THE DIAGNOSIS OF ANEURISM OF THE AORTA BY THE AID OF THE LARYNGOSCOPE.

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It is well known that, in cases of aneurism of the arch of the aorta, symptoms referable to the air-passages not unfrequently arise. The diagnosis of these cases is often much facilitated by the use of the laryngoscope. Such a case I saw lately with my friend and former pupil, Dr. Richards; the patient being under the joint care of Drs. Butler and Richards, of Winchester.

A gentleman, Mr. H., aged 33, had noticed occasionally for a year past that, after running hard, his breathing became short, and attended with a noise in the throat. About six weeks before I saw him, while running after a wounded hare, his breathing became so difficult that he could run no longer, and he was nearly suffocated. At the time of his visit to me, on the 30th October, the chief symptoms were cough, stridulous breathing, and occasional difficulty of swallowing. The voice was clear and loud; and I remarked to Dr. Richards, before looking into the larynx, that, if the obstruction which caused the stridor were in the larynx, there would almost of necessity be more or less hoarseness and feebleness of voice. A laryngoscopic examination showed the larynx healthy, except slight congestion and redness of the mucous membrane; and while he drew a deep inspiration through a widely open glottis, as seen in the mirror, we heard loud stridor, evidently originating below the larynx. Then the question arose, Is there an aneurism pressing on the trachea? No unusual pulsation could be felt anywhere. No abnormal sound was heard over the front of the chest; but, on auscultation over the upper dorsal spinous processes, the tracheal stridor was heard there almost as plainly as when the stethoscope was placed over the front of the trachea itself; and the voice in that situation had a loud bronchophonic character. In addition to these physical signs, the respiratory murmur was decidedly more feeble over the right than over the left lung. We came to the conclusion that an aneurism of the transverse aorta, pressing on the trachea, and narrowing the tube, was the cause of the stridor, the occasional dyspnoea, and the dysphagia.

I did not see the patient again; but I learn from Dr. Richards that he became steadily worse for some time, and rapidly so a few days before his death, which occurred exactly three weeks after his visit to me. The difficulty of breathing progressively increased, until it became a constant struggle; there was occasional dysphagia; and he died from apnoea on the 20th November. Dr. Richards has kindly sent me notes of the examination made twenty-four hours after death, and a photograph of the aneurism, which I have had copied upon wood.

At the back of the transverse aorta there was a shallow pouch an inch and a half in diameter; at the upper part of this pouch an oval opening, with smooth rounded margins, and scarcely large enough to admit the tip of the little finger, communicated with an aneurism about the size of a walnut; this tumour pressed backwards on the trachea

just above its bifurcation; the cartilages were eroded; and the posterior wall of the aneurism was mainly composed of mucous membrane, which over the most prominent part of the tumour had an ulcerated opening a quarter of an inch in diameter, so that a clot of fibrine alone inter-

vened between the blood in the aneurism and the air in the trachea. The canal of the trachea was nearly filled by the aneurismal tumour, which, as may be seen in the woodcut, projects more over the right than over the left bronchus, and so explains the comparative feebleness of the respiratory murmur over the right lung. The recurrent nerves were found to have no connexion with the tumour.

The case whose history I have here briefly given, is a type of a class of cases. A similar case is at present under my care in the hospital. The chief features of these cases are, that an aneurism at the back of the transverse aorta presses on the trachea, narrows the canal, and so causes frequent loud cough, dyspnoea, and stridor; the stridor is tracheal, not laryngeal; while the voice remains clear and loud, and the laryngoscope shows the larynx quite normal, or the mucous membrane only slightly congested. The tracheal stridor and the sound of the voice are distinctly heard over the upper dorsal spinous processes, in consequence of the trachea near its bifurcation being pressed backwards against the bodies of the vertebrae, so that the sound is conducted through the bones. The symptoms in these cases may steadily or rapidly grow worse, but are not liable to sudden paroxysmal increase, except after unusual exertion, by which the aneurism becomes distended, and so the pressure on the trachea is temporarily increased.

