

Select clinical cases : including cases of labio-glosso-laryngeal paralysis, exophthalmic goitre, arterial pyaemia, general chronic arteritis, saturnine gout / by Samuel Wilks.

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Select clinical cases.

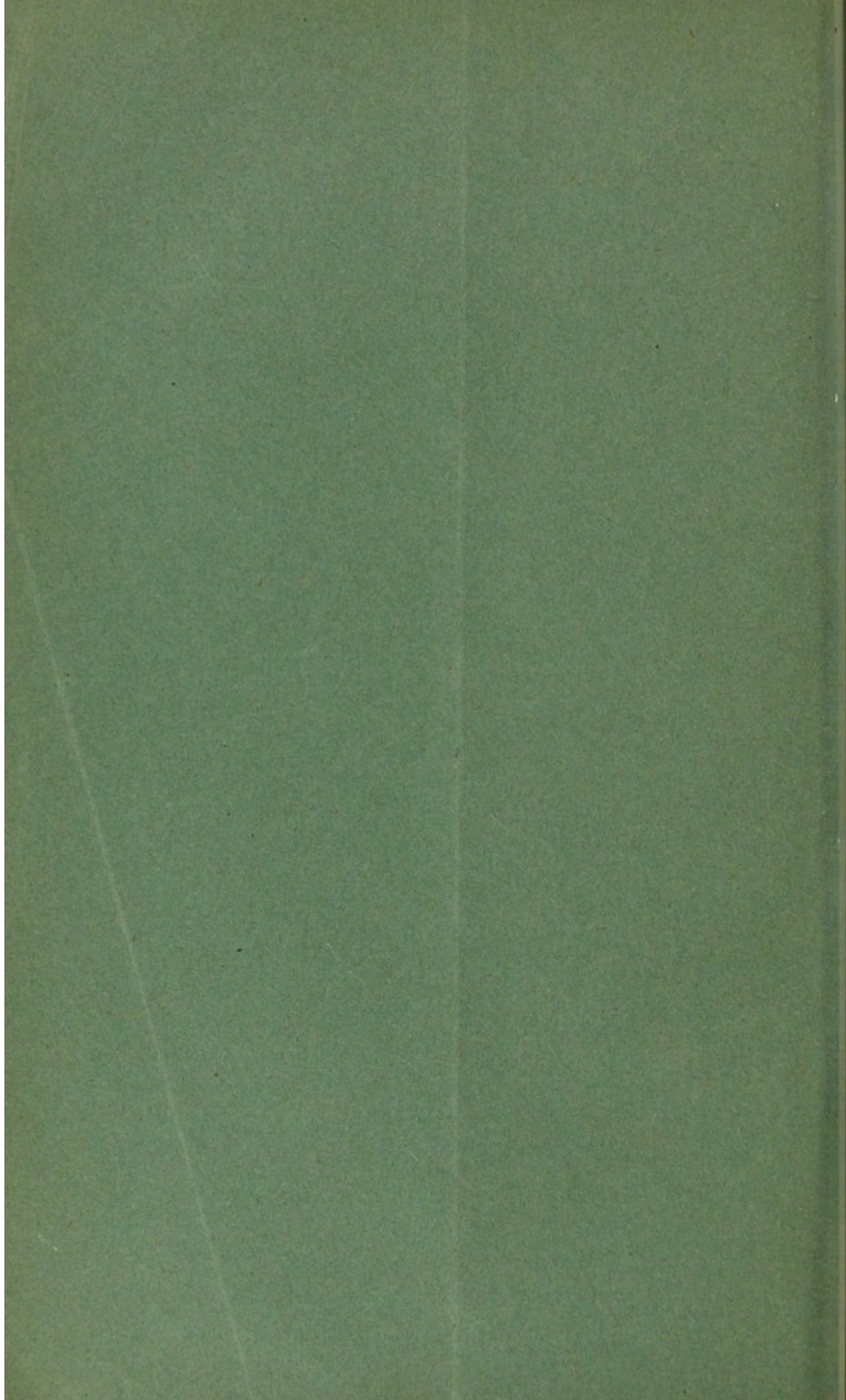
17
From the Guy's Hospital Reports

for 1869-70

With Dr. Wicks'

Kind regards

Dec. 17





SELECT CLINICAL CASES,

INCLUDING CASES OF

LABIO-GLOSSO-LARYNGEAL PARALYSIS; EXOPHTHALMIC GOITRE; ARTERIAL PYÆMIA; GENERAL CHRONIC ARTERITIS; SATURNINE GOUT.

By SAMUEL WILKS, M.D.

IN reviewing the cases that have been in my wards during the last year, I have selected those for record which illustrate some of the maladies that have most recently attracted the attention of the profession; maladies which have now received distinct appellations, and been added to the list of our nosology. It may be remarked how a clinical observation and study of pathology have in many instances enabled us to place together certain morbid conditions which were before isolated, and yet at the same time it must also be remarked how further researches in the wards and post-mortem room have obliged us to recognise fresh groups of ever-recurring symptoms and morbid appearances, compelling us to seek for new names; and, as it were, to frame new diseases. In the past volumes of these Reports there will be found essays by the members of the hospital staff on diseases quite unknown to our forefathers, such as Bright's disease, Addison's disease of the supra-renal capsules, the peculiar affection of the lymphatic glands, which has been styled Hodgkin's disease, lardaceous disease, internal syphilitic affections, besides various essays on newly-described skin diseases. I now offer a few remarks on the subjects of labio-glosso-laryngeal paralysis, exophthalmic goitre, arterial pyæmia, general chronic arteritis, and saturnine gout.

LABIO-GLOSSO-LARYNGEAL PARALYSIS.

I publish a few cases of this form of disease in order to show that those accurate observers, Duchenne and Trousseau, were right in assigning a distinctive name to a malady displaying such distinctive phenomena as this does, being due in all probability to a very limited lesion of the medulla oblongata. The mere concurrence of certain definite symptoms might have been sufficient to warrant the belief that an important anatomical centre had been touched ; but now, since the publication of the lectures of Trousseau, this has been made more than probable by the researches of Mr. Lockhart Clarke, who has demonstrated how certain nerves which are implicated in this form of paralysis are associated at their origins in the medulla ; and all that is wanting to complete the history of the disease is the careful dissection of the nerve centres in a certain number of cases of those who have died of this form of paralysis. The anatomist and the physiologist have in fact informed the clinical physician of the precise spot which is affected, and it only remains for the pathologist to prove it. Of the cases given below only one was fatal, and that was in all probability not a pure and simple example of the disease, and thus the present communication will not afford any great addition to its pathology.

The publication of Trousseau's lectures removes the need of occupying our space with a lengthened description of the disease, which he has styled "*labio-glosso-laryngeal paralysis* ; suffice it to say, that it appears as definite in its characters as does hemiplegia, or any other distinct form of paralysis ; a person indeed may be suddenly seized with a fit, and very shortly display all the symptoms of the disease. It is a paralysis, as the name implies, affecting the lips, mouth, tongue, and larynx ; and therefore, as might be supposed, the functions of eating, swallowing, and talking, are much interfered with ; the nerves known as the seventh, eighth, and ninth, being in part paralysed. Should the affection have come on suddenly, or should it have been developed slowly, the phenomena are the same. These are so striking, that the nature of the case is soon evident. The face has lost its expression from a partial paralysis of the facial nerves, and should the sufferer attempt to speak, it is in

vain, for beyond making a few unintelligible noises, his power of utterance has gone. The reason for this will be found in a weakened condition, not only of the muscles of the face and of the tongue, but of the larynx itself. The lips can be adjusted only for the formation of certain letters, as Trousseau has fully explained; the tongue can be but slightly moved, and cannot be thrust out of the mouth; and when the patient is asked to cough, he produces only the faintest sound in his larynx, not being able to close the organ. At the same time he eats with difficulty; he cannot collect the food in his mouth; he is obliged to assist with his fingers to extract it from his cheeks, and place it at the back of the tongue, when it is swallowed with difficulty. For the same reason the saliva cannot be retained, but is constantly pouring from the mouth; the muscles of the soft palate hang down flabby, so that the posterior nares cannot be closed, and both the velum and larynx may have lost some of their sensibility. The appearance of such a patient is generally very striking and characteristic; he is seen with a pocket handkerchief to the mouth, with a vacant expression, or making a few grotesque movements of the face in the endeavour to force out a word, and with a slate or paper before him on which he writes down all his wants. The speech, it may be remarked, is not merely thick, as in simple facial paralysis, nor is there that meaningless gabble which is heard in the aphasic patient; but it is either utterly gone, or only a syllable in a nasal twang can be produced at a time after violent attempts to set the muscles in motion; there is, in fact, a paralysis of all the parts employed in talking.

It may be remarked that though the capability of speech has entirely gone, from a paralysis of the nerves which supply the muscles employed, yet the trunk of the nerve need not be wholly paralysed, nor have other parts supplied by it lost the whole of their functions. Thus the face may be fallen and the mouth paralysed, but the patient has power to close the eyes, showing that the orbicularis palpebrarum is not affected. In the same way, although the larynx is paralysed for talking, it is unimpaired for breathing. This would show, Trousseau observes, that for its two separate functions, vocalisation and respiration, it must have two nerves, supplied from different sources. Now, the recurrent is almost the sole motor

nerve to the muscles of the larynx, and, consequently, if it is injured or pressed upon the organ is wholly paralysed and the patient is suffocated. It would follow, then, that this nerve is a compound one, and sends a twofold stimulation to the muscles by filaments having their sources in the centres of respiration and vocalisation. Marshall says: "When the roots of the spinal accessory are cut, the operation does not impair any of the respiratory movements, but swallowing is interfered with and the voice ceases, the animal emitting only a bubbling noise. Extirpation of one accessory nerve causes hoarseness. Thus it appears that the spinal accessory governs the momentary and voluntary opening or closure of the glottis and tension of the vocal cords necessary for the production of the voice, or for the exercise of general muscular effort, whilst the respiratory movements of the glottis are under the control of the pneumogastriks." It has long been considered that there is a region in the medulla which may be called the respiratory tract, a region to which branches of all the nerves engaged in the respiratory process may owe their origin; in like manner it would appear that as a large number of parts are engaged in the act of talking, so the nerves supplying them must be stimulated from a common centre, and thus the explanation how so complex a function should suddenly cease from lesion of one small spot. Now, the proof of this lies in the dissections of Mr. Lockhart Clarke, which demonstrate the connection between the facial, vagus, hypoglossal, and laryngeal nerves. The latter are, in fact, branches of the spinal accessory which, joining the pneumogastric, are given off as the recurrent laryngeal motor nerves. The spinal accessory has two origins: the lower from rootlets arising from the antero-lateral substance of the spinal cord and lower part of the medulla, and collected into the external branch to supply the sterno-mastoid and trapezius muscles; the upper from a special nucleus behind the central canal, which, going to form the internal branch, proceeds to the vagus, and is subsequently distributed to the larynx, pharynx, and palate. If, then, the centre whence this proceeds be injured, the larynx loses that power which this nerve had previously induced, that is, there is loss of vocalisation whilst the respiratory power remains. Mr. Lockhart Clarke has shown that there is a close anatomical connection between the nuclei

of the hypoglossal, vagus, spinal accessory, facial, and trigeminal nerves. There is a column of cells forming the nuclei of these nerves which supplies all the parts used in speaking. It may be true that for several other combined movements there may be controlling centres, and thus there has long been a very general belief that the grouping together of several muscles in the limbs for a common movement is due to the stimulation of various filaments of nerves from special centres in the cord.

