

in that direction. This again would still further increase the stenosis at the point of passage of the esophagus through the diaphragm by tending to cause a sharp kink of the tube at that point.

The literature of the subject has been comprehensively reviewed in an article by Dr. H. Strauss, of Berlin, Germany, which formed the subject of a lecture and demonstration at the Nineteenth Congress of Internal Medicine at Berlin. Among the theories given to account for the condition may be mentioned the following:—

1. Congenital weakness of the oesophageal wall as urged by Strümpel.

2. Abnormal relaxation or elasticity of a Mehnart's oesophageal entromere.

3. Pressure of the aorta upon the lower portion of the oesophagus, leading to a slight degree of stagnation which, it is argued, sets up repeated irritations of the mucous membrane which lead to spasms of the cardiac region of the oesophagus.

4. Strümpel considered that in his case a bend of the oesophagus in its lower portion had impeded the passage of the oesophageal contents.

In the transactions of the Pathological Society of London, Vol. 39, p. 103, Handford reports a case of dilatation of the oesophagus without stricture. The history given is similar to that given above as regards the difficulty of swallowing and the regurgitation of food, but differs in regard to the cardiac symptoms and the mode of death. The seat of obstruction in this case was exactly at the point where the oesophagus passes through the diaphragm, and it is noted that there was no intrinsic stricture of the oesophagus, since the opening would readily admit the finger. There was no induration or thickening which could point to a cicatricial condition or new growth. It is noted, however, that the aorta was dilated to some extent, and the cause of the obstruction is attributed to the pressure of the oesophagus against the unyielding central tendon of the diaphragm by the dilated aorta. The condition of the diaphragm itself is not noted, nor is there history of hicough, as was present in my case, but it seems to me possible that the fault here may have been primarily in the diaphragm, since it is difficult to understand how simple dilatation of the aorta could produce such an effect upon the oesophageal opening through the diaphragm.

I have above given my reasons for holding the diaphragm responsible, in this case, for producing an extrinsic stenosis of the oesophagus, probably primarily as a spasmodic condition but subsequently passing on to an organic lesion due largely, if not altogether, to hypertrophy of the pillars of the diaphragm.