

ARTICLE IV.—*Notes on Two Cases of Stricture of the Œsophagus.*—  
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DURING the course of the past summer I have met with two cases of stricture of the œsophagus, notes of which I have brought together before the Society by way of contrast. The one malignant in its nature, and presenting some interesting points in the diagnosis; the other simple, with dilatation and hypertrophy of the walls above the point of stricture.

CASE I.—On 26th June 1877, I was called to see J. R., ætat 68, on account of severe pain referred to the right side of the sternum, about its middle, and difficulty of swallowing. The patient informed me that, for the last few weeks, there had been difficulty of swallowing which had gradually increased.

On examination, he appeared to be fairly nourished. The percussion of the chest was normal. On auscultation, there was slight accentuation of the second aortic sound. The sounds in the other cardiac areas were normal. The pulses at the wrist were unequal, the right being smaller than the left. The pupils were regular and contractile. Abdominal organs normal. On asking him to swallow, there was an evident difficulty both with fluid and semi-solid food, solids not being attempted. None was regurgitated, although care had to be exercised lest this should happen. The act of deglutition caused no increase of pain.

At first I had some hesitation as to the exact diagnosis, whether the symptoms were to be accounted for by the presence of an aortic aneurism, or by malignant stricture of the œsophagus. The unequal pulses, the pain, the accentuated aortic second, and difficulty of swallowing, looked like aortic aneurism; but the rapid emaciation soon showed that, although there might be some aortic dilatation present, the chief lesion was malignant stricture of the œsophagus. During the course of the disease the pain continued. The difficulty of swallowing increased, and although there was a distressing feeling of hunger, food sufficient to appease it could not be taken. There was considerable restlessness at night, which had to be quieted by opiates.

Four days before his death the patient spat up a quantity of dark foetid material, and after this he gradually sank, and died on the 6th of August.

*Post-mortem Examination.*—The body was much emaciated. The left ventricle of the heart was somewhat dilated. The first part of the aorta was dilated. The middle zone of the right lung was in a state of red hepatization. There was an ulcerated communication between the left bronchus and the œsophagus, about half an inch from the bifurcation of the trachea.

The œsophagus, for about  $2\frac{1}{2}$  inches below this point, was much narrowed on account of epithelial cancer which invaded the wall of the tube, and the surface of which was ulcerated at different points, the chief ulceration having opened into the left bronchus as described.

Dr Wilks, in narrating a case somewhat similar to this in the tenth volume of the *Transactions of the Pathological Society of London*, says, with reference to the death by pneumonia in this case, such was the mode of dissolution in by far the majority of instances, and might be attributed to three causes: to the extreme debility and low powers of the patients, whereby an inflammation is likely to be set up; to an extension of the disease of œsophagus into the root of the lung; or an implication of the pneumogastric nerve in the cancer, whereby its nutritive influence is removed from the lung (as in Reid's Experiments). The latter is a cause which has been long held to be an efficient one by Dr Wilks' colleagues, and especially maintained by Dr Gull.

To these three causes may we not add a fourth, as seen in the present instance? Here the inflammatory affection of the right lung was probably caused by some of the cancerous material from the œsophagus having gone down the right bronchus into the lung, and there set up an irritation. The ulceration had ruptured into the bronchus four days before death, as on that day there was a dark and very foetid expectoration.

Niemeyer says, "that cancerous strictures generally affect the upper and lower third, more rarely the middle third." Klebs (*Handbuch der Pathologischen Anatomie*, Band I. page 161) says, "that the neighbouring segment to the bifurcation of the trachea is the most frequent seat of such strictures." In most of the recorded cases which I have met with, and in the only two cases I have examined post-mortem, the stricture has been at a point corresponding to the bifurcation of the trachea.