There is another distinct class of cases in which laryngeal symptoms result from pressure of an aneurism on the pneumogastric nerve or its recurrent branch, the muscles of the larynx being either paralysed or thrown into a state of spasm. I have published a case of this kind in the fifteenth volume of the *Pathological Transactions*, p. 72. In that case, the symptoms bore a striking resemblance to those of laryngitis. It is not only of scientific interest, but also practically important, to distinguish the two classes of cases; and the laryngoscope affords much assistance in the diagnosis. When pressure on the nerves causes spasm or palsy of the laryngeal muscles, the voice is usually feeble and husky. With laryngeal spasm, there will be stridulous breathing, which may come and go quite suddenly, often without obvious external exciting cause, like paroxysms of infantile laryngismus stridulus. In these cases, the stridor is laryngeal, not tracheal. Inspection with the mirror during the paroxysm of spasm shows closure or great narrowing of the glottis, without structural change within the larynx, as in cases of hysterical laryngismus. When the spasm relaxes, the larynx appears quite normal. When pressure on one recurrent nerve has caused unilateral palsy of the laryngeal muscles, one arytenoid cartilage, with its vocal cord, may be seen to remain motionless when vocalisation is attempted; and the voice is feeble and husky. In these cases, the tubular breathing and bronchophony resulting from pressure of the trachea against the vertebrae are absent. Now the chief practical advantage of a correct diagnosis between stridor and dyspnoea the result of direct pressure on the trachea, and stridor from laryngeal spasm, or palsy, the result of pressure of an aneurism on a nerve, is this, that, in the latter class of cases, temporary relief may be afforded, and life prolonged, by tracheotomy; whereas in the former class tracheotomy is obviously quite useless, since the obstruction is below the point where the trachea can be opened. When laryngeal symptoms result from spasm, repeated doses of chloral have a decided influence in lessening the spasmodic tendency. Chloroform inhalation, also, relaxes laryngeal spasm, and therefore it may sometimes be employed as one means of distinguishing spasm of the larynx from a more permanent obstruction in the larynx or trachea.

THE SYNTHESIS OF ACUTE RHEUMATISM.*

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THE lactic acid theory of acute rheumatism has of late somewhat declined in popularity. The arguments of Prout, the experiments of Richardson, and the statistics of the alkaline treatment, have all failed to establish it firmly. Like many other theories resting on surer grounds, it has suffered from the scepticism of the day. The mint water treatment and other similar manifestations of nihilism have served, more or less, to strengthen the bias in favour of doubt, and to increase the number of sceptics. For some years past, I was myself a disbeliever in the lactic acid theory; and it is to lay before the profession the facts which have recently recalled me to my allegiance that this paper is published. These facts, when added to the arguments which have been adduced by many previous writers, and to the experimental proofs which we already possess, will, in my opinion, strengthen

* Read before the Birmingham and Midland Counties Branch.

the evidence which points to lactic acid as the poison of rheumatic fever.

In the *BRITISH MEDICAL JOURNAL* of February 25th, 1871, I read with much interest an account of Dr. Cantani's observations on the lactic acid treatment of diabetes. At that time, I was engaged in completing an inquiry into the effects of different drugs on the sugar-excretion in diabetes. I determined to add one more drug to my list, and to complete my research by observing the effects of lactic acid.

A man (Wright) who had just come into the General Hospital under my care, suffering from diabetes, offered me the opportunity. His age was 31, and he had been ill some four months before his admission. By trade he was an iron-caster, and up to this attack of illness he had been a healthy man, and had never suffered from rheumatism. He was married, and had several strong, healthy children. On a mixed diet, he passed during the first week of his stay in hospital an average of 180 ounces of urine daily, containing 49 grains of sugar in the ounce. On a strictly animal diet, continued two weeks, the sugar fell to an average of 36 grains an ounce, and the urine passed to an average of 116 ounces daily. The skin was dry and branny. The sugar-excretion remained pretty stationary on strict diet, but lung-symptoms began to manifest themselves, and steadily increased.

On March 8th, I ordered the patient fifteen-minim doses of lactic acid dissolved in an ounce of water four times a day. The dose was doubled the next morning, and in the afternoon he complained of acute pains in his joints, and flying pains about his limbs. In the evening, as these pains had increased, the medicine was discontinued by order of the resident medical assistant.

On March 10th, no lactic acid mixture was taken, and the pains gradually ceased.

On March 11th, I saw the case; and, regarding the occurrence of the joint-pains as a mere coincidence, repeated the lactic acid in fifteen-minim doses three times a day. On the evening of the 12th, he again felt pains in his joints; and on the morning of the 13th, "the small joints of the fingers of both hands, the wrists, and, in a less degree, the elbows," were noted by the resident medical assistant, Mr. E. A. Elkington, to have become "red, swollen, and painful." On my visit, I was much struck by the appearance of these joints, which were typical specimens of acute rheumatic arthritis. In the evening, both wrists, the small joints of the fingers, and the elbows were all red, hot, swollen, tender, and painful. The heart-sounds were clear. The temperature in the morning was 100; in the evening, 101 Fahr. He had moderate perspiration. Pulse 90, soft and full. The joints were wrapped in cotton-wool, and the lactic acid was discontinued.

