourth.—At the end of the third Sessional year, in all the Subjects of the Curriculum. First Prize, £40; Second Prize, £35.

The above Prizes are not awarded unless the Candidates possess sufficient merit.

Honorary Certificates are given to those Candidates who pass creditable Examinations.

TREASURER'S GOLD MEDALS.

Two Gold Medals are given annually by the Treasurer to the Students who, having completed their third year, most distinguish themselves at Special Examinations in Clinical Medicine, and Clinical Surgery. One Medal is awarded in each Subject.

SINGLE COURSES OF LECTURES

MAY BE ATTENDED ON THE FOLLOWING TERMS:

Anatomy, Physiology, Demonstrations and Dissections, Medicine, Surgery, Chemistry, Midwifery, on the payment of Five Guineas for each Course of Lectures.

Materia Medica, Medical Jurisprudence, Botany, Practical Chemistry, Comparative Anatomy, on the payment of Four Guineas for each Course.

Fee for Attendance on either the Medical or Surgical Practice of the Hospital:—Three Months, Ten Guineas; Six Months, Fifteen Guineas; Perpetual, Twenty-five Guineas.

Several of the Lecturers have vacancies for resident private pupils.

MEDICAL OFFICERS.

Physicians.—G. H. Barlow, M.D.; Owen Rees, M.D., F.R.S.; S. O. Habershon, M.D.

Assistant-Physicians.—S. Wilks, M.D.; F. W. Pavy, M.D., F.R.S.; W. Moxon, M.D.

Surgeons.—Edward Cock, Esq.; J. Hilton, Esq., F.R.S.; J. Birkett, Esq.; A. Poland, Esq.

Assistant-Surgeons.—J. Cooper Forster, Esq.; Thomas Bryant, Esq.; Arthur Durham, Esq.

Obstetric Physician.—HENRY OLDHAM, M.D.
Assistant Obstetric Physician.—J. BRAXTON HICKS, M.D., F.R.S.
Surgeon-Dentist.—J. SALTER, Esq., F.R.S.
Surgeon-Aurist.—JAMES HINTON, Esq.
Eye Infirmary.—*Consulting Surgeon,* JOHN F. FRANCE, Esq.
Surgeon.—A. POLAND, Esq.
Assistant Surgeon.—C. BADER, Esq.
Medical Registrar.—C. HILTON FAGGE, M.D.
Surgical Registrar.—G. EASTES, ESQ.
Apothecary.—JAMES STOCKER, Esq.

LECTURES, &c.

WINTER COURSES.

Medicine.—DR. OWEN REES and DR. WILKS, Mondays, Wednesdays, and Fridays, at three.

Clinical Medicine.—DR. BARLOW, DR. OWEN REES, and DR. HABERSHON.

Surgery.—MR. BIRKETT, and MR. POLAND, Tuesdays, Thursdays, and Saturdays, at half-past three.

Clinical Surgery.—MR. COCK, MR. HILTON, MR. BIRKETT, and MR. POLAND.

Anatomy, Descriptive and Surgical.—MR. COOPER FORSTER, and MR. DURHAM, Tuesdays, Wednesdays, Thursdays, and Fridays, at nine.

Physiology and General Anatomy.—DR. PAVY, Mondays, Wednesdays, and Fridays, at a quarter-past four.

Demonstrations on Anatomy.—MR. BANKART, DR. PYE-SMITH, and MR PHILLIPS, daily.

Demonstrations on Morbid Anatomy.—DR. MOXON, daily, at half-past two.

Clinical Lectures on Midwifery and Diseases of Women.—DR. OLDHAM and DR. HICKS.

Chemistry.—DR. ALFRED S. TAYLOR, Tuesdays, Thursdays, and Saturdays, at eleven.

Experimental Philosophy.—DR. C. HILTON FAGGE, Wednesdays, at twelve.

Lying-in Charity.—DR. OLDHAM and DR. J. BRAXTON HICKS.

Curator of the Museum.—DR. MOXON.

