ileo-cæcal valve are produced by different agencies, the amount of obstruction greatly varies. Thus in five cases the little finger could be barely passed through the valve; in one this could not be effected; in another the opening was only one quarter of an inch in diameter; in five it only admitted a quill, and in one a needle; in one it was almost completely closed, and in four no opening at all could be discovered. Of course the effects of the constriction also vary, for wherever it was very narrow the small intestines were found to be vastly distended with fluid, the muscular coat of the ileum was hypertrophied, and the results of vascular congestion were seen either in recent extravasations of blood or in the slate colour of the walls of the gut; the colon was usually contracted, although in many cases it contained a small amount of feces. In Case 2 adhesions were found between a coil of the small intestine and the cæcum and between the sigmoid flexure and the rectum, and communications had been established between these portions of the intestinal canal, so that the effects of the distension must have been considerably lessened. I have found three other cases on record in which a similar process had taken place; in one a communication had been established through the appendix vermiformis, which was much dilated, and was adherent to the ileum; in the others intestinal fistulæ had been established between the small and large intestines. Old adhesions have been often met with, and are probably the results, as in Cases 2 and 3, of a previous attack of local peritonitis, or of inflammation excited by the great distension to which the gut had been subjected.

(To be concluded.)

Remarks

ON

LARYNGEAL PARALYSIS AND INEQUALITY OF THE PUPILS, AS TENDING TO AID AND ALSO TO MISLEAD IN THE DIAGNOSIS OF THORACIC ANEURYSM;

WITH SOME OBSERVATIONS ON THE

STATE OF THE PUPIL AFTER DEATH AND THE EFFECT OF ATROPINE.

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THE occurrence of laryngeal symptoms, and particularly of laryngeal paralysis, as also the appearance of inequality in the pupils, have been known for many years as affording important evidence of the presence of aneurysmal and other thoracic tumours. Two patients who were in my wards at the same time afforded illustrations of the value of these signs, and also of the danger of trusting too much to their significance.

In the one case we had a well-marked pulsating tumour in the upper part of the chest, extending into the right side of the neck. The history of the case went back for more than a year. The examination after death showed that the aorta was dilated and that a large aneurysm arose from the arch in front of the origin of the great vessels. The pneumogastric and recurrent nerves on the right side were found considerably stretched over the tumour, and somewhat adherent to its wall. An attempt was made to include the sympathetic in the dissection of the parts removed; but although it was evident from the position of the tumour that it must have been implicated, it was found that it had been partially destroyed in removing the aneurysm and its connexions. The laryngeal symptoms had appeared three months before death; and on admission the right vocal cord was found to be paralysed. During his stay in the ward (six or seven weeks) the dyspnoea was often extreme, and he had had several attacks, almost fatal in character, due to spasm of the glottis, terminating with a crowing inspiration. These attacks were aggravated by the growth of the tumour leading, a few days before death, to displacement of the larynx

and trachea to the left of the middle line. The only point in this part of the history worthy of note is that the tumour and paralysis were on the right side. It would seem from the statements of authors that this symptom is much commoner when the tumour is on the left side. The state of the pupils was noted particularly on admission; they were quite equal. Their size was measured by means of the scale enclosed in Nettleship's pocket case, the round black spots in which are numbered so as to express the diameter in millimetres. Both pupils when shaded were $7\frac{1}{2}$ mm.; when exposed to bright light, $4\frac{1}{2}$ mm. On being measured again, three weeks before death, when a marked inequality had appeared, the figures were: right pupil when shaded, $4\frac{1}{2}$ mm.; when exposed to light, 4 mm.; left pupil when shaded, $6\frac{1}{2}$ mm.; when exposed to light, 5 mm. This inequality persisted to the end of the case, and was frequently verified as essentially the same in degree. The exact condition at the moment of death was not noted, but at the post-mortem examination, thirty-six hours later, I found both pupils dilated and quite equal. This dilated and equal condition of the pupils after death, where myosis dependent upon aneurysm was present during life, was noticed in the earliest cases recorded of this symptom.¹ This condition of the pupils after death in cases of paralysis from aneurysm is just what might be anticipated; for such tumours do not usually destroy the nerves implicated, and so when relief to the aneurysmal pressure occurs at the time of death the paralytic condition might pass away at the end; and, further, after death the sphincter of the pupil, like other muscles, becomes no doubt relaxed; and so the condition of the pupil should not be different from what is observed in other cases immediately after death occurs. Some observations have been made in my wards on this point, for which I am indebted to the sister of my wards, on whose care in such a matter full reliance can be placed; and although not sufficiently numerous to warrant an authoritative statement on the subject, I may say that if observed *immediately* after death, the pupils were usually found moderately dilated, or sometimes even very markedly so, from $6\frac{1}{2}$ mm. to $8\frac{1}{2}$ mm. being common; none were noted under 5 mm., and once even 10 mm. was found. No doubt the influence of age, the nature of the illness, and other conditions affect the state of the pupils at the time of death. But on pursuing the inquiry after the removal of the body to the dead-house, my assistant, Dr. Williams, and I found that the pupils often changed their size shortly after death, and speaking only from a few cases, I believe there is a marked tendency towards contraction, so that in a young child the reduction found was from $7\frac{1}{2}$ mm. to 4 mm. within sixteen hours, some distinct diminution in size being found in various other cases. In the case of a person recently dead, the influence of atropine in causing very marked dilatation of the pupil is readily shown² by putting a few drops of a strong solution of atropine into one eye and none into the other, when a comparison can be made in an hour or two. Sometimes the effect of the atropine appears just to be sufficient to neutralise the tendency to contraction as manifested in the diminution of the other pupil. Even when put in immediately after death, we did not see the *extreme* dilatation so often produced by strong applications during life. If some hours be allowed to elapse after death before atropine is added, the effect is less pronounced; and it seemed as if after four or six³ hours the susceptibility to atropine was almost lost. The atropine, however, may continue to act even after well-marked rigor mortis has been established. This was a point to which I directed attention, as it seemed to me that the progressive contraction of the pupil after death was concurrent with, and might be dependent on, the development of rigor mortis. Even, however, when the action of the atropine is well marked on the dead eye, its influence is short-lived. This may account for its not producing the extreme dilatation so often seen during life. Within a few hours of its production, say