It appears remarkable that a small area in the medulla oblongata, or even the olivary body, coinciding with a physiological centre as that of articulation, should be picked out to undergo a rapid or slow morbid change. So remarkable is the fact that it might be worthy, in the first place, of inquiry whether or not experience justifies us in declaring that definite parts of the cerebro-spinal centres having special functions are more prone to disease than other portions of the brain and spinal cord taken indifferently; whether, indeed, all parts are not equally liable to inflammation and degeneration, but it is only when certain physiological portions are affected that we are enabled to apply our names, because then the seat of disease has made itself manifest by the implication of nerves whose function is known, whilst in other cases we are content to use such expressions as cerebral or spinal disease. Although I believe this to be to a certain extent true, yet I consider it is proved that those parts of the cerebro-spinal system which have definite physiological properties are more liable to disease than other spots taken indiscriminately; if so it may show, as is most probably the case, that the vascular supply of such physiological centres is accurately defined and circumscribed, or it may be that a centre having a definite function, being the focus of a number of nerve filaments proceeding from it for a special purpose, must soon be affected if any of these filaments be primarily attacked, seeing that morbid processes quickly proceed along given anatomical tracks. If, then, degenerative processes occur in connection with a morbid state of blood-vessels, and if the anatomical supply bears a relation to defined physiological areas, the explanation of such parts being selected for chronic disease is not so difficult; and if again morbid processes proceed rapidly along nerve filaments, we can under-

stand also how parts having intimate relations are concurrently affected. It is a remarkable circumstance that there is no disease of the nervous system, as far as I am aware, which may prove fatal, and even show a well-marked lesion or degenerative change after death, but may have its counterpart in a functional and curable disorder. This must clearly show that, although in actual blood effusions or softenings the seat of disease may appear accidental, these centres must have been affected from a more precise anatomical reason. For example, an effusion of blood in the corpus striatum productive of a hemiplegia may appear to be accidental, but a temporary hemiplegia of a few hours' duration, as seen after an epileptic fit, and due to a derangement of a part having a very limited area, can only be accounted for on the supposition that physiological districts of the brain are anatomically defined. It is the same with diseases of the spinal cord, every variety of which may occur as a temporary disorder. Let it be said, for example, that paralysis of motion and sensation imply definite lesions of the motor and sensory tracts, then the function of these tracts must be in temporary abeyance to account for the paralysis which occurs in those patients who perfectly recover. Again, let it be said that distinct nervous lesions are discovered in cases of progressive muscular atrophy; we must suppose that in those very severe cases which recover these same special parts of the cord were for a time powerless. Or, if in locomotor ataxy it be shown that the posterior columns of the cord have undergone grey degeneration, it must be believed that those same parts are involved in some way in the cases which recover. All these circumstances would show that of the various nervous ailments which we meet with a large number have a determinate and fixed character owing to certain distinct anatomical and physiological centres being involved. In hysteria it is known that every possible nervous disorder may be simulated, and amongst these I have seen a tolerably fair example of labio-glosso-laryngeal paralysis.

It is worthy of note that in the disease especially under consideration the symptoms appeared suddenly in some of the cases, in others they were of slower progress. It is not remarkable that in some cases the motor tracts should be also involved, and that, combined with the symptoms above

mentioned, there should exist also various degrees of paraplegia or paralysis of the limbs.

I have seen several cases of this form of disease in private. In one, an old lady, lately dead, the disease had been progressing for some years; her difficulty of swallowing had been so great, that on one occasion a probang was passed down the throat, in order to see if there was any obstruction. In another case, a woman of middle age, the attack came on suddenly, as one of ordinary hemiplegia. She rapidly recovered the use of her limbs, so as to be able to walk two or three miles daily, but she remained speechless; she could not protrude her tongue, and could scarcely open her mouth; she was fed with a spoon, and the saliva was constantly dribbling from her mouth. In the case of a lady somewhat older, whom I watched for two or three years, the attack came on as a fit during dinner; she fell off her chair, and was taken up to bed; it was found that her senses had not left her, but she was unable to speak. In a day or two she got up, and appeared very well; but she never spoke again, and could not swallow without great difficulty. She subsequently attended to her household affairs, would play cards with the family, and walk three or four miles daily, but she was obliged to communicate all her wants by writing. Her greatest trouble, however, was the inability to hold her saliva, which was constantly dribbling from her mouth. She had finally a fatal apoplectic attack, in which the effused blood ploughed up the pons varolii; but at its lower part there was an old brownish cyst.

In the case of a woman who was under my care in the hospital some years ago with this form of disease, combined with partial paraplegia, she was unfortunately allowed to feed herself, and on one occasion a large piece of meat stuck in her throat and choked her—an accident not unlikely to happen in this disorder.

The first case which I describe, and the only one in which I have been able to obtain a post-mortem examination, was not a simple case of labio-glosso-laryngeal paralysis, but was associated with progressive muscular atrophy, and therefore the appearances described by Dr. Moxon must be taken as exponents of the latter disease as well as the former. On the first visit to such a case it might have appeared to be

simply one of muscular atrophy or Cruveilhier's paralysis, which was now approaching its climax by implication of the muscles of respiration. The history of the case, however, did not bear out this supposition, and, moreover, there was no evidence of wasting of the tongue or muscles of the larynx. Supposing the disease had begun in the medulla, as one of labio-glosso-laryngeal paralysis, and had then spread to the motor strands of the cord and the motor nerves, it would be a good confirmation of the fact that progressive muscular atrophy has a central origin, and is not a local disease.

CASE (from the Report of the Ward Clerk, Mr. Mallam).

Labio-glosso-laryngeal Paralysis, combined with Muscular Atrophy. Atrophy of the Medulla Oblongata with Atrophy and Degeneration of the Spinal Motor Tracts and the Motor Roots of the Nerves.

William C—, æt. 46, admitted under Dr. Wilks, November 9th, 1867, and died December 28th. He was a leather-dresser by trade. Five years ago he was in the hospital for rheumatic fever, since then he has enjoyed good health until June last, when he began to experience some soreness in the throat and difficulty in swallowing. Towards the latter end of September he lost partial use of his hands and legs, the left side being most affected; but he continued at work until three weeks before admission, when he fell down and was unable to rise again. He has gradually been getting worse since.

On admission he was seen to be a short, old-looking man, with his head sunk between his shoulders, and he had a vacant expression of countenance. He was thin, and his muscles flabby. His eyesight had of late become much impaired; but his pupils contracted under the influence of light, and there was no paralysis of the muscles of the eyeball. He had had pain for some time in the course of the fifth nerve, tactile sensibility was good over the face, and the muscles of mastication appeared to act well. There was a want of expression in the face, and although the mouth was not drawn to either side, the orbicularis oris had lost some of its power, as the saliva was constantly running out of the corners of the mouth; the

buccinators appeared quite useless for the purpose of mastication, and he was obliged to press the food out of his cheeks with his fingers whilst eating. He could close the eyes, and the hearing was good. The back of the throat and soft palate appeared sensitive when touched, but the contractility of the latter seemed much impaired. He had much difficulty in swallowing food, and had to wash it down with fluid. He appeared to have lost power over the tongue, being able to move it but slightly. He spoke very indistinctly and thickly, so that his words were scarcely intelligible. It was seen also that he could scarcely move his chest, and that the breathing was mostly diaphragmatic; the chest was resonant, but on auscultation was found to be full of râles. He had great difficulty in expelling the mucus, being quite unable to cough out. As regards his limbs, there was a general deficiency in power, especially on the left side, so that he was scarcely able to support himself, and had very little use of his arms. The muscles at the same time were wasted, as was seen more especially in the arms, the wrists dropped, the fingers were flexed, and the interossei atrophied.

It will be seen that this man was partially paralysed in his limbs, and had almost lost the power of eating, swallowing, talking, or coughing, from paralysis of certain muscles above named. It will also be observed that, besides the labio-glosso-laryngeal paralysis, he had progressive muscular atrophy.

He was ordered to be galvanized with the continuous current every day for a quarter of an hour, one pole to be placed behind the mastoid process on the left side, the other lower down on the spine, and to take quinine mixture. The extensors of both hands were brought into action when either the induced or the continuous current was applied; but as regards the interossei, those of the right side were alone affected.

It appeared as if the galvanism was giving some tone to his muscles, and he expressed himself better, but at the same time it was evident that he was in constant danger of suffocation, from the accumulation of mucus in the air-passages; his slight hacking cough was constant and most distressing, and he would wake up in the night in fear of imminent choking.

He continued on with much the same symptoms, having great trouble in expectorating and difficulty in swallowing, so

that he had to push his food to the back of the mouth. He then began to be troubled with various neuralgic pains in the face, in the eyes, in the throat, and along the arms.

About a month after admission he appeared to have gained some power, he walked in the ward, he could raise the right arm over his head, he could move the tongue better and articulated more distinctly. About this time he had a fall which hurt his head and kept him in bed; after this he became worse, very low-spirited, speech less distinct, and appetite bad. The mucus collected in his chest, and his power of expectoration became lessened, and it was evident that he could not live long, and on Dec. 28, 1867, he died.

Post-mortem examination by Dr. Moxon; external appearances.—Frame short, slight, square built; features short, coarse, square; great wasting, especially of hands, the muscles of which were very deficient; complexion earthy, sallow.

Head and Spine.—There were no nodes, or other signs of disease of the cranial bones. The calvarium was removed, and the occipital part of the bony base cut out; the arches of the vertebræ were removed; and the cranial and spinal dura mater, their contents, and the cervical nerves, to the outer edges of the scaleni, were all removed together; the processes of dura mater in the sella-turcica, and the sphenoidal fissures, only being cut. The brain was tough and hard. There were no signs of formative disease; the changes required to be looked closely for. But on opening up the visceral arachnoid, there was a most obvious atrophy of the roots of the hypoglossal nerve, which had quite lost the natural white opaque appearance of nerves, and were little thin gelatinous threads as they crossed the corpora olivaria. In the same condition were the inner roots of the spinal accessory, and, also, very markedly, the whole of the anterior roots of the spinal nerves, especially the cervical, and least the sacral. The anterior view of the cord was remarkable, the outer aspect was flat, not round; yet it was harder than natural, so that mere flaccidness was not the cause of this; the anterior roots, also, came from a line much nearer the middle line than is natural. On section, the anterior half of the white matter was atrophied; it was white, harder than natural, and on the section it stood out, while the grey matter receded; the latter was larger than natural—it was darker,

containing obvious vessels, and at the lower part of the cervical cord it was double the natural size, and showed a red colour finely mingled with yellowish white: this part, so affected, was not of great length; generally the redness and largeness of the grey part, and the thin hard shell, or coat-like layer of white matter, made the pathological state of the spinal cord. In the medulla oblongata, as seen from the front, nothing diseased was visible, except the state of the nerve roots, as before stated. But on opening up the arachnoid over the fourth ventricle, and drawing down the medulla oblongata to look at the fourth ventricle, there was a very striking diseased appearance, without obvious derangement of anatomical position; there was a red-grey change of the calamus scriptorius, so that the nib of this was quite involved, and from the nib upwards and outwards, for half an inch, there ran this change. The lining membrane of this ventricle and its choroid plexus were of deeper colour than usual.

