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.