¹ Dr. W. T. Gairdner. Clinical Medicine, Edin., 1862, p. 534. The case was observed in 1855. See also a case observed by Dr. Walshe in 1853, mentioned in a footnote to his Diseases of the Heart, 4th edition, London, 1873, p. 496.

² Curiously enough, the effect of the instillation of atropine has actually been proposed as a test of death (!), but the dilatation of the pupil in dead eyes, and even in those which have been removed, seems to be well known. See Dr. Argyll Robertson's interesting paper in the Edin. Med. Journal, Feb. 1869, vol. xiv., p. 703.

³ This I find is the average time named by Maschka, within which marked post-mortem changes can be recognised in the eye. Handbuch der gerichtlichen Medicin, Band iii., S. 308. Tübingen, 1881-82. ■

twenty-four hours after death, we usually found the dilated pupil had become reduced to the size of the other; this of course presents a strong contrast to the persistence of the dilatation of the pupil for some days with which we are familiar during life. These facts are of course open to various interpretations, but they seem to me, particularly the one last mentioned, to lend some support to the theory of the dilatation of the pupil from atropine being due not only to its paralysing the sphincter, but also to a direct stimulation of the dilator of the pupil.⁴

In leaving this interesting subject, I may say that I am aware that my observations are too few to warrant a conclusive statement of the case, and as my opportunities for following up such an inquiry are not likely to enable me to do so myself I have requested one of my former assistants to study the behaviour of the pupil after death, and he has already made some progress in the inquiry.

In the second case, where the signs of laryngeal paralysis and inequality of the pupils helped to mislead us, there were very obvious signs and symptoms of cardiac disease, great dyspnoea, enlargement of the heart, and a double murmur, audible down the sternum. But there was also evidence of dilated aorta, as shown by an area of undue dulness on percussion extending from the upper boundary of the heart towards the right sterno-clavicular articulation, and also by an undue pulsation in the jugular fossa. Moreover, we had at times a very distinct sense of impulse immediately under the left clavicle. There was also substernal pain, and this extended down the left arm, but there was no swelling. The pulses were throughout quite equal, and the tracings had a well-marked aortic regurgitant character. Frequently, also, we had, from the time of admission, a slight inequality of the pupils; this was not persistent, but when present the left pupil was always the larger of the two. These facts led me to suspect, almost from the first, the presence of an aneurysm occupying chiefly the descending part of the arch of the aorta, acting on the sympathetic by causing irritation and so leading to dilatation of the left pupil. The patient, a man of forty-two years, had an old history of rheumatism, but not of syphilis. The total duration of his illness from first to last was fully two years; he was in the wards on three separate occasions, and the observation of the case extended over a period of thirteen months, during the whole of which time the suspicion of aneurysm was entertained. The symptoms varied in degree, but remained much the same in kind. Repeatedly pain and friction in the left side indicated the presence of a pleurisy: towards the end of the case a little dropsy, with slight albuminuria, supervened; and the termination was marked by hæmoptysis lasting a few days. The physical signs always pointed to an enlarged aorta, but there was no pulsation over this area, and the pulsation felt under the left clavicle on the first admission ceased to be perceptible; the pulsation in the jugular notch became much less, and the murmurs shifted their position of maximum intensity, so that the diastolic murmur was heard best to the left of the cardiac dulness instead of over the sternum, as before. The pupils continued all through the three residences to show at times a slight inequality, the left being the larger. With these changes in the physical signs, without anything more than a temporary amelioration, the idea of aneurysm was still entertained, and it was supposed that it might have grown downward or backward, so as to remove it from, rather than approximate it to, the surface within reach. The lungs remained tolerably free from signs of any serious change; there had been the repeated pleurisy on the left side, there were frequently some bronchial râles, and towards the end the left lung seemed more implicated. No marked dulness and no great enfeeblement of the respiration were detected. About two months before death the patient thought he had contracted a cold, and there was more cough. This was noticed to have a laryngeal quality, and after the first violence of the cough subsided a laryngoscopic examination showed the left vocal cord to be paralysed. The larynx had been examined soon after his first admission, and found normal. This new symptom, still pointing to the left side, seemed to remove all the hesitation naturally felt up to this point in the diagnosis of aneurysm; this was now regarded as well established. The laryngeal symptoms persisted to the end, but examination of the parts by the mirror was only practised twice or thrice, owing to his weak condition.