The natural curves of the spine, backwards in the dorsal, and forwards in the lumbar region, were very much exaggerated, so that the last lumbar vertebra and the promontory of the sacrum obstructed the entrance to the pelvis; the chest was barrel-shaped, and there were two (second and third) right ribs broken, and united at the attachment of the pectoralis minor.

Chest.—The costal cartilages were partially ossified. The right lung was much marked with pigment; the texture coarse, as from atrophic emphysema.

Abdomen.—The viscera were normal, except that the right kidney was moveable. Its position was such that the long axis of the organ was forwards instead of downwards, and it rested below on the colon, so that the colon had a renal flexure instead of a hepatic. Thus, when the abdomen was opened, and the parts as yet undisturbed, the lower end of the kidney projected forwards, below the liver, to the right of the gall-bladder and above the colon; the end of it came by the duodenum forwards, the side of the organ touching the second flexure of that bowel; it was kept in this place by a suspensory ligament of peritoneum, that came from the lower face of the liver, and branched, so that one arm went to the duodenum and the other to the end of the kidney. The

attachment of the omentum to the duodenal end of the stomach and transverse colon was below and internal to the lower end of the kidney, and the colon fitted into the deep recess below it; this recess went almost to the hilum of the organ behind, and so gave the kidney as good a "mesentery" as the ascending and descending colon usually possess, or, indeed, even a better.

Cæcum contracted, appendix free, hanging into the pelvis.

Liver perfect in outline, capsule thin, tissue dark, healthy; half an ounce of dark bile in gall-bladder.

Labio-glosso-laryngeal Paralysis. (Reported by Mr. CHALMERS.)

Dominick K—, æt. 31, residing at Woolwich, was admitted into Stephen Ward, 19th August, 1867. The patient, a brick-layer's labourer, is a single man, and of temperate habits. He always enjoyed good health until the middle of July, when he went to bed well, but was unable to rise the following morning, having lost the use of his legs and arms during the night. His left side was paralysed in a greater degree than the right, and his speech and power of deglutition were also affected. He had been under medical treatment up to the date of admission, and his health had become slightly improved in consequence. He was able to walk without a stick, but with a tottering gait, though he was scarcely able to raise his feet from the floor. He could stand on the right leg without support, but not on the left, and his left knee was stiff.

Mastication and deglutition were difficult, and the tongue was only capable of very slow protrusion and retraction. The upper part of his face was unaffected; he could close his eyes firmly and quickly, but the lower part of his face was almost motionless; he could not whistle, and there was a want of expression in his countenance. His speech was thick, so that it was difficult to understand what he said. Six to eight ounces of saliva flowed daily from his mouth.

His urine passed from him very slowly, and at times he had to wait a few minutes before he could micturate. His urine was not albuminous; his bowels were regular; his tongue was clean, and he was in no pain; his appetite was very good.

August 21st.—He was free from pain. Ordered Mist. Quinæ ter die.

22nd.—The salivary glands were much excited, and fluid was constantly secreted.

26th.—Saliva was much less in quantity, and the patient felt better.

28th to 30th.—He was improving.

September 2nd.—He was stronger, though he did not feel able to walk without a stick. His throat was not sore, but felt somewhat constricted. His speech was somewhat clearer.

7th to 12th.—He had more power in his legs, and was able to get up and down stairs with the aid of a stick.

23rd.—He was able to retain his saliva, but there was very little improvement in his speech.

October 2nd.—There was very little change. His face had more expression in it, he was free from numbness, and his tactile sensibility was perfect.

7th to 26th.—There was very little change. His vocal cords were seen, by the aid of the laryngoscope, to move freely, both during respiration and when he made an effort to utter a sound; but when he tried to cough the vocal cords scarcely moved at all, and he was quite unable to effect his purpose, a slight hacking movement of expiration being all he could accomplish. This was probably due to a loss of co-ordination, as the vocal cords could be moved during speech.

29th to December 12th.—There was very little improvement, though the patient said he felt better, and the lower portion of his face was less paralysed.

December 14th to 26th.—He was a little better, able to speak with a little more clearness, and to swallow with a little less difficulty.

He was readmitted at the beginning of the following year, and remained in about two months, during which time his condition somewhat improved. He could walk about the ward, though dragging his legs, and had some more power in his arms. As regards his speech, he made a great contortion of his face in order to produce a word; but it was more intelligible than heretofore. On examination of the larynx with the speculum the right vocal cord moved slightly, but the left not at all. He seemed to have power over the soft palate to raise it.

Labio-glosso-laryngeal Paralysis.

Mary Jane D—, æt. 51, had been in the hospital on several occasions ; first in 1864, then in 1866, and again in 1867. She was a married woman with a large family. Her history as she endeavoured to relate it by monosyllables and by writing was, that on May 14th, 1864, she went to bed quite well, but awoke early in the morning, finding the right arm quite powerless and the right leg weak ; the speech was somewhat affected, but improved in the course of the day. In four or five months the arm recovered sufficiently to enable her to use her needle, and she remained tolerably well until the following May, when she had another attack, but on this occasion her jaw was almost fixed, so that she had great difficulty in speaking and eating. During the following six weeks a gradually increasing paralysis came over her, affecting all her limbs and her face.

When she was admitted on January 31st, 1866, she was observed to be a thin, short, old-looking woman, having an anxious expression of countenance, and not able to walk with vigour from some weakness of the legs ; the arms were also somewhat weak, but more especially the right. She had almost total loss of utterance, so that on endeavouring to speak she only made some almost unintelligible noises ; the voice was also weak. She had some difficulty in opening the mouth, which was drawn slightly to the left side ; she also had some difficulty in closing the right eye, and the lower lid of the left eye was slightly drawn down ; there was thus a more or less paralysis of all the muscles of the face ; no loss of sensation. For a long time she had been unable to swallow any solid food, and had been living on liquids, always taking care to place everything far back on her tongue. There also appeared to be some paralysis of the right pillar of the fauces ; some dimness of sight ; tremor of lips. She had headache, especially over the forehead. She used a slate and pencil to communicate her wants. Heart and lungs healthy. She remained in hospital until September.

She was admitted under Dr. Wilks, October 30th, 1867, and remained in for six months without much alteration in her condition. She sat in her chair all day long, as she could not walk well ; the arms were also weak, although she was able to

write. She had lost complete power of utterance, and was in the habit of putting down all her wants on a slate. She usually sat with a handkerchief to her mouth to catch the saliva which was constantly dribbling from it.

Labio-glosso-laryngeal Paralysis.

John O—, a young man, admitted under Dr. Wilks' care, Feb., 1869. He was a sailor, and whilst at Singapore had what was believed to be a sunstroke. Thirteen months afterwards, whilst on his voyage homewards, he had a fit, in which he fell down and lost the use of his left side, including the face, but he did not lose consciousness. He was gradually recovering, when six weeks afterwards he had another similar attack. He has been a temperate man, and has never had syphilis. He had partial paraplegia, besides the symptoms of the affection under discussion. He was only just able to stand, and the power in his arms was but slight. When he first came in he was quite unable to articulate, but after a few days he was able to make some noises, which in a few instances could be interpreted into words. His efforts to speak were painful to witness, as very little resulted from them beyond a few meaningless words. Although he was able to make these faint noises he had not the slightest power to cough. He moved his chest well, and inflated his lungs thoroughly, but every now and then he sighed as if there was some restraint upon the respiratory apparatus. Both facial nerves were partially paralysed; he was unable to screw his lips together in order to whistle or to form labial letters; and some others such as 'k,' he was quite unable to pronounce. He swallowed with difficulty, the nurse feeding him with a spoon, and placing the food at the back of the tongue. He slobbered very much, the saliva constantly running from the corner of his mouth. On examining the throat there was no movement of the palate when he drew in his breath. He put out his tongue a little distance and very slowly. His mind was quite clear. Sent to the West of England to his friends.

Labio-glosso-laryngeal Paralysis.

Mr. H—, æt. 27, a fine young man, who had lived rather freely, and might very probably have had constitutional syphilis, was seized with a fit on the night of January 5th, 1869. This appeared to be of the ordinary hemiplegic character, arising from effusion of blood. Feeling ill he attempted to get out of bed, and then he fell, the noise produced arousing those in the house, who found him on the floor and put him to bed, and when I saw him a few hours afterwards he was paralysed on the left side, but was quite conscious; he rapidly recovered, and at the end of a month was able to walk about and return to his employment. He had never, however, completely regained the strength of the arm and leg. On August 3rd he again had a fit, but on this occasion it was of an epileptic nature, and soon afterwards he had another, and then a succession of them for a few hours. In these attacks he struggled violently, but he said never lost his consciousness; and between the paroxysms he talked quite rationally. On the following day he was better and had no more fits, but it was observed that his speech was failing, and at the end of the week he could not utter a word. When I saw him again and during some weeks afterwards (even to the present time), he was the subject of the complaint under consideration in its most marked form. His face had lost somewhat of its usual expression, and when he smiled the mouth on the right side was slightly drawn up, at the same time the lips were well retracted so as to show the teeth, proving that the orbicularis oris still retained much of its power. He could also close his eyes. If asked to speak he opened his mouth and laughed, but could not utter a single word. Not only was he incapable of forming a word with his lips, but his larynx failed to produce the feeblest note. On asking him to cough it was only once that a slight gurgling was made, in all other attempts he could not produce the faintest sound. The saliva was running from his mouth, necessitating the constant use of a handkerchief. When requested to drink, he allowed a good deal of the fluid to escape from his mouth. He was said to have much difficulty in eating and swallowing, the food collecting in his cheeks, and thus it was generally placed far back

on the tongue to enable him to grasp it. He could protrude his tongue a little distance from the mouth, but it was done slowly and with effort. On examining the throat, the velum was seen to hang loosely down. He had no power to raise it, and touching it with the feather of a pen did not excite it to action. He, however, said he could feel it being touched. His intellect was quite clear.

EXOPHTHALMIC GOITRE.

I introduce the subject of exophthalmic goitre mainly for the purpose of enabling me to record a fatal case in which the post-mortem examination was carefully made by Dr. Moxon. The accompanying plate affords a very good representation of the disease, showing the wasted frame, enlarged thyroid gland, and prominent eyeballs. I may just remind the reader that the examination was made more especially to search out a cause for the disease in the nervous system, according to the prevalent theory. It is now many years since Dr. Graves, Dr. Begbie, and others, observed some of the more striking features of this remarkable complaint, but it is only of recent years that the whole combination of symptoms has been recognised. These symptoms, which characterise the disease, are a protruding eyeball, an enlarged thyroid, palpitation of the heart, and a thrill in the blood-vessels; in addition there is a general wasting, great muscular prostration, and a capricious appetite. (In the fatal case to be described there was vomiting occasionally, and at other times the appetite was ravenous.) The heart beats very frequently, perhaps at the rate of 150 per minute; but, as a rule, no abnormal sounds are discoverable. Sometimes a faint systolic infra-mammary bruit, rarely a systolic over the base of the heart, may be discovered, but there is generally no evidence of organic disease. Over the larger vessels there is always a loud "bruit de diable" to be heard, or a loud rushing sound like the roaring of the sea, and at the same time a remarkable thrill given to the finger when placed upon them. The heart, on examination after death, has been found in some cases enlarged, or the left ventricle dilated, but this in all probability has been a result rather than a cause of the disease.

The thyroid, which is of so great a size during life, has been found in some cases simply hypertrophied, in other cases merely enlarged from an increase in the number and size of the blood-vessels. In the same way the eyeballs have become prominent, from the surrounding fat and cellular tissue containing more blood; but in some very chronic cases it has been asserted that there has been a production of new connective tissue.