On March 14th, in the morning, there was a decided improvement in all the joints; the swelling had much diminished, but heat and pain were still present. Temperature 100; pulse 84. In the evening, all the small joints of the fingers were much better. The wrists were still affected, and he complained of a good deal of pain in the knees, which had hitherto escaped. The heart-sounds were clear. Pulse 90. Temperature 100.8.

On March 15th, the joints were better. The temperature in the morning was 98.6; in the evening, 99.4.

On March 16th, he said that his arms were quite well; his legs nearly so. He had slept much better.

On March 17th, all pains in the joints were gone. Temperature 98.2. Pulse 72.

During the next twelve days, no lactic acid was administered. The case was put clearly to the man, and, as he had felt benefit from the acid mixture and had passed less urine during its use, he elected to run the risk of acute rheumatism. Accordingly, on March 29th, I prescribed seventy-five minims of lactic acid dissolved in twenty ounces of water. This was to be taken as a drink in the course of the twenty-four hours. During the next five days, no rheumatic symptoms appeared. The pulse rose twelve beats on and after the third day; the temperature, which had been previously elevated by the lung-complications, showed no marked change, but on the fourth and fifth days remained steadily at 99°, instead of varying, as it had done for some time previously. On the morning of the sixth day (April 4th), he complained of having had a bad night from joint-pains, which had disturbed him very much, and which came on suddenly after midnight. On examination, the metacarpo-phalangeal and first phalangeal articulations of the first and second fingers of each hand were found to be red, swollen, hot, and painful; the slightest movement aggravated the pain, and he could not on this account pick up anything with his fingers. The pulse was 102. The temperature, which on the previous evening had been 98.2, had risen to 99.4. The heart-sounds were clear. The acid mixture was stopped, and in the evening the pain in the knuckles was less, and the redness had diminished; they were, however, still stiff. No other joints were affected. Temperature 99.2.

April 5th.—His hands were much better, and, of his own accord, he resumed his lactic acid drink, and took about thirty minims of acid in the course of the forenoon. In the evening, the pains had returned in the knuckles, which were swollen, red, and tender. He discontinued the acid, had a fair night, and on the morning of the 6th, found his hands free from pain. He again resumed the lactic acid, and took up to 4 P.M. the remainder of the bottle, containing about forty-five minims of acid. In the evening, at 9 P.M., the pain and swelling had returned in his knuckles, and his left wrist was also affected. He now gave up the acid for two days, and the joint-symptoms gradually disappeared.

The acid drink was resumed on the 9th, and continued to the 13th, but he only took about thirty-five minims of acid a day. He experienced no inconvenience, except flying pains about his joints, till the night of April 13th, when he was disturbed by severe pain in the right wrist, which was found in the morning to be red, swollen, painful, and hot, and was a typical specimen of a rheumatic joint. Pulse 98, full and soft. There was copious perspiration, of acid reaction. The heart-sounds were clear. The elbows and knees became painful and stiff the next day. The joints were all wrapped in cotton-wool as before; and in the course of four days nothing remained except a little stiffness in the right wrist. After a week's interval, the acid was again taken, with like results.

The man now had gained so much experience as to the first indications of a coming attack in his joints, that he was allowed discretionary power as to the time and manner of taking the mixture. By trying it first in small doses, so as not to take more than twenty minims of acid a day, and stopping it for a day or so whenever the joints threatened, he managed to continue the acid for some weeks. Gradually he increased the dose, as advised, and early in June was able to take from forty to fifty minims daily. During this month, he had two sharp attacks of rheumatism in the hands and wrists. By the end of June he was taking seventy-five minims of acid daily; and on July 6th, this was increased to 100 minims. On the 7th, he began to experience considerable pain and stiffness in his joints, and kept his bed (he had been up daily previously) on account of the pain caused by walking. On the 8th, these symptoms were worse, and in the evening his wrists and elbows were very stiff and painful, but the knees were less so. The temperature had risen to 100.6. The acid was stopped. On the next morning, he was better. Temperature 99. The joints were less painful and stiff; there was no redness and no swelling. On the 10th, he again took the acid, his joints feeling much better, and the temperature being only 98.4. In the course of the day, he took 100 minims of the acid; and by the evening the pains had returned in his wrists, elbows, and knees. Temperature 100.6; pulse 100, full and soft; skin moist and perspiring. On the morning of the 11th, his right wrist was red and swollen; the left less so. The knuckles of his right hand were also red, swollen, and painful. His left knee was red, swollen, and very painful and tender. He complained also of pain in the left side, but the heart-sounds were found to be clear; pulse 88; skin still moist. The mixture, which had been stopped on the previous night, was discontinued till July 17th, by which date all the rheumatic symptoms had subsided. After this the man only remained in hospital seventeen days. During this period, he, of his own desire, resumed the acid drink, and on one occasion took as much as 125 grains of acid in the course of twenty-four hours. During the last fortnight of his stay in hospital, he had no severe pains in his joints, and whenever flying pains warned him, he discontinued the medicine for a day.