⁴ This opinion as to the action of atropine on the living eye is supported by the authority of von Graefe. See Dr. Argyll Robertson's paper already quoted, *Edin. Med. Journal*, vol. xiv., p. 705.

The post-mortem examination was made by Dr. Newman. The aneurysm so confidently looked for on the left side did not exist, there was, indeed, a dilated aorta, and even the beginning of a small pouching towards the right side just above the valves; this might, no doubt, have developed into a regular aneurysm, if the patient had lived, but it had evidently nothing to do with the laryngeal paralysis on the left side. The aortic valves were quite incompetent, much diseased, and very hard and calcareous; this no doubt accounted for the murmurs; the heart was enormously enlarged. The cause of the left-sided laryngeal paralysis seemed to be due to the state of the left lung, which was found adherent and of a brownish colour; it had the appearance of being compressed and shrunken. The recurrent laryngeal nerve was found to pass through a mass of enlarged glands, just at the arch of the aorta. These existed at the root of the left lung and in the posterior mediastinum. They were of a brownish colour, like the left lung itself, but were not hard. The larynx was somewhat congested.

The explanation of the laryngeal paralysis seemed to depend on the dragging downwards of the left recurrent nerve, due to the greatly enlarged heart, and favoured by the shrunken left lung; and this downward displacement might account for the lessened pulsation in the jugular fossa and under the left clavicle, as the case went on. The implication of the recurrent nerve in the glands cannot be wholly set aside, although their softness would no doubt lessen the chance of their causing irritation, or at least of producing paralysis. The inequality of the pupils was not of the degree or of the kind to warrant much stress being laid upon it, and may fairly be set down as an accidental occurrence, although when it concurred with laryngeal paralysis it was somewhat misleading.

The explanation of the laryngeal paralysis on the left side suggested above was the only plausible method of accounting for this misleading symptom which we could think of at the post-mortem examination. I did not then know that such a cause of laryngeal paralysis was actually recognised; but on mentioning the case at the time it occurred to Dr. John McIntyre, one of my former pupils, who has now devoted himself to laryngology, he told me that in the Vienna clinics this view seemed to be so well recognised that the diagnosis of such a condition was frequently discussed at the examination of patients. I have not been able to lay my hand on any published statements on the subject, although I believe such records do exist. We must, therefore, bear in mind that the occurrence of laryngeal paralysis on one side, in the course of a case with symptoms pointing to the chest and strongly suggestive of aneurysm, does not necessarily imply the existence of aneurysmal disease or of thoracic tumour.

A COMBINED ICE AND ETHER-SPRAY FREEZING MICROTOME.

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DURING the thirteen years that have elapsed since I invented the freezing microtome, the method of freezing has come into general use as an aid to microscopical research in physiology and pathology. It has also become of much service in aiding the practical study of these subjects, especially when they have to be taught to large numbers of students. The original ice and salt freezing microtome described by me in the *Journal of Anatomy and Physiology* for 1871, vol. v., was somewhat faulty. The improved form of the instrument (Fig. 2) described by me in *THE LANCET*, 1873, vol. ii., has been constantly used in my laboratory since that time. We have no difficulty in making with it from four to five hundred sections of the retina, kidney, or other organ, in the course of an hour. When large numbers of sections are required, it is, I think, always advisable to use a mixture of ice and salt as the freezing agent, on the ground of economy, and also because large pieces of tissue can be frozen quickly by it and kept frozen any length of time. We made a fair trial of the ice-freezing microtome devised by Williams and made by Swift of London, in the hope that its large freezing-box and method of regulating the thickness of the sections might