Virchow has some remarks on this disease in his work on tumours, and in the first place discusses the question as to the priority of its alleged discoverers. He would not with Trousseau style it Graves' disease, thinking that the Germans have an equal right to call it Basedow's disease. Virchow himself styles it "*struma exophthalmica*," and refers to Lebert's scientific title of '*Tachycardia strumosa*.' It may be said that there are authors who claim priority of discovery for Dr. Stokes and Dr. Bégbie. In reference to the condition of the organs, Virchow states that the enlargement of the thyroid is due mainly to an enlargement of blood-vessels, especially of the veins, but that it may go on to a fibrous induration. In nearly all cases he found the heart enlarged, the left ventricle being dilated, but the valves sound. As regards the eyes, the prominence is due to a change in the fatty tissues of the orbit, which may be hypertrophied, but generally the orbital structures are merely in a hyperæmic condition.

The fact that such a remarkable combination of symptoms is repeatedly met with, is sufficient to show that there is some special derangement of the animal machinery, and various have been the theories to account for it. There are those who have been content to attribute the whole of the symptoms to anæmia; and this supposition has been supported by the fact, that the complaint is frequently met with in young girls who are suffering from chlorosis. Constantly a patient who at first sight has simply this malady, is found, besides the palpitation of the heart and throbbing in the vessels, to have some enlargement of the thyroid and prominence of eyeballs. Others have thought that a primary affection of the heart might cause the complaint, and no doubt in many cases as a part of the general congestion, there is great fulness of the blood-vessels, prominence of eyeballs, and enlargement of the neck. Such cases

may, however, only tend to show, that whatever promotes undue activity of the heart and engorgement of vessels, whether from too great retention of blood or want of tone in the vessels, is productive of symptoms similar in kind to those witnessed in exophthalmic goitre. Of late years, and more especially since the functions of the sympathetic have been studied, it has been conjectured that the cause is in the nervous system; it has been proved that one of the functions of the sympathetic is vaso-motor, and that a paralysis of the nerve, by removing the tension-force from the arteries, allows them to expand, and thus the part of the body to which they are distributed becomes more vascular and rises in temperature. It is thus conjectured, that if the cervical sympathetic nerves were paralysed, the carotids and their branches would expand, that they would send more blood into such structures as the eye and thyroid, causing them to enlarge, and that at the same time the vessels would throb and the heart palpitate. In confirmation of the view that the disease is really a neurosis of the cervical sympathetic, it has been stated by Trousseau and others that in some dissections the ganglia have undergone an enlargement, or been indurated by inflammation of the cellular tissue around them, and indeed have been virtually destroyed.

If further observations show that the cervical ganglia are invariably involved, the phenomena will be accounted for in some such way as indicated; but at present it must be remembered, that experimental physiologists have shown no results at all comparable with what is observed in exophthalmic goitre after division of the sympathetic. When Bernard irritated that portion of the cord lying between the sixth cervical and fourth dorsal, and which he styled cilio-spinal, he obtained many of the same effects as when he irritated the sympathetic in the neck, a spasm of vessels and contraction of the pupil; on the other hand, a division of the sympathetic produced increased vascularity on that side, with rise of temperature and dilatation of the pupil; but, as far as I am aware, there were not produced any of the phenomena witnessed in exophthalmic goitre; indeed, with so little accuracy do the results of the physiologists appear to be known, that whilst some authors have considered the disease to be due to a paralysis of the sympathetic,

others have spoken of an increased action of this nerve as an explanation for the pulsating vessels, prominent eye, and palpitating heart. Supposing that a paralysis of the sympathetic would account for the dilatation of the vessels in the neck and other phenomena, it would not, according to the experimental physiologists, account for the rapid action of the heart, since they say that a paralysis of the sympathetic would diminish the heart's action, a fact which is corroborated by cases of accidental injury. For example, in a case of fracture of the dorsal spine, I have known the beats of the heart reduced to less than forty, an effect produced through the communications of the spinal nerves with the sympathetic.

Physiologists say, indeed, that the heart is stimulated to action by the sympathetic, and that if this nerve be paralysed the heart would soon cease to beat; this they say may also be brought about by the action of the pneumogastric nerve, which has a regulating or even retarding influence over the heart's action. Thus, to quote Marshall, "In a beheaded criminal electric shocks applied to the pneumogastric nerve suddenly stopped the heart's action, whilst stimulation of the sympathetic re-excited the movements; hence that the vagus exercises an inhibitory action, &c. Galvanism applied to the cervical part of sympathetic, or branch connecting it with the cervical cord, produces remarkable acceleration of the heart's beats. At the same time, when the experiment is performed in the rabbit, it causes dilatation of the blood-vessels, blood accumulates in them and they become dark, and at the same time there is increased temperature with perspiration."¹ Whilst then physiologists maintain that the effects of a destruction of the sympathetic is to paralyse the heart, we cannot assume that such a paralysis has taken place in exophthalmic goitre, where different phenomena obtain; nor, indeed, the opposite opinion that the nerve is over active, for if so it would diminish the calibre of the vessels of the neck and head, and retard the circulation. Whilst, therefore, we may be looking for a nervous cause, we cannot admit in theory that a destruction of the

¹ A remarkable case of a lad was shown to me, by Mr. Cockell, of Dalston, who was accustomed to perspire on one side of the upper part of his body. No cause for it was discoverable, but some months afterwards it was observed that his back was growing out.

sympathetic is that one; if, however, observation shows the contrary, the physiologist must be at fault.

In reference to any other cause productive of want of tone in the vessels being sufficient to account for the symptoms, one cannot but observe the great approach to exophthalmic goitre in many cases of chlorosis; in some young girls this is so marked that it becomes a question as to what is the nature of the malady. Thus, during the last year I have seen three young ladies in whom the difficulty of the diagnosis was very considerable; one young girl had been ill nearly two years; she was anæmic, had short breath, and had all the usual symptoms of chlorosis; the usual remedies, however, had failed to cure her, and she sought further advice. On examining her I found her thin, with some enlargement of the thyroid and violent palpitations of the heart, but no protrusion of the eyeball. I confessed to considerable doubt as to the nature of the case. A very similar one, but more marked, I have quite recently seen. This young lady had been treated as if suffering from ordinary chlorosis, but without effect. She had had amenorrhœa for eight months, had become thin, and had suffered with palpitation. I found her pulse 156 per minute, no bruit in chest, but very loud in the neck; the thyroid enlarged and the eyeballs prominent; the condition of the eyes had been observed by the friends. In this case, although all the ordinary symptoms of chlorosis existed to a casual observer, yet on closer examination they were found to resemble those rather of exophthalmic goitre. The question then arises whether, in speaking of these two affections, we are dealing with two diseases which merely have some symptoms in common, or whether they have pathologically a much closer intimacy. One may observe that in cases of ordinary chlorosis, the patient is often very anæmic, but showing no great loss of flesh, and that a cardiac anæmic bruit exists; in the cases under discussion the patient is thin, not necessarily very pale, and the bruit at base of heart is often wanting; that is, no abnormal sound is produced at the origin of the larger vessels from impoverishment of the blood, whilst local bruits exist in the smaller vessels, as the carotids. The failure of the usual ferruginous remedies also, is a test of the nature of the complaint. Thus a girl of 17, who came to me with amenorrhœa, eyes somewhat prominent and thyroid en-

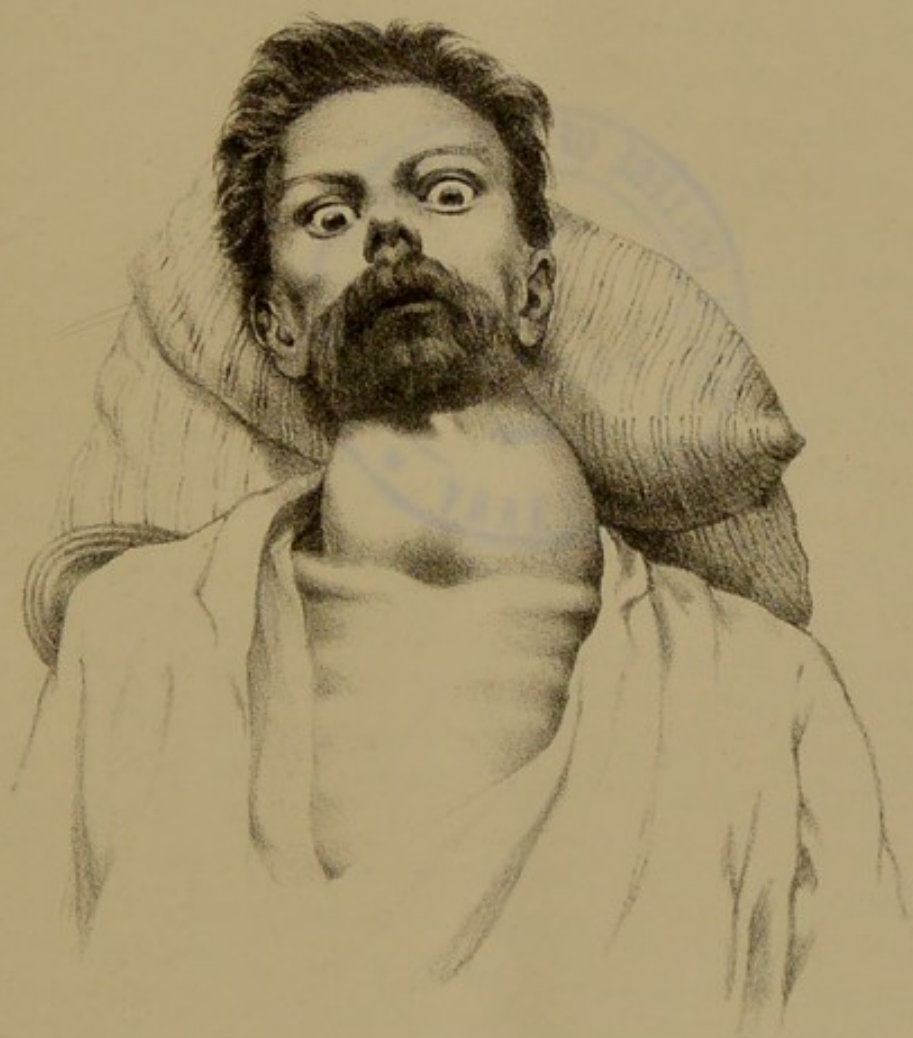
larged, and with heart beating at rate of 150 per minute, but with no bruit, had obtained no good after three months' treatment by iron. It is remarkable, however, that there should be proneness to the complaint in young girls. Thus a child of 13, whom I have seen several times, and who probably is now dead, had the symptoms in a most marked degree. She was so ill on her first visit to me that she could scarcely walk into my study. She was greatly emaciated; her eyes were starting out of her head, her thyroid was much enlarged, the heart was beating 168 per minute, and there was a most remarkable thrill in the vessels of the neck when the finger was placed upon them. She had also stridulous breathing.

Exophthalmic Goitre. (Reported by Mr. LACEY.)

Charles H—, æt. 26, admitted under Dr. Wilks, October 24th, 1868. His father, mother, and all his brothers and sisters were alive and well; but it was said that an uncle and a brother had suffered from ordinary goitre. The patient had always lived in or near London, and of late years at Dalston, where the New River water is drunk.