While the above case was under my care in the hospital, it so happened that another diabetic patient of mine, in visiting the wards, met Wright and compared notes with him. From him he heard such a favourable report of the acid treatment, that he requested me to order him the same medicine if I thought it suitable. I did so. A drink consisting of seventy-five minims of lactic acid in a pint of water was prescribed. Of this he took daily as much as contained thirty to fifty minims of acid; and on the fourth day he came to me complaining of a sharp pain in his right knee, which rendered the joint stiff, and made walking very painful. He also mentioned that he had less severe pains in his other joints, and expressed his opinion that he had caught a cold, which had produced rheumatism, a disease from which he had never before suffered. There was no swelling or redness of the knee or other joints. His skin, which had hitherto been harsh and dry, was soft and moist. The acid mixture was discontinued, and in two days the pains had entirely ceased. During the next month, he made several attempts to take the acid mixture, but it was always followed in a day or two by pains in the joints. Early in May, he managed to take the mixture for a week, and then was laid up with such severe joint-pains, that I was called to visit him, and found him in bed with

pains in his elbows, shoulders, ankles, and knees, and, as he said, all over him. None of the joints were swollen except the right knee, which was faintly red, decidedly swollen, and very tender and painful. The other joints were simply stiff and painful on movement. The skin was freely perspiring. Pulse 96, full and soft. The acid mixture was stopped, the joints were wrapped in cotton-wool, and alkalies administered. In the course of a week, all the symptoms had disappeared, and the patient was able to walk about and resume his ordinary habits. This patient had never passed more than twenty-four grains of sugar an ounce while under observation. The excretion was generally not over fifteen grains an ounce.

REMARKS.—The above record contains an account of the joint-symptoms which were observed in two cases to follow the administration of lactic acid. In the first case, at least six well marked arthritic attacks occurred; in the second case, under conditions less favourable for observation as to duration of treatment and place, one well marked attack occurred. The phenomena corresponded in all respects to those which are characteristic of acute articular rheumatism. They came on when the acid was taken, and ceased when it was discontinued. When moderate quantities of the acid were tolerated, an increase in the dose was succeeded by the painful inflammation of the joints. Coinciding with the development of the articular affection was the appearance of perspiration, at first only slight, but afterwards, in the more severe attacks, copious and acid.

These facts have dispelled the last lingering doubt in my mind as to the truth of the lactic acid theory of rheumatism. At first I doubted the connexion between the administration of the acid and the production of the rheumatic phenomena. In my scepticism, I regarded it as an accidental combination. The recurrence of the joint-symptoms, however, on March 13th, following distinctly on the repetition of the lactic acid mixture, shook my disbelief. The coincidence of joint-attacks with the use of the drug might occur once, and I thought even a second time; but, when I found it occur over and over again, there was no room left for the hypothesis of coincidence. To refer Wright's attacks to a series of accidental combinations requires, in my opinion, a much livelier faith than to accept the lactic acid theory of acute rheumatism. If to some, Wright's case presents not evidence enough in the beautifully typical character of the artificially produced disease, and in the precision with which it could be manufactured at the will of the experimenter, then the second case comes in to refute any explanation founded on the assumption of an idiosyncrasy on the part of one patient.

In health, no doubt, much larger quantities of lactic acid than any given in my cases would be excreted without producing any perceptible disturbance in the bodily functions. The acid would escape by the skin, the kidneys, or, after oxidation, as carbonic acid and water. It cannot be justly argued that the quantities of acid taken by my patients were too small not to have escaped in this way. The conditions under which the drug was given must be borne in mind. In diabetes we have a state of suboxidation very unfavourable to the conversion by oxidation of new compounds; and in Wright's case this was aggravated by the serious pulmonary complications. Associated with these, there was a dry and branny state of the skin highly unfavourable to the elimination of the lactic acid by one of the common channels. Lastly, the well known persistent acidity of the urine in diabetes points to a pre-existing hyperacidity of the fluids. These considerations are, I think, important, as defining the conditions under which the experiments were made—conditions most favourable to the development of the specific effects of the lactic acid. It was the combination of all these which rendered Wright so susceptible to the action of the drug. By the absence of one of them (the lung-complication), and the minor degree of glycosuria, we may probably explain the slighter susceptibility in the second case. The larger doses of acid which Wright was able to take occasionally, towards the close of his stay in the hospital, find an explanation partly in his more careful management of the remedy, partly in an acquired toleration of it, and partly in the great improvement which occurred under treatment in the state of the respiratory organs and in the sugar-excretion.

