

THORACIC OESOPHAGEAL DIVERTICULUM

WILLEM P. STEENKAMP, JNR.

Surgeon, Cape Town

This is neither an article on a known though comparatively uncommon condition nor a report on a series of cases. The case reported however presents etiological and clinical features that warrant reporting it.

Oesophageal diverticula are classified into pharyngeal, thoracic and epiphrenic according to locality. Etiologically again they are divided into traction or pulsion depending on the causative factor present. Boyd¹ thus stresses the point that, as in hernias and aneurysms the diverticulum owes its origin to pressure from within or traction from without.

To the writer's mind this theory holds good for pulsion only in the case of cervical (pharyngeal) diverticula, since in that region no traction is present.

Thoracic oesophageal diverticula, i.e. those below the bifurcation of the trachea, are usually due to traction exerted by old tubercular lesions, whereas epiphrenic ones, i.e. those just above the diaphragm, are true pulsion diverticula due to obstructed deglutition caused by cardiospasm, or a low oesophageal obstruction caused by the scar tissue resulting from syphilis, tuberculosis, or a high gastric ulcer involving the oesophageal entrance into the stomach. These diverticula are small—often multiple. Lindskey and Liebow² consider an already existing weakness of the oesophageal wall as the main causative factor. Sellors³ states that although pulsion diverticula are fairly common phenomena they practically always occur at the upper end of the oesophagus, being so rare in other parts of the oesophagus that they can safely be disregarded.

Mogendie⁴ found 10% of oesophageal diverticula to be thoracic but does not state whether these cases were mid-thoracic, like the case presently to be described, or epiphrenic. Lahey and Warren⁵ report that out of a series of 374 cases there occurred 9 epiphrenic pulsion diverticula. Here again a truly thoracic one is not mentioned.

The case to be reported was interesting etiologically in that it conformed to neither of the etiological theories advanced by the aforementioned authors. There was no pulsion or traction factor present, nor was there a history or clinical evidence of lues or tuberculosis

or obstruction lower down. Professor Saint⁶ however advanced the very interesting theory that where there has been a large intake of alcohol over a prolonged period of time an inflammatory reaction of the nerve supply of the oesophagus sets in. There is a resulting weakness of the oesophageal musculature followed by diverticulum formation. The case reported conforms to this theory.

CASE HISTORY

K. was kindly referred to me by Dr. P. Brink. The patient was complaining of a continuous feeling of nausea followed by copious vomiting approximately 2 hours after a meal. Two years previously he had only noticed epigastric discomfort, but the condition had deteriorated till he now was forced to seek medical advice.

The past history was good except for an intake of alcohol well above usual extending over the past 6 or 7 years. There were no serious illnesses or operations.

Physical examination revealed a fairly well nourished male aged 46 years. Except for slight deafness all the other findings were within normal limits.

Considering his age and the symptoms complained of, peptic pathology was the first consideration and he was referred to Drs. Le Roux and McCallum for a barium meal. Only one plate of an excellent series of pre-operative films is here reproduced (Fig. 1) and the diverticulum is shown to be mid-thoracic, that is to say the ostium, although owing to its size the belly is practically resting on the diaphragm and partly posterior to the heart, is just below the bifurcation of the trachea.

Operative treatment was decided on. Since the blood supply of the oesophagus is none too copious it was difficult to decide on the best operative approach, since simple extirpation would leave a very wide defect in the oesophagus and thus a great possibility of post-operative sloughing of the suture line with the complications following an intrathoracic leakage.

A very feasible alternative would be anastomosis of the diverticular pouch through the diaphragm with the stomach. This would be fairly easy technically



Fig. 1

but subsequent regurgitation of acid stomach contents into the pouch might then cause anastomatic ulcer formation.

The last alternative would be amputation of the oesophagus above the diverticulum and anastomosis with the stomach drawn up through the diaphragm into the thorax.

Since the last measure would be quite a formidable operation equivalent in magnitude to an abdomino-thoracic total gastrectomy with all its possible complications, extirpation of the sac was decided on as probably being the lesser of many evils.

The thoracic cavity was entered through the 10th interspace after removal of the 9th rib. Adhesions or other signs of previous intra-thoracic pathology were conspicuous by their absence.

Oesophagus and vagi were identified but the diverticulum was nowhere to be seen. However, after the pulmonary ligament had been split, which enabled the assistant to lift the lower lobe of the lung upward and at the same time displace the heart to the left, the diverticulum was exposed in its entirety.