Three months since he observed that his throat was growing full, and as it rapidly increased he sought medical aid from Mr. Cockell. Under the administration of Pot. Iodid. gr. v and Tr. Iodi $\mathfrak{m}\mathfrak{x}$ internally, and the application of Tr. Iodi locally, the growth was stayed for a time, in fact it slightly decreased. His eyes at this time were observed to be slightly protuberant. The improvement was only temporary, for he soon rapidly grew worse, became very thin, and was often sick; his appetite, however, returned.

On admission he was seen to afford a striking example of the disease; he was extremely emaciated, and so feeble as to be obliged to keep his bed, and to be scarcely able to raise himself into a sitting posture. The wasting appeared to affect more especially the muscles; the temporal muscle was exceedingly atrophied, displaying the malar bones in all their outlines. The muscles of limbs also were extremely atrophied. At the same time his appetite was very good, and at times even voracious, so that whilst he could scarcely support himself on his arm in bed, he would rapidly consume the whole of the dinner





put before him. The most striking features in the case were the enlarged neck and prominent eye, which made his appearance most remarkable. His eyes seemed as if starting out of his head, a large part of the globe being visible. The thyroid formed a large tumour on his neck, projecting more forward than his chin; its division into three lobes was most marked, the left was the largest, the thyroid cartilage still seen above the middle one. The neck measured from top of sternum to Pomum Adami four inches, and around the neck nearly eighteen inches.

The heart was beating 152 per minute, the action regular, no distinct bruit, but the systolic sound was murmurish, and no anæmic bruit to be heard in the chest. In the neck there was a remarkable thrill felt on placing the finger on the blood-vessels, and more especially over the carotids, particularly on the right side; there was also a "bruit de diable," of a most intense character, the sound reminding the listener of the loud roaring of the sea.

He had a slight hacking cough, of a laryngeal character, and voice somewhat husky, as though the thyroid was exerting pressure upon or actually encroaching on the trachea. Respiration natural, rather quick, 28 per minute. The skin was natural, temperature slightly higher than normal, being 99.2; his bowels were regular, appetite good, some thirst.

Dr. Wilks remarked that the experience of all had showed iodine to be of little use, and ordered him good living with eggs and wine, and Mist. Ferri Co., with Sp. Amm. Arom. ʒss, three times a day.

A daily report was taken after a few days. On October 31st he seemed much better. The throat, on measurement, was somewhat less in circumference, the eyes appeared less prominent, and the pulse 130. He had not vomited since he came in. Temp. 99.3.

November 4th.—Appeared still somewhat better, was able to sit up a little in bed, and the enlargement in the neck was somewhat less.

16th.—The last three days had not felt so well, weaker, and appetite not so ravenous, and once or twice vomited his food.

Dr. Wilks said he would try and see if aconite had any

influence over the heart's action, and ordered him Tr. Aconiti, $\mathfrak{m}\mathfrak{v}$ every four hours.

20th.—He continued the treatment until to-day. Sometimes the pulse was as low as 120, and at others 140. He was able to sit up a little, slept well, and expressed himself as better. As, however, it was clear that the aconite was producing no sensible effect, it was ordered that he should return to the iron, taking this time Ferri Tartarati gr. x, Sp. Amm. Arom. $\mathfrak{z}\mathfrak{ss}$ three times a day.

30th.—His strength was improving, so that he was able to leave his bed, and walk a little in the ward. Pulse 120, when quiet; measurement around the gland in neck fourteen and a half inches, and from sternum to pomum Adami three and three quarter inches; it felt much harder.

December 20th.—Continued much in the same state until present time; was able to rise and walk about the ward, but did not increase in flesh, for he grew, if possible, thinner; when he was walking about his pulse reached 150, when in bed it once descended as low as 108. On this day he complained of cough, and expectorated some bronchial mucus, also had pain in his right arm, so that he was unable to raise it.

29th.—Since the last date he had been growing worse, the cough was distressing him, and much mucus was brought up tinged with blood. The chest was resonant on percussion, and physical signs gave no other indication than those of bronchial disturbance. The respirations were 40. The heart-beats 140. The cough and expectoration continued. He got weaker and weaker, and died on the 31st.

Post-mortem examination.—*External appearances.*—Oval, straight, sharp-featured face; average height; lightish brown hair, and scanty beard of the same colour; grey iris; the body much wasted, equally so in all parts; the face much sunken, the eyeballs prominent, if compared with those of ordinary dead bodies, yet not strikingly so if compared with those of a living person. The neck measured fifteen and a half inches at its root across the thyroid gland; the fulness caused by the size of this was still remarkable. There was a small bed-sore, commencing over the sacrum. There was a slight degree of anasarca near the ankles.

Head.—Cranial bones of natural thickness, arachnoid and

pia mater thicker than natural on the upper surface; few Pacchionian bodies, arteries natural, grey matter pale, ventricles rather large, posterior cornua closed by adhesion; little venous blood in brain, which was very pale.

Spine. — Membranes all quite healthy, one small plate on the posterior surface of the arachnoid; exterior of medulla spinalis, to naked-eye observation, perfectly natural; it was uniformly rather a soft than a hard cord, but not morbidly so.

Pupils natural size; arcus, none.

Cervical glands small; thymus, none.

Heart. — As the body was exceedingly wasted, the heart should have been small, whereas it was of natural size; thus there was comparative hypertrophy, though the size was not greater than natural. Not any clot in right heart; muscular fibre a little fatty; coronary arteries natural.

Vessels. — The superior thyroid arteries, especially the right, were very large, but only in proportion to the size of the gland. Their coats were very healthy. The veins, too, were much larger than natural, yet they were only proportionately enlarged.

Abdomen. — Partial peritoneal adhesions of the liver and omentum. Stomach contracted. Liver small, healthy-looking, 37 ounces, capsule thin, tissue dark, lobulation indistinct, yet apparently healthy; gall-bladder small — it contained two drachms of yellow bile.

Spleen. — Four and a half ounces, firm, but rather flaccid; Malpighian corpuscles rather less visible than usual; practically a healthy spleen.

Kidneys. — Eleven ounces, dark colour, thin capsule, substance remarkably hard; there was some excess of fibrous tissue in them.

Thyroid. — Large, but retaining its natural form and proportions. The right lobe enlarged upwards, the left downwards, the middle part upwards, the left lobe at its lower end went three quarters of an inch below the upper edge of the sternum into the chest. In this situation it lay so as to bend the trachea to the right; it was packed between this and the pleura; and it lay on the spine posteriorly, touching the left side of the œsophagus for a length of three inches. In this

situation it compressed the thoracic duct, between itself and the top of the lung and spine. From the pressure of the trachea out of its natural shape, it is probable that this compression of the thoracic duct was not insignificant in the causation of the wasting and voracity.

The section of the gland showed its texture coarse looking, but not different from that of a healthy gland, except generally in the larger size of the lobules. At one point in the upper part of the left lobe was a simple cyst of the size of a cherry. The cyst had an opaque wall and softish contents, into which some blood had been extravasated.

The *cervical sympathetic* at its lower part was difficult to dissect, being imbedded in fibrous tissue, to which it was adherent. The middle ganglia were absent, the inferior (on both sides, but especially on the right side) were not grey, as they should be, but quite white, and, instead of being separable, and covered with a thin membrane, they were coated with a dense fibrous tissue that prevented one seeing any grey matter; the upper ganglia, also, were white and opaque looking; the same, however, might be said of the thoracic and abdominal ganglia.

The ganglia were reserved for subsequent examination by Dr. Moxon, who has kindly drawn up for me the following statement of the appearances observed:

"The ganglia were hardened in chromic-acid solution, and fine sections were taken both longitudinally and transversely. These sections were mounted in glycerine, and examined with 1.5th inch objective.

"The ganglionic nerve-cells did not show any morbid condition. The large nucleolus, the large nucleus, and the pigmented patch within the cells, were like those in ordinary sympathetic ganglion-cells. The large size of the cell and the pigment capsules of nucleated substance forming a single layer or coat of smaller cells around the great nerve-cells were also natural.

"The nerve-fibrils passing in bundles through the ganglion were quite normal. No instance of granular degeneration could I find in thirteen sections.

"The blood-vessels were permeable throughout and occupied generally with healthy looking blood-corpuscles. It seemed to me that the capillaries were larger than usual.

"The connective substance between the bundles of the nerve-fibres was fibrillar, and, as I believe, was in considerably greater amount than is present in ordinary ganglion-tissue.

"On the other hand, there were no 'granule-cells,' such as we see accompanying so many chronic changes in the substance of nervous centres. And there were no

'colloid' or 'amyloid' corpuscles, such as accompany, generally, those chronic changes (sclerosis) which are not accompanied by 'granule-cells.'

"And thus the microscopic characters of these ganglia, like the macroscopic, only gave us, as signs of disease, an apparent excess of fibrous tissue in the structure of the ganglionic tissue, with an apparent enlargement of the capillary vessels. On the other hand, against this we find the natural minute elements of the ganglion to have a healthy appearance.

"Whether this excess of fibrous tissue be really a diseased state connected with an altered function of the ganglia, or whether it be an unimportant accidental condition in this particular sufferer from exophthalmos, each must judge for himself.

"The presence of an excess of fibrous tissue in the structure of an organ is a common morbid state, and characterises especially those conditions known as cirrhosis. But when a state of cirrhosis exists there is always more or less of another change, viz. the destruction of the functioning elements of the organ, *e.g.* in the liver, of the cells; in the kidney, of the tubes; in the lungs, of the air-vesicles. Now, it is to the destruction of the functioning tissue, rather than to the presence of the excess of fibrous tissue, that the symptoms of cirrhosis are to be ascribed. It is very doubtful whether perfectly formed fibrous tissue, such as exists in these sections of ganglia, would itself be a cause of disturbance. It is complete, and not changing. It amounts to a scar. I am therefore inclined to think that the fibrous tissue in this case is accidental to the real cause of the exophthalmos. This is the more likely, as another observer has discovered a red and swollen state of the ganglia as a cause of exophthalmos. Those who have spent much time in demonstrating anatomy know how much the sympathetic ganglia vary in size and appearance.

"It may, of course, be that these variations are keys to parts of pathology as yet not worked out, and it may equally be that the fibrous state in this case and the swollen state in the other are stages of the same change. But if it be so, we have yet to prove it.

"On the whole, then, I cannot say with confidence that the cervical ganglia in this case were diseased."

Exophthalmic Goitre.

Sarah P—, æt. 36, a widow residing at Reigate, admitted under Dr. Wilks, February 24th, 1869. She stated that six weeks ago her throat began to swell, and at the same time her friends observed that her eyes protruded. She sought medical advice, and was ordered iodide of potassium internally, and tinct. of iodine externally to the throat. She had lately suffered much from palpitation of the heart. On admission, she was seen to present all the usual features of the disease in a moderate degree. The thyroid body was enlarged, measuring three inches across; the eyeballs were markedly protruded, but not excessively so. The heart's action was regular, 125 per minute; no bruit. Murmur in the neck over the blood-vessels. She was ordered Tinct. Ferri Perchlor. $\mathfrak{m}\mathfrak{x}$, Tinct. Digitalis $\mathfrak{m}\mathfrak{x}$, in

water three times a day. At the end of about three weeks she expressed herself as feeling better: the eyes protruded less, and the thyroid was less swollen; the palpitation, however, still existed, and the pulse was much the same. There now appeared a slight systolic bruit over upper part of sternum.