I refrain for the present from discussing the bearings of my observations on the therapeutics of rheumatism. The effects of the lactic acid on the excretion of sugar will be considered, with other modes of treatment, in a future paper. In this communication, my object has been to lay before the profession facts which have an important bearing on the origin of a common and serious malady. If, by pointing out the nature of the poison of acute rheumatism, they help in the smallest degree to improve therapeutics, they will not have been observed in vain.

ABSTRACT OF A CLINICAL LECTURE ON A CASE OF RUPTURED OR LACERATED KIDNEY, FROM A RAILWAY ACCIDENT: SUPPOSED FRACTURE OF THE OS PUBIS, AND RECOVERY.

Delivered at St. George's Hospital on October 24th, 1871.

By PRESCOTT HEWETT, F.R.C.S.,

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THE case to which I intend to call your attention to-day, gentlemen, will be of interest, as the injury is not a common one, and not unfrequently terminates fatally. The man is lying in the Oxford Ward; and, from the symptoms which he had, I suppose he has had rupture or laceration of the kidney. He is a fireman on the railway, and while on the line he was caught between two carriages, the buffers compressing the lower part of the belly and pelvis. I mention especially the lower part of the belly and pelvis, because we shall have to determine whether there has been injury of the pelvis. When brought in, he was in a state of collapse, complaining of great pain in the lower part of the belly, and especially in the left loin. Now, in an accident of this kind, it is not easy at first to tell which organ is injured. After awhile, however, if the man pass urine, and you find that there is blood in it, you may fairly suppose that his kidney is lacerated. Well, after this man had been in the hospital some time, he passed blood in his urine; and, as the pain was in the left loin, it was inferred that it was the left kidney which was ruptured. For a day or two he had still more pain, and then there came symptoms of peritonitis, spreading from the left side down into the iliac region. He was admitted on the 27th September, during my absence. On October 2nd I saw him. During these few days he was under the care of Mr. Rouse, and was treated for laceration of the kidney and peritonitis. He was somewhat recovered when I saw him; blood was no longer passed in the urine, and the peritonitis, ending in marked thickening, was subsiding, inasmuch that he was lying perfectly flat on the bed, and his legs were stretched out. You will bear in mind that the mere posture of the patient is an indication of his state, especially when the injury is about the belly, and has led to peritonitis.

Now what had been the treatment in all this mischief? It was that of perfect rest and opium. It was under this treatment that he so much improved. The only thing which I had to do was to carry it on. He went on gradually improving, and within a fortnight the whole of the hardness about the left side of the belly had disappeared; there was not the slightest pain, and the pulse was natural. The opium was gradually left off, and the man was put upon a better diet. He is going out to-morrow, about a month after the accident.

The mischief in this case was evidently in the front part of the kidney. I believe this from the fact of the patient having peritonitis. You will find that I have reason for believing this from the anatomical relation of the kidney—covered in front by the peritoneum, but behind by cellulo-adipose tissue. Had the rupture been behind, diffuse cellular inflammation would have occurred, and not peritonitis.

And now as to the probable injury of the pelvis. About a week ago, the man was allowed to get up and walk about the ward. He did this for a day or two, and then began to complain of pain in the right groin. I made him lie down in bed, and examined him, and I there found an extravasation of blood, which had not existed there before. It was exactly as if he had been bruised. This ecchymosis of the skin did not make its appearance until three weeks after the man had been in the hospital. What did this discoloration of the skin here mean? In the injury he met with, there must have been some extravasation of blood on the right side in the iliac fossa; and I strongly suspect, from the limitation of the ecchymosis, that this man has had a fracture—probably a mere crack—of the horizontal branch of the os pubis. These discolorations of skin, appearing some time after severe injuries, are most useful in the diagnosis of fractures which have not been suspected. I am all the more induced to call your attention particularly to this point, because I find no mention made of the fact that discoloration thus appearing over the sternum may lead to the diagnosis of a fracture of this bone, when there was no direct injury done to this region, but to the spine.

Now with such an accident as extensive laceration of the kidney, is it possible for the patient to recover? I have seen many cases in our wards in which I have had no doubt that there was extensive laceration, and in which recovery took place. This is one of these cases. The man has had laceration of the kidney, if the symptoms indicate anything. He may by and bye have, it is true, some ulterior mischief in this region, but he has recovered meanwhile. I have placed upon the table