SUMMER COURSES.

Demonstrations on Cutaneous Diseases.—DR. WILKS, Wednesdays, at one.

Materia Medica.—DR. HABERSHON, Tuesdays, Thursdays, and Saturdays, at three.

Clinical Medicine.—DR. WILKS, DR. PAVY, and DR. MOXON.

Clinical Surgery.—Mr. Cooper Forster, Mr. Bryant, and Mr. Durham.

Midwifery.—Dr. Oldham and Dr. Braxton Hicks, Tuesdays, Wednesdays, Thursdays, and Fridays, at a quarter to nine.

Medical Jurisprudence.—Dr. Alfred S. Taylor, Tuesdays, Thursdays, and Saturdays, at ten.

Pathology.—Dr. Moxon, Saturdays, at a quarter to nine.

Ophthalmic Surgery.—Mr. Poland and Mr. Bader, Mondays, at a quarter to nine.

Aural Surgery.—Mr. Hinton.

Dental Surgery.—Mr. Salter.

Comparative Anatomy and Zoology.—Dr. Pye-Smith, Tuesdays and Saturdays, at a quarter-past twelve.

Use of the Microscope.—Mr. Durham, Mondays, at half-past twelve.

Botany.—Mr. Johnson, Tuesdays, Thursdays, and Saturdays, at half-past eleven.

Practical Chemistry.—Dr. Stevenson, Mondays, Wednesdays, and Fridays, ten to one.

Demonstrations on Operative and Manipulative Surgery.—Mr. Bryant, Wednesdays, at three.

The Summer Clinical Course commences in May, and terminates in July.

The Wards are visited by the Physicians and Surgeons on the same days and hours as throughout the Winter Session.

The Registrars, and the Demonstrators in Anatomy and Chemistry, assist Pupils in their Studies, and in preparation for the various Public Examinations.

THE LIBRARY, MUSEUMS, AND MODEL-ROOMS, ARE OPEN DAILY TO THE STUDENTS, FROM NINE O'CLOCK A.M. TILL FIVE O'CLOCK P.M.

Mr. Stocker, *Apothecary to Guy's Hospital, is authorised to enter the Names of Students.*

ASTLEY COOPER PRIZE.

The Ninth Triennial Prize of Three Hundred Pounds,

Under the Will of the late Sir Astley P. Cooper, Bart.,

WILL BE AWARDED TO

THE AUTHOR OF THE BEST ESSAY OR TREATISE

ON THE DISEASE KNOWN AS "PYÆMIA."

The Condition annexed by the Testator is, "That the Essays or "Treatises to be written for such Prize shall contain original experi- "ments and observations, which shall not have been previously pub- "lished, *and that each Essay or Treatise shall* (as far as the subject "shall admit of) ***be illustrated by preparations and by drawings,*** which "preparations and drawings shall be added to the Museum of Guy's "Hospital, and shall, together with the Work itself and the sole and "exclusive interest therein and the copyright thereof, become thence- "forth the property of that Institution, and shall be relinquished and "transferred as such by the successful candidate."

And it is expressly declared in the Will "that no Physician, or "Surgeon, or other officer for the time being, of Guy's Hospital or of "St. Thomas's Hospital, in the Borough of Southwark, nor any person "related by blood or by affinity to any such Physician, or Surgeon, "for the time being, or to any other officer for the time being in "either of the said Hospitals, shall at any time receive or be entitled "to claim the Prize." But, with the exception here referred to, this Prize is open for competition to the whole world.

Candidates are informed that their Essays, either written in the English language, or, if in a Foreign Language, accompanied by an English translation, must be sent to Guy's Hospital on or before January 1st, 1868, addressed to the Physicians and Surgeons of Guy's Hospital.

Each Essay or Treatise must be distinguished by a Motto, and be accompanied by a sealed envelope containing the Name and Address of the Writer. None of the envelopes will be opened, except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations and drawings, will remain at the Museum of Guy's Hospital until claimed by the respective writers or their agents.