After this and during nearly the whole of the next month she was much worse, owing to the irritability of the stomach, for which bismuth, effervescing, and other usual medicines were prescribed. She would be better for a day or two and then vomit everything she took. In the beginning of May the sickness subsided, the thyroid was much less, and the pulse was often down to 110. At the same time she was very thin and feeble. She left on the 20th, her general state of health not being so good as on admission.

Exophthalmic Goitre. (Reported by Mr. W. T. P. DOUGLAS.)

Jane E—, æt. 39, admitted under Dr. Moxon, September 1st, 1869. Is single, and up to four years ago served as a cook, but was compelled to leave her work in consequence of palpitation of the heart; about the same time her friends noticed that her eyes had become more prominent. For six months previous to the commencement of palpitation, she had suffered with frequent cephalalgia. There is no history of fright.

She has twice been an in-patient of this hospital; on the first occasion, a year ago, in Esther ward under Dr. Habershon; during that time galvanism was used, first to the spine and afterwards locally to the thyroid body. Under this treatment she was relieved. Four months ago she was in a surgical ward for an ulcer on the leg.

Latterly the thyroid gland has increased in size.

She is moderately well nourished, but anæmic; her face has a wild and excited expression, caused principally by the prominence of the eyes.

The thyroid gland is enlarged, the right lobe more so than the left. The circumference of the neck over the centre of the gland is twelve and seven eighths inches. A thrill is communicated to the fingers placed at the side of the swelling, and on auscultation a continuous murmur is heard, together with a bruit synchronous with the beat of the heart.

A blowing systolic murmur is heard over the heart and great vessels, loudest at the sternum in a line with the cartilage of the third rib. There is slight accentuation of the second sound. Pulse 134. Breathing sounds normal; vision occasionally misty; is troubled with sleeplessness; breathing at times difficult; no change of voice; appetite good and at times excessive; no vomiting; tongue clean; bowels regular; no catamenia for two years.

She afterwards came under the care of Dr. Wilks, who ordered her Tinct. Aconiti, which she took for a month. For some days the heart's action was lowered. She is still (Oct., 1869) in the hospital.

CAPILLARY EMBOLISM OR ARTERIAL PYÆMIA.

Did space allow I could report several cases of this disease exemplifying the constitutional symptoms attendant upon the passage of disintegrating fibrin through the system, but I will content myself with reminding the reader of the importance and frequency of the complaint, although it is one which is constantly overlooked. It may be safely said that there has been no more important addition to pathological science than the doctrine of embolism; the facts included in it have been at once recognised by the profession as throwing a light upon cases which before were most obscure. It is remarkable, however, that only one portion of the statements in Dr. Kirkes' paper seems to have been generally apprehended by medical men,—that portion which alludes to the effects of the blocking of a large vessel by a vegetation carried from the heart: such as the plugging of a cerebral vessel, and the attendant paralysis; or the plugging of an artery in a limb, and the consequent gangrene. But there is another, and equally important, part of his paper in which he speaks of the blocking of the smaller vessels in the parenchymatous organs, with constitutional symptoms. The results seen in the organs have long been known and described by Rokitansky under the term capillary phlebitis, but the severe and even fatal symptoms often attendant thereon have not yet been sufficiently recognised. By the term embolism, I say, is generally implied the

case of the blocking of a large vessel and the resulting local symptoms, but under it should be included the equally important and common case of the obstruction of the smaller arteries, with attendant constitutional symptoms. I have for many years been in the habit of insisting upon this both in the post-mortem room and in the wards. Formerly, I was accustomed to show from a strictly pathological point of view how changes occurred in the arterial system analogous to those which take place in the venous:—that as in phlebitis some morbid matters, products of inflammation, being taken up by a vein and carried inwards through the circulation, give rise in the internal organs to depositions of a similar kind in them, so in the arterial system disintegrating fibrin of the blood may be carried from the centre of the circulation to the periphery, and there give rise to further deposits of a like fibrinous matter. Latterly, I have been enabled to show clinically that in both cases there are attendant febrile symptoms with characteristic arthritic pains and occasional rigors; and that just as there is a venous pyæmia having its source on the outside of the body, so there is an arterial pyæmia having its origin within. The term ‘pyæmia’ is, of course, not used in its strict etymological sense; but then it must be remembered, that a rigid application of the word is not required in the more ordinary case of contamination of the venous blood; it is for the objectors to use the term “septicæmia.”

This form of affection, I believe, is far from uncommon, as pointed out in a lecture, of which an extract is given in ‘British Med. Journal’ of March, 1868. That it is overlooked arises from the circumstance that a severe organic disease exists generally at the same time, and that this is considered sufficient to account for the symptoms and death. A patient, for example, is in hospital for valvular disease of the heart arising, perhaps, from rheumatism at some former period; whilst under observation he may have febrile attacks attended by articular pains, but these are regarded as touches of the primary complaint; and when after death the valves are seen covered with vegetations, and the spleen and kidneys full of softening fibrinous masses, these are in no wise regarded as having been instrumental to the fatal event, but as mere accidents of the disease. It must, however, have often, I believe,

occurred to the observer that the derangement of the valvular apparatus was scarcely sufficient to cause death. I can myself recall more than one instance where a patient died several weeks after an endocarditis, and the cause was attributed to heart disease, but where the valves were apparently quite efficient. The cases above all others which afford the most striking examples of arterial pyæmia are those where the endocarditis has left the valves of the heart altogether structurally uninjured, and, therefore, where no mechanical causes resulting from heart derangement can possibly have produced death. Such an instance I mentioned in the lecture above referred to, where a medical man was seized with all the symptoms of pyæmia, in so marked a degree that the only question discussed by his attendants was the probable source of infection. This turned out not to be in the veins at all, but in the arterial system, where the origin of the disintegrating fibrin was found to be an aneurism in the auricle of the heart. Such a case was a simple one of death by arterial pyæmia without any organic lesion. Of course, this constitutional affection may be seen in conjunction with that of local embolism, as in a case I took to the Pathological Society, where a man, besides having in the profunda artery of the leg an embolon threatening gangrene, had articular pains in all the limbs, with febrile symptoms dependent on the circulation of smaller particles of deleterious matter through the system.

The occurrence of fibrinous masses in the solid organs of the body has long been known, and as early as the year 1832 a kidney thus affected is portrayed in these Reports. Rokitansky described the condition under the name of capillary phlebitis, and states his opinion that it is due to some spontaneous disease of the blood. It was mainly to refute this that Dr. Kirkes published his paper, in which he proved that particles of fibrin were carried to a distance from the heart, where they had previously been formed. This constituted Kirkes' originality. I have, however, never given up the opinion that in many cases the deposit may have occurred from a primary change in the blood itself, since often there is no proof of the existence of a primary endocarditis; in fact, there has been reason to believe that in some cases the deposit found on the valves of the heart has occurred simultaneously with the de-

posits in the solid viscera. It must be admitted that if, on post-mortem examination, there be found associated with these deposits in the viscera some vegetations on the cardiac valves, it would be presumptuous to deny that an endocarditis might have been the origin of the whole train of subsequent events; yet, on the other hand, it would be a practical error not to be awake to the possible occurrence of arterial pyæmia, because there is no history of a primary cardiac affection. For my own part, although Kirkes may be right in the main, I think there is every reason to believe that Rokitansky's statement is equally true, that deposits may occur from changes in the blood itself. Practically I am sure the supposition of such an event will enable us to recognise cases otherwise obscure.

The facts, then, are these — there is the simple case of endocarditis, or the case where vegetations are covering chronically diseased valves, and as a result the deposition of fibrinous material in the capillaries of organs and other parts of the body. In such a case the symptoms and death by arterial pyæmia are, I believe, frequent enough, although not so generally recognised as they should be. I would also insist that, irrespective of a history of a primary heart affection, such symptoms of pyæmia should lead us to a careful examination of the heart in all cases, when the existence of a bruit may at once suggest their true nature. Whether in such a case the vegetations on the valves which are productive of the morbid sound existed previous to the formation of deposits elsewhere, and were the source of them, or whether they occurred simultaneously from a blood-change, is a question often as difficult to decide after death as before it. Then, again, we may meet with cases where the patients present all the symptoms of pyæmia, and where, failing to find any source for the blood infection on the surface of the body to contaminate the venous blood, we may conjecture that the pathological processes are going on in the arterial system, even though we fail to detect any morbid sounds in the heart; for a post-mortem examination sometimes shows that the viscera are affected in the manner above stated, when the interior of the heart is altogether healthy.

One reason I have for believing that in many instances the source of infection is not the carrying away of vegetations

from the heart is, that in the obscurer kind of cases the effects are very slow in developing, whereas in the instances where large portions of fibrin are carried away in the stream of blood the effects are more sudden and momentous. In these chronic cases the disintegration of the fibrin is slower, the smaller vessels in the viscera are occluded, and the organs which suffer are other than those most usually selected in marked heart disease. Thus the spleen not only has isolated masses within it, but the whole organ becomes enlarged by the deposition throughout it. In the same way the liver may be enlarged and hardened, and also the lungs. If in such a case the disease of the blood were due, not to some morbid process originating in the fluid itself, but to a change wrought upon it by the lining membrane of the heart, I should conceive that the latter, although in a sufficiently unhealthy state to be competent to effect this change, was not covered with vegetations, so as to give rise to a bruit, or to account directly for the deposits in the organs by simple transmission. In time, of course, such vegetations might arise, and produce a murmur.

In these cases it is probable that the first symptoms which attract attention will be the constitutional ones, and that febrile symptoms and occasional rigors will suggest the existence of ague; in fact I have seen several cases of pyæmia, both venous and arterial, treated for miasmatic fever. After a short time it is possible that the liver and spleen may be felt enlarged, and still the obscurity remain. At a later period a bruit may be heard, which may be styled aortic or mitral, according to position. The febrile symptoms continue, and the patient, perhaps after a protracted illness, dies; the organs are found affected as described, and vegetations on the valves of the heart. There may be appearances suggesting an old cardiac disease, and accounting for the deposition of fibrin; but it is equally probable that there may be nothing in the heart to indicate an older change than that observable in the solid viscera. I have now seen so many instances of this, both in hospital and private practice, that I recognise them as belonging to a class, although I am often unable to state the origin of the blood change.

That endocarditis with vegetations on the valves is not necessarily a primary affection is seen in the fact that it

may result from an ordinary venous pyæmia; for example, a healthy man may fracture his leg so as to necessitate its amputation; after a time he may have pyæmia, and then an endocarditis. In scarlatina, after the usual recovery, a secondary fever of the nature of a pyæmia is often seen, in which pains in the joints occur, and not unfrequently an endocarditis, so that it is not uncommon for an organic disease of the heart to be traced back to an attack of scarlatina. More than this, in post-mortem examinations of children who have died of this disease, and even before the accession of well-marked pyæmic symptoms, fibrinous masses may be found in the spleen and kidneys.