CASE II.—On the 21st of August 1877, I was consulted by C. E., aged 52, on account of vomiting. The patient was a short thin man, and on interrogating him I ascertained that for upwards of twenty years past he had occasional difficulty of swallowing; but that, when a difficulty occurred, he was able to overcome it by being struck between the shoulders, or if he rubbed his hand along the line of the gullet in the neck. During the last few years the food had been frequently vomited, and the difficulty of swallowing increased, but that for two or three days past he had been able to retain nothing. On examination I found the patient much emaciated, and the wall of the abdomen collapsed, so that the head and body of the pancreas could be felt. The heart and lungs were normal as regards percussion and auscultation. The urine was normal in quantity and colour, sp. gr. 1125, and the heat and nitric-acid test revealed the presence of one-seventh of albumen. On requesting him to swallow, I found he could readily do so, but after an



interval varying from two minutes to half an hour, the food, whether solid or liquid, was returned. I never accurately ascertained whether the quantity swallowed was the same in amount as that brought up, but to all appearance it was the same or nearly the same. From a consideration of the symptoms I concluded that the patient was suffering from a non-malignant stricture of the œsophagus, with a dilatation above the seat of stricture, and that the kidneys were in the contracted stage of interstitial nephritis. A week after I first saw the patient, Dr G. W. Balfour saw him with me in consultation, and confirmed my diagnosis. He advised me to try and feed him by means of an œsophageal tube, and also suggested the advisability of performing gastrotomy, should I fail in getting food into the stomach by the œsophagus.

I attempted to introduce a long gum-elastic œsophageal tube, No. 6 size, and after getting it in as far as it would go, I allowed milk, both by syphon action and by means of a syringe, to flow through the tube. After a short time the milk regurgitated, and the tube had to be withdrawn. The tube had evidently not reached the stomach, and the milk accumulated in the sac above the stricture. I tried a prostatic gum-elastic catheter, No. 9, with a similar result. As the attempt to introduce food into the stomach had failed, nutrient enemata were administered thrice in the course of twenty-four hours. During this time the patient was able to swallow all kinds of liquid and solid food, which gave rise to no feeling of discomfort, but all or nearly all was returned.

If the operation of gastrotomy was to be tried, the sooner it was done the better. I therefore asked Mr Chiene to see the patient. Mr Chiene attempted to pass both the œsophageal tube and the catheter, with and without the stylet, but was unable to get the tubes into the stomach. On carefully considering the case Mr Chiene was of opinion that the operation might be tried, although the existence of the kidney complication was an objection, but not an insuperable one. But on putting all the arguments for and against the operation fairly before the patient, he decided not to be operated upon. Although at first the enemata seemed to nourish, after they had been used for three or four weeks, the patient objected to their being used any longer. They were therefore discontinued, and as the quantity of food which reached his stomach must have been almost *nil*, he became weaker and weaker, and sunk on the 12th of October.

*Post-mortem Examination.*—Body very much emaciated. Heart small in size, and left ventricle concentrically hypertrophied. Lungs emphysematous at anterior margins. Kidneys—right, congested, and cortical substance contracted to half the natural size. Left, congested, cortical substance still more contracted than right. Spleen small, but normal. The œsophagus and stomach I handed to Mr D. J. Hamilton, Pathologist to the Royal Infirmary, who has most kindly favoured me with the following report:—

“The portion of the œsophagus measures—

In length=10 inches.

Circumference at upper end	=	$1\frac{5}{8}$	inches.
„ at $2\frac{1}{2}$ in. lower down	=	$2\frac{1}{2}$	„
„ at $4\frac{1}{2}$ in. from upper end	=	2	„
„ at 6 in. „ „	=	$2\frac{3}{4}$	„
„ at 8 in. „ „	=	3	„

Thickness of Coats—

At upper end	=	$\frac{1}{4}$	„
At $2\frac{1}{2}$ in. below upper end	=	$\frac{1}{4}$	„
At $4\frac{1}{2}$ „ „	=	$\frac{7}{16}$	„
At 6 „ „	=	$1\frac{1}{2}$	„
At 8 „ „	=	$\frac{5}{8}$	„