The sac was lifted from its bed and put under tension—slight since it was found to be friable and started bleeding where touched by the forceps. The diverticu-

lum was now amputated but *not* too flush with the oesophagus wall, so as to leave a liberal edge which could be folded inwards and thus avoid future stricture formation. Mucosa was separated from media and serosa and the two cut edges joined with a continuous catgut no. 00. Media and serosa were now joined in a separate layer and reinforced with interrupted silk stay-sutures.

There was no bleeding throughout the operation. But the old adage has it, 'When in doubt drain', and the thorax was accordingly closed in the usual manner after institution of underwater drainage.

Two pints of blood were transfused during and after operation, although this was hardly necessary since patient's condition remained excellent throughout.

Post-operatively a Ryall's tube was left *in situ* and small feeds given through the tube on the 2nd day. The patient, however, was very restless and on the second post-operative night pulled the Ryall's tube out. Realizing what he had done he tried to put the tube back again, and the resultant noise attracted the attention of the Night Sister! Needless to say the worst was feared after this heroic effort. However, he was none the worse for his attempted co-operation and after liberal doses of C_2H_5OH had been added to the milk he settled down nicely and never looked back again.

He made an uneventful convalescence and after 2 weeks was enjoying a full meal.

Pathological examination of the sac was made by Drs. Clegg and Finlayson who reported as follows:

Specimen Received: A diverticulum removed from the oesophagus. It was flabby and pouch-like having a brown outer surface and a pink colour inside where the mucous membrane

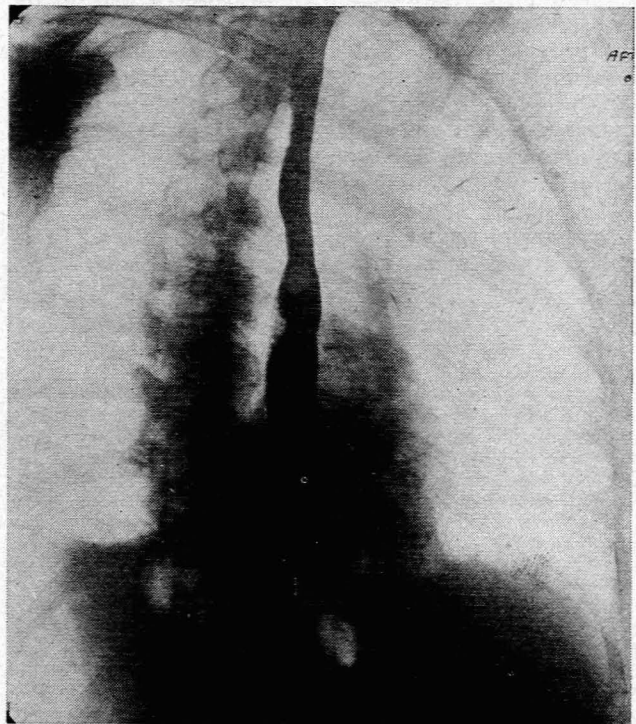


Fig. 2

was situated. There was also a tiny yellow spot where there may have been an ulcer.

Microscopic Examination: The diverticulum is lined by stratified squamous epithelium which appears orderly and benign. It is broken at the place where the yellow spot was seen macroscopically and an ulcer exists but there is very little evidence of active inflammation although the floor is made of very congested connective tissue. The muscularis mucosae is well preserved but the outer muscle walls (both longitudinal and circular) are practically non-existent, only traces being apparent here and there. The appearances are benign and no evidence of neoplasia could be seen.

Fig. 2 is an X-ray photograph taken 1 month after the operation. At that time the patient was enjoying normal meals and gaining weight.

SUMMARY

The etiology of oesophageal diverticulum is reviewed.

A new theory is put forward, viz. that alcoholic neuritis leading to muscular degeneration is a causative factor.

A case is described which appears to support this theory.

I wish to thank Mr. H. Katz for his valuable assistance at the operation as well as in post-operative care.

REFERENCES

1. Boyd, W. (1947): *Surgical Pathology*. London and Philadelphia: William Saunders.
2. Lindskog, G. E. and Liebow, A. A. (1953): *Thoracic Surgery*. New York: Appleton.
3. Sellors, T. H. (1933): *Surgery of the Thorax*. London: Constable and Co. Ltd.
4. Magendie, J. (1950): *J. méd. Bordeaux*, **127**, 793.
5. Lahey, F. H. and Warren, K. W. (1954): *Surg. Gynec. Obstet.*, **98**, 1.
6. Saint, C. F.: Personal communication.