During the last few years, since my attention has been drawn to the subject, I have seen many instances of arterial pyæmia, and my mind reverts to cases occurring at an earlier period, and not at all explicable by the pathological doctrines of that day. In a paper on Pyæmia in Vol. 7 of the present series of these Reports, I relate a few cases where there was no evidence that endocarditis was the primary affection. One was the case of a man who was admitted after discharge from prison; he was extremely low and depressed, and thought to be suffering from fever. He shortly died, when the viscera were found full of masses of softening fibrin, and a vegetation existed on an aortic valve, but when this was removed the endocardium showed no evidence of inflammation. Another case of Dr. Habershon's, which had previously been reported by him in Vol. 5, was that of a woman in a state of extreme poverty. Being very ill she was sent to the hospital as a case of fever; the febrile symptoms were high, the tongue brown, the pulse quick, and there were daily rigors, followed by heat and sweating; no cardiac bruit could be heard. She remained exceedingly ill, and was in that state which is usually called typhoid, when some blebs came out on the skin, and she had pains in all the joints and the rigors continued. On the third day after admission a systolic murmur was heard, she gradually grew more prostrate, became delirious and died. On post-mortem examination the spleen and kidneys were found full of softening masses of fibrin, and there was a slight roughness on the border of the mitral valve, as if vegetations might have once existed there. In some cases, as I have said, the liver and spleen may

be enlarged, and thus the observer's whole attention may be given to the abdomen. I well remember how such a case, when I was a pupil, puzzled Dr. Addison. A woman for three months had had fever with rigors, supposed to be ague, and, with this, enlargement of the liver and spleen. After death the heart was found diseased, although no evidence was given of it during life. In looking through the '*Transactions of the Pathological Society*,' I have no doubt that several cases of enlarged spleen were of the nature indicated, and as early as the year 1851, about the time when Dr. Kirkes was engaged in his researches, a case was brought to the Society by Dr. Hare of a young man, *æt.* 25, who gave as his history that he took cold six weeks before, and that this was followed by pains in the joints, palpitation, and enlargement of the abdomen. When under care he had a large liver, a large spleen, and albuminous urine; also *œdema* of the ankles, and a double murmur over the cardiac valves. After death there were found vegetations on the aortic and mitral valves; the spleen and kidneys were much enlarged, hard, elastic, with a yellowish deposit.

I would say, therefore, that arterial pyæmia is a by no means uncommon affection, and that it is seen frequently in chronic heart disease; but the symptoms are overshadowed by the more severe ones attendant on the valvular imperfection, or, if observed, regarded merely as rheumatic. Also, that it may be often met with where there is no history of a primary heart affection, although an endocarditis at the time of the occurrence of the symptoms may exist. Also that it should be suspected in cases of obscure febrile conditions, especially if accompanied by rigors, and more especially where the liver and spleen have been found to be slowly increasing in size.

GENERAL CHRONIC ARTERITIS.

The following case is offered as a contribution to the subject of arterial disease, a form of malady sufficiently rare to warrant a detailed report. It will be seen that some of the principal arteries in the body were so thickened and obstructed that the term arteritis might correctly be applied to the disease. The great point of interest lies in its cause, in the question why, at a particular time, the arterial system should have commenced

to undergo this change? We naturally suppose the cause to be a general or constitutional one, and thus we reflect necessarily on such diseases as gout, rheumatism, or syphilis, as possibly instrumental in its production. As regards the latter there can be no doubt that the blood-vessels may be affected by the gummatous change, like all other tissues in the body, but in the present case there was no evidence that the patient had ever suffered from the malady. As regards rheumatism, it is an interesting question whether a general arteritis is ever associated with an endocarditis, leaving out the minor question as to whether the serous membrane or the blood is the part which is primarily affected. There are certain facts, such as the occurrence of aneurisms in young persons who have had acute rheumatism, which would encourage the idea of the whole circulating system being sometimes affected in this disease.

CASE.—Joseph J. P—, æt. 38, a cheesemonger, married, and leading a regular life. I was asked to see this man not far from the hospital by Mr. Rendle, on Sept. 8th, 1868. I found him lying in bed excessively ill, complaining of great pain in his limbs, apparently due to some arterial disease. His right hand was cold, and some of the fingers were quite white, and in the condition usually styled dead. I immediately felt for the pulse, but could discern none. I then proceeded to look to the arteries in the body, and was surprised to find the pulsation either extremely feeble or almost deficient. The heart's action was tumultuous, and gave the impression of an organ that was not quite healthy, but no actual bruit was discoverable. On inquiring into the history, and especially as to an attack of rheumatic fever, I could only learn that about five years before he had an illness resembling typhoid fever, which left him with some contraction of the hamstring muscles. He was, however, able to follow a laborious business until about fourteen weeks ago, when he became ill and weak, with violent pains all over him, sometimes in the limbs, sometimes in the chest or abdomen. He was also often sick, and had a cough. After I saw him he got sufficiently better to resume work; but at the end of two months from this time, becoming again worse, he was admitted into the hospital on November 3rd, 1868.

He was then too ill to sit up, was very thin, and complained of pains all over him, especially in the legs. These pains he described as being of the most excruciating character, as running down the limbs, and entirely preventing him sleeping. His feet and hands were warm; but on examination of the arteries, no radial pulse could be felt on the right side; on the left side it was slight. Pulsation in the femoral arteries could sometimes not be felt, at other times very obscurely; the carotid could only be felt beating with the greatest difficulty. The heart was beating quickly and irregularly, and gave the idea of a dilated organ. Its pulsations numbered from 150 to 200. At base of lung some signs of consolidation existed, and there was expectoration of some bloody mucus. There was also some albumen in the urine. The ancles slightly œdematous, the temperature of right hand rather less than that of left.

Considering that mere feebleness of circulation arising from weakness of the heart was not sufficient to account for the symptoms, I believed that the man was suffering from a chronic arteritis, that the vessels were becoming blocked, and the pains in the limbs were thus caused. Soon after admission his appetite improved; he slept better, and he consequently expressed himself as feeling stronger than he had done for some time.

At the end of November he again became worse; the pains in the legs, especially in the left, having become more severe. These were worse of a night, and often obliged the sister of the ward to send for the House-physician. The patient would suddenly wake out of sleep and utter a cry in an almost hysterical manner, and complain bitterly of the awful pains in his limbs; although the legs pained him, they felt numbed or dead. This occurred on several nights, so that Mr. R. Stocker was obliged to administer morphia by subcutaneous injection, after having tried nitrite of amyle and other remedies in vain. As a rule, when the arterial system was examined, no pulsation in the right radial artery could be distinguished, very slight pulsation in the left radial, no pulsation in the left femoral, very slight in the right, none in the tibials, very slight in the carotids. Moving the legs generally aggravated the pain.

During the whole of December and January he remained in bed, and his symptoms were much the same; the pains in the limbs were his great trouble, and to relieve these he had morphia injections. In the beginning of February his appetite failed, and he had frequent sickness; after this he gradually sank, and died on the 13th.

Post-mortem Examination.—External Appearances.—Body rather spare; no dropsy; complexion yellowish; bluish purple coloration of dependent half of body, patched on the sides and abdomen also.

Head.—No nodes; bones of average thickness; arachnoid and pia-mater natural; grey matter of sulcus between second and third left frontal convolutions was in a state of yellow atrophy for the space of half a square inch; about three inches in a line behind this there was a recent softening in the grey matter, also another small one in the grey matter of the right posterior lobe. The deep parts were quite healthy and firm; little venous blood in brain.

Chest.—Costal cartilages ossified; a good deal of adhesion about the bases of the lungs on each side, chiefly at the left side; the lungs generally contracted well and appeared healthy, but at the base of the left was a small cavity of size to hold a small chesnut; about this was a puckered shrunken state which had affected a good deal of the tissue, so that the lower lobe was on this account small.

Heart.—Weight about 16 ounces approximately; hypertrophy not equal to the dilatation; dilatation chiefly of the left ventricle; right side, black loose clot in all cavities; left side, black loose clots; no disease of the right side and no ante-mortem clots. Mitral valve slightly inflamed at its edge. Ventricle, its lower and hinder part, and in less degree the posterior set of muscoli papillares, were in a state of fibroid degeneration, *i. e.* shrunken and converted into a fibrous tissue. The ventricular surface of this tissue was thick, hard, and callous to the depth of about one sixth of an inch or less, varying with the col. carneæ, which were chiefly affected; outside the fibrous layer the tissue sank in the section from being slack; it was also vascular and congested, so as to have somewhat the appearance of cavernous tissue. Here the affected part was tolerably well defined, and had yielded a little, so as to form an aneurism of the heart to

a low degree. The septum had some fibrous patches up its inner face.

Arterial System.—The aorta and arteries of the neck and axilla were about average specimens; they were not perfect, but were elastic and smooth and of natural colour, only flecked and patched here and there with superficial atheromatous degeneration. The right brachial artery was permeable as far as the ulnar, but here it became suddenly occluded, while the rest of the ulnar artery was quite closed up, and reduced to a fibrous cord, in the interior of which a solid cord of the size of a common pin—a mere thread—was present, and fastened to the walls; the artery was thus wholly obsolete.

The right common iliac, and external and internal iliac, arteries were entirely full of old adherent brown clot, so closely adherent that the wall of the vessel would split rather than part from the clot. There was, at the bifurcation of the femoral, a gap in the continuity of the clot; it ended above the bifurcation and began again below the origin of the profunda; and at both the ending and commencing parts the clot was white, and like fibrous tissue, and could not be distinguished from the coats, which here were swollen and atheromatous; the atheroma, indeed, appeared to be here in the clot also, as well as in the arterial coat. This part was narrower than the rest of the vessel. The dorsal arteries of the feet were empty and small, but free in channel; the left femoral was full of clot of the same appearance as that on the right side, from Poupart's ligament downwards, and the lower end of the clot, as on the other side, was abrupt, and seemed to be older, being fibrous and inseparably adherent, and apparently containing atheroma in it. The right femoral vein was also occupied by ante-mortem clot, and its tributaries had firm clots in them.

Abdomen.—Stomach, red within; no p. m. solution; duodenum pink; no ulcers in large intestine; liver 60 oz., thin capsule; structure natural, ramosely injected; half ounce of bile of dark colour in gall bladder. Pancreas healthy. Spleen 3 oz., rather hard, no Malp. corp. There were the signs of a very old embolic patch in it. Supra-renal capsules healthy. Kidneys 10 oz.; natural; the left had in it two large rather old embolic patches, sunken and yellow; also another, out of date, as a deep fibrous scar. Bladder and testes healthy.

A somewhat similar case was in the hospital some years ago under Dr. Barlow, and the patient afterwards dying in St. Bartholomew's, an account of it was published by Mr. Savory in the 'Med.-Chir. Transactions' for the year 1856.

The girl was 22 years of age at the time of her death, and for several years previously had been in delicate health, but there was no history of her ever having suffered from a definite illness. She complained of general weakness, tremors, and pains in the limbs. No pulse could be felt in either arm, there was only a feeble pulsation in the right carotid, and a very doubtful one in the left. She subsequently had necrosis of the parietal bone, and the brain becoming involved, she died with cerebral symptoms. On post-mortem examination the aorta throughout its length, as well as the vessels to the lower extremities, were healthy, but those given off to the head and upper extremities were remarkably diseased. They were extremely thickened, and in some parts obliterated; where this was the case, a slender hard fibrous cord occupied the vessel, and was adherent to it. The thickening was due to the formation of a new connective tissue between the inner and middle coats; this existed in some places as a hard, yellow deposit. This change extended from the innominate artery along all the main vessels of the arm, and along the carotids.

THE ASSOCIATION OF GOUT WITH PLUMBISM.