“The thickening is chiefly situated in the muscular coat, but there is also very considerable thickening in the submucous coat. More especially is this latter condition seen at the cardiac extremity. The mucous membrane is thickened throughout its entire extent, and its surface is covered with superficial ulcers, about from one to two lines in diameter, of an oval or rounded shape, and merely affecting the superficial layers. They are not mere epithelial erosions, but have a distinct loose edge which can be lifted up. The largest and best marked ulcer is seen immediately above the cardiac orifice. Indeed there are two here, one a little smaller than the other, and several smaller ones around them. There is considerable constriction of the cardiac orifice, the forefinger being with difficulty admitted, and this seems to be due to the contracted submucosa. The mucous membrane is thrown into folds by this, and when looked at from above or below they are seen to completely close the orifice. There is no constriction in the region of the diaphragm, everything in this situation being apparently normal. The stomach, for an adult, is very small, and its coats, more especially the mucous, are somewhat thickened. It contains no food. The mucous membrane is also thrown into folds, but there is no trace of ulceration. The pyloric ring is well marked, and the orifice admits the forefinger.

“On comparing the œsophagus with a normal one, I find the following measurements—

Normal œsophagus.

Greatest circumference,  $1\frac{1}{2}$  inch.

Greatest thickness of walls,  $\frac{1}{8}$  „

“The difficulty in swallowing is, in all probability, to be accounted for thus:—The starting point of the lesion has, apparently, been an inflammation of the mucous membrane, giving rise to thickening of its substance, and to a number of follicular ulcers. The inflammation has evidently extended to the submucosa, and



produced thickening of it also. Contraction of this latter coat has occurred at the cardiac opening of the stomach, while above this the œsophagus is dilated. The dilatation is, however, not universal. At  $4\frac{1}{2}$  inches from upper extremity it only measures 2 inches in circumference, while above and below this the circumference is greater. At 8 inches from the upper end (or two inches from the cardiac orifice) it measures as much as 3 inches, forming within this and in the upper regions a large sac.

“The food in passing downwards would first fall into the upper sac, and would then meet with a slight obstruction. After passing this it would fall into the large sac in front of the cardiac orifice, and no doubt would accumulate here, and that for two reasons, viz., (a) The contraction of the cardiac orifice, and the peculiar folded condition of the thickened mucous membrane, acting like a valve. (b) The food having lost a certain amount of downward force in passing through the constricted portion at  $4\frac{1}{2}$  inches below the upper extremity. The food would tend to be ejected more readily on account of the ulcerated condition of the mucous membrane giving rise to reflex spasm. The existence of several large ulcers immediately in front of the cardiac opening would be specially unfavourable to its transmission past this spot.

“The hypertrophied condition of the muscular coat is evidently due to efforts in ejecting the food.

“The cause of the difficulty in swallowing may be summed up as being in all probability twofold, viz. :—

“1st, Mechanical contraction of the cardiac extremity of the œsophagus.

“2d, Reflex spasm from the ulcerated condition of the œsophagus.”

A case is recorded by Dr Barker in the *Pathological Society's Transactions* for 1859, where there was extensive dilatation of the œsophagus without stricture, which gave rise to no symptoms during life. In the case now under consideration, the dilatation and narrowing of the tube, although giving rise to a certain amount of difficulty of swallowing, did not prevent the patient being nourished. But the occurrence of an inflammation of the mucous membrane, leading to ulceration about two months before death, prevented food reaching the stomach by the reflex spasm still further narrowing the tube; and also for this reason, even a small catheter was unable to pass through a stricture which after death would nearly permit the forefinger to pass.

Klebs considers that the cause of the dilatation in those cases where there is no stricture is to be looked for in an inflammation or fatty degeneration of the muscular coat; while in those instances where there is stricture, the hypertrophy and dilatation results from the obstruction to the passage of the food. He gives one case where there was dilatation of the thoracic part of the œsophagus, with no stricture of the cardiac extremity. The muscular coat of the stomach was hypertrophied, and there was stenosis of the pylorus.