It has long been observed, according to Garrod in his work 'on Rheumatism and Gout,' that workers in lead are very prone to gout. The association of this disease with such an accidental occurrence as the absorption of lead is so remarkable and so little in accord with what is positively known of the effects of plumbism on the one hand, or the causes of gout on the other, that the fact has been received with incredulity by some, and dismissed at once from their attention by others. Thus, when I have alluded to the circumstance, I have constantly heard it ignored as resting only on an imaginary basis, or what is more usual the observation had not until then been heard of; I therefore make reference to it not with the hope of being able to throw any more light on so obscure a sub-

ject, but simply to endorse by my own experience the statement of those who have long observed an intimate connection between this constitutional disease and lead poisoning. I add, by way of report, two or three cases, but I should have had no difficulty in finding a dozen to confirm the fact. I also allude to it to remark that not only does it appear that persons affected with plumbism are liable to gout, but that they are the subjects of the same pathological changes in the various tissues of the body as occur in sufferers from the latter disease. Thus we see degeneration in the blood-vessels, and a liability to granular disease of the kidneys, as in gouty persons. These facts make the subject one of great interest, and one of larger pathological bearing than the association would at first suggest. For example, if gout be due mainly to hereditary predisposition, we should be forced to admit that the connection of this disease with plumbism is due simply to the increased susceptibility of gouty persons to the mineral, and be forced to the conclusion that lead exerts its deleterious influence only on certain constitutions; whilst in others, of course, it is inert. If, on the other hand, besides considering the influence of hereditary taint, gout is immediately set up by a mal-assimilation which is productive of an excess of uric acid, we should be obliged to admit that not only may want of bodily exercise, and the imbibition of malt liquors and wine be instrumental in this result, but that other agencies may be at work, and amongst these the action of lead. Supposing that this metal, like some other causes, acts by interfering with assimilating processes, and so leading to the formation of excess of uric acid, we may then account for the ordinary arthritic symptoms, diseased kidney, and other morbid conditions. For it may be remarked that the direct effects of lead on the system are not the same as those seen where gout has intervened. Lead simply tends to deteriorate the tissues; the muscles waste; the cerebro-spinal system also atrophies, and the patient becomes cachectic and utterly enfeebled in mind and body.

The well known treatise of Tanquerel des Planches makes no mention of gout, or indeed of any affection of the joints, as a consequence of lead poisoning. He devotes a chapter to saturnine arthralgia, but he intends by this expression to refer

merely to pains in the limbs (*douleurs névralgiques des membres*), which so frequently accompany other symptoms of plumbism. Tanquerel dwells mainly on the effects of lead on the nervous system; and these, indeed, have always been the best observed symptoms from the time of the ancients; tremors, convulsions, and epilepsy having been noted by Hippocrates and Aretæus. In modern times it has been suggested whether the last named symptoms were due to the kidney, and were, in fact, uræmic; to test this question Dr. Rosenstein (*Virchow's 'Archiv'*) performed experiments on dogs by administering to them small doses of acetate of lead, and he found that they soon had convulsions, but in no instance did he find albuminuria, or kidney disease.

In the cases, however, alluded to, where gout has been developed, the tendency to pathological changes has been the same as in true gout. In the example which immediately follows, the combination of effects will be seen.

(Reported by Mr. W. A. Marsh).

CASE.—Edward C—, æt. 44, living at Rotherhithe, admitted under Dr. Wilks, December 2nd, 1868. His occupation was that of a colour grinder, and at this he had been engaged for sixteen years. He was of steady habits. His family history was good. During the time above mentioned, he had suffered from lead poisoning, principally from colic, on six different occasions, and on one he had what he believed to be rheumatic fever. About two years ago he was under Dr. Wilks for dropped wrist, and it was then observed that his muscles were undergoing a progressive muscular atrophy. It appeared that about seven years before his last admission, his hands became affected and became gradually weaker since. He continued on, however, when five weeks ago his legs and abdomen became swollen, and thus eleven days before admission he was obliged to desist.

On admission, he was extremely weak; he could scarcely raise his arms from his side owing to the attenuation of the muscles, those of the forearms and hands being principally affected; the *interossei* being almost gone, and the fingers flexed on themselves. He had a blue line on the gums. The abdo-

men was enlarged, and fluctuated on percussion, the legs and scrotum were also œdematous. The edge of the liver could be felt below the ribs; the breathing was oppressed, and there was much bronchial expectoration. The heart appeared healthy, no bruit being discernible, and the urine was carefully examined without finding any albumen.

There was a long and daily report taken of this case, but it contained nothing of importance to point to the cause of this man's extreme distress and feebleness. The abdomen became more distended and tender, and the breathing much embarrassed; it is probable that the respiratory muscles had somewhat degenerated, and thus the difficulty of breathing which could not be accounted for from disease of the viscera. His hands were now so feeble that he could grasp nothing, and thus could not make use of his upper extremities to assist him in breathing. At the end of January he was tapped, and much relieved by the operation. He afterwards again became much oppressed in breathing, and on February 8th was obliged to leave his bed and sit constantly in a chair. Nearly every day he had paroxysms of difficult breathing, due apparently to want of muscular power. After a time he was unable to sit for any lengthened period, and for some days before his death spent many hours of the day standing. He died at last, February 27th, 1869.

Post-mortem.—*External appearance.*—Flat, square-oval face, with long, irregular features; body very spare—indeed, wasted; slightly sallow complexion; considerable dropsy of the lower extremities, much of the abdomen. A small sore beside the buttock, also another small deep ulcer in the right lumbar region. No scar of syphilitic bubo.

Head.—Cranial bones natural, arachnoid and pia mater not thick, cerebrum rather tough and slack, no signs of old apoplexy, little venous blood in brain.

Chest.—Costal cartilages very much ossified, so that the saw had to be used. Acute recent pleurisy; the usual transversely ridged appearance of the lymph, which was in small quantity. Both pleuræ showed old thickening over the lower lobes, contracting the substance of the lung; there were extensive effusions in both cavities.

The lungs were healthy in texture, except for the com-

pression of the lower lobes; the bronchia were full of pus, of watery appearance, with some froth.

Larynx.—Quite healthy. Cartilages converted into bone.

Heart.—Twenty-one ounces, very much hypertrophied and dilated enough to have held a common left ventricle; no change in pericardium; right side of heart contained tough fibrin in small quantity; left side contained black loose clot; left auricle thick; mitral valve a little thick at the edge, the valve was, however, competent, as tested by tying the aorta and filling the ventricle; aortic valves natural, muscular fibre healthy; coronary arteries thick; all the arteries thick in upper limb.

Pharynx.—Right tonsil hypertrophied; on section it was throughout indurated by fibrous tissue in quantity,—cirrhosis of the tonsil. The left was less affected.

Abdomen.—The peritoneum everywhere thick, and contracted on the viscera; a layer as thick as a sixpence could be raised from the flanks, and left an apparent peritoneum still present on the surface.

Stomach intensely reddened, and coated with a tenacious mucus.

Small intestine as deeply red as the stomach, and in parts ecchymosed, the surface coated with starchy mucus, of yellow colour, much less glairy than that in the stomach. The same state extended through the colon. No ulcers anywhere present.

Liver, 48 oz., rounded in form; the capsule coated with a thick false membrane, with the usual areolated appearance; in front, the false membrane had so shrunk down upon and compressed the liver, that on removing it, the edges of the organ were revealed, folded back most curiously, like leaves folded in a bud.

The gall bladder, also, which was flattened down by the false membrane, on the removal of the thick layer, came up to its natural size and position; it was without gallstones.

Mesenteric glands rather large. Lumbar glands twice the natural size. Spleen, 5 oz.; its capsule moderately thickened, in parts the substance had more fibre than natural, no malformation; corpuscles visible.

Supra-renal capsules healthy; much fat in cortex.

Sympathetic nerve appeared quite healthy. It was white

and large, and connected to parts around by loose connective tissue.

Urinary Organs.—Kidney, 7 oz., dark, finely mingled colour. Cortex wasted; no appearance of structure; some small cysts. Pyramids usual size; many urate of soda grains. Bladder compressed by the thick peritoneum; a small cyst just in place of middle lobe of prostate; the muscular layer was far thicker than natural, to overcome the resistance of the thick peritoneum. Testes both healthy.

Upper Limbs.—The extensor muscles changed to fibrous tissue, of slack consistency and watery appearance; no increase of, or change in, the surrounding atmosphere of cellulosity, which was delicate as usual. The supin. brevis, and the deep extensors, were most diseased; the ex. carp. rad. brev. also very bad, the superficial extensors too, but the ext. minimi digiti less so; the nerves did not appear to be changed, and certainly were very perfect, compared with the muscles; the pronator teres was much changed, and where it adjoins the supin. brevis. The flex. subl. dig. was also changed by a band of fibrosity crossing it at its middle. Under the microscope, the muscles appeared quite converted to fibrous tissue. Gouty deposit in both great-toe-joints, to moderate extent, especially in the synovial membranes, and in the parts corresponding to the sesamoid bones.

CASE.—William M—, æt. 44, admitted into Philip Ward under Dr. Wilks, April 2nd, died April 20th, 1868. He had worked in lead, and had a blue line on the gums. Married. Ill health commenced with gout nine years ago; he had usually three attacks a year. Had never been accustomed to drink much. While in hospital seemed often confused and unable to answer questions coherently. Was pale, and skin waxy. Conjunctivæ were watery; tongue furred; first sound of heart distant. Complained of fixed pain across abdomen. Had a discharge from urethra, rendering micturition difficult and painful; urine albuminous, sp. gr. 10·11. He expectorated a dark, bloody sputum, and was very restless at night. Gout was painful in both feet, afterwards in right wrist. On April 13th he had a well-marked pericardial rub, audible anteriorly and posteriorly. On the 14th he became delirious at night.

On the 18th became very delirious, constantly moving with choreiform movements, for about twenty-four hours before death.

Post-mortem examination.—Good development, nourishment moderate, complexion fair, red hair, very little dropsy of legs, cranial bones rather thick, otherwise healthy; dura mater tough, arachnoid opaque, cerebrum appeared healthy, ventricles of brain had some granules on septum lucidum, very little venous blood in brain, membranes of spinal cord as those of brain, excepting pia mater, which was very much congested. The spinal cord itself contained a spot of ecchymosis in its centre, and the grey matter was much congested, the capillaries being beautifully injected; right lung very œdematous, left bronchus very œdematous, weight of heart twenty-five ounces, left heart very much hypertrophied, right not so much as left, pericardium covered with recent lymph, very little clot in right side of heart, the right auricle dilated, the ventricle dilated and hypertrophied, left side of heart very little clot, left auricle dilated, as well as ventricle very greatly hypertrophied, valves of heart quite healthy, aorta (ascending) not good, loss of elasticity; spleen healthy, weight of kidney six ounces, which was small and granular; the cortex almost all gone, joints of lower limbs filled with gouty concretion.

CASE.—John W—, æt. 48, admitted under Dr. Wilks, February 3rd, 1869. He was an engineer on the South-Western Railway, and worked in lead. He was a temperate man, and there was no hereditary history of gout. For a long time past he had been troubled with what he called rheumatism, which at last obliged him to give up work.

He was a cachectic-looking man, as if he had suffered long from illness; on his right elbow an inflamed bursa, which contained some deposit of urate of soda. Over the metacarpo-phalangeal joint of fore finger is an ulcer, in which is a quantity of mortar-like stuff. His wrist and hip also swollen and tender. Over patella of left knee-joint is also a deposit. A deposit of urate of soda in auricle of right ear. He was in the hospital about six weeks, and got much better. He took during the whole time the Tr. Ferri t. d., and a dose of lithia in the morning.