OESOPHAGECTASIA IN A CHILD.

BY

J. A. M. CAMERON, M.B.,

Assistant Pathologist, Victoria Infirmary, Glasgow,
Research Student in Pathology, University of Glasgow.

Dilatation of the œsophagus from cardiospasm is a rare condition in children, and this example is of further interest because for the first time, as far as I know, an acute inflammatory lesion affecting the cells of Auerbach's plexus in the lower end of the œsophagus is shown to be associated with its development.

At death this male child was six years old. Since the age of six months he had suffered from attacks of vomiting which appeared immediately after food. Intervals of ten days to three weeks occurred in which this did not happen, and once for six weeks he was free. For four weeks before admission to hospital he vomited immediately after every kind of food or drink; a few days after admission to hospital he died.

At eight months he had broncho-pneumonia and thereafter developed phthisis for which he was treated in a sanatorium for about a year; when two-and-a-half he had whooping-cough.

Post-Mortem Examination.

A post-mortem examination was made by Dr. Anderson, and the only points of interest in the autopsy protocol were, that a scar of healed phthisis was found in the apex of the left lung, congestion was present in the right base, and calcification of one or two bronchial glands was noted. No evidence of active tuberculosis was seen anywhere. The pyloric and pelvic-rectal areas were of normal size and appearance.

The œsophagus (Fig. 1) was $5\frac{1}{4}$ in. in length from the cricoid to the cardia and showed a fusiform dilatation measuring $2\frac{1}{4}$ in. at its greatest circumference: at the cardia the measurement was $\frac{1}{2}$ in. The circular muscle fibres were hypertrophied from the cardia throughout the dilated area, the thickness of the wall being approximately $\frac{9}{32}$ of an inch at the cardia, $\frac{3}{16}$ at the maximum dilatation $2\frac{1}{4}$ in. above the cardia, and gradually narrowed to normal thickness about 1 in. below the cricoid. Several minute erosions of the mucous membrane were found throughout the affected area.

Microscopic Examination.

Section made just above the cardia showed the epithelial layer to be pitted with numerous erosions, and absent in other parts (Fig. 2). The submucous coat was invaded by masses of lymphocytes and endothelial cells with congestion of vessels, and the muscularis mucosæ was increased in thickness, while hyaline changes in some of the fibres were noticed. There was definite and striking hypertrophy of the circular muscle layer, and here and there clumps of inflammatory cells occupied the interspaces; fibrotic changes in some of the fibres was seen and hyaline and fatty change in others.

In the longitudinal layer, as usually happens in these cases, not much alteration was evident, although here too degenerative changes can be seen and a few inflammatory cells were scattered throughout it.

In the intermuscular layer the exudation of leucocytes and endothelial cells was abundant. The ganglia as a whole were affected and presented in places a glassy, hyaline appearance with only an odd cell showing a nucleus, while the capsule was lined and surrounded by numerous leucocytes and endothelial cells. In one or two places an area of what appeared to be colloid was found where nerve cells would ordinarily be expected. The capillaries were congested and the endothelial cells were swollen. The most careful search for tubercle bacilli was in vain, but some Gram-positive diplococci, presumably pneumococci, were found (Fig. 3).

DISCUSSION.

This is the eighth of a series of cases of cardiospasm which I have examined histologically, and all show lesions in the intermuscular area. The first three were examined for Dr. Brown Kelly, and the results were communicated by him in his Semon Memorial Lecture in 1926. The others will be reported in detail at another time. Meanwhile it is sufficient to note that some degree of fibrosis with degenerative changes in the nerve cells of Auerbach's plexus was present in all. Similar results have been reported by Hurst from examinations made by Rake and the late Adrian Stokes.

Until the case now under consideration was examined I was inclined to look upon the ganglionic changes as athero-sclerotic in origin, as the thickened blood vessels formed rather a characteristic feature of the sections, but the direct and indisputable evidence of bacterial invasion shown here makes a toxic basis for all the more credible, especially as the antecedent development of an acute febrile condition is not uncommonly reported in the literature of this disease.

In the normal act of swallowing the œsophagus is straightened out from its 'repose' position by the muscles of the tongue and hyoid bone. These

Fig. 1. Oesophagectasia.
Child—6 years old.

Fig. 2. High power drawing of section through cardiac canal, showing inflammatory exudate.

pull the larynx and cricoid plate away from the spine, and the inferior constrictor of the pharynx then starts the bolus on its course through the œsophagus. According to the researches of Kronecker and Meltzer the bolus at any point in its course causes contraction of the area just above it and relaxation below, the integrity of the cells in Auerbach's plexus governing this reaction. In a case such as I have described here the reflex is altered by the ganglionic changes and only contraction follows the stimulus. The retention of food slowly dilates the viscus and the circular muscle hypertrophies in an attempt to expel its contents. A striking feature of practically all recorded cases is the persistence of the hypertrophy throughout the dilated area; at times indeed, it appears to develop proportionately and *pari passu* with the enlargement.

Fig. 3. Microphotograph showing degeneration of nerve cells with leucocytic exudation.

REFERENCES.

- Kelly, A. B., *J. Laryng. and Otol.*, Edin., 1927, XLII, 4.
Walton, A. J., *Brit. J. Surg.*, Bristol, 1925, XII, 48
Hurst, A. F., *Lancet*, Lond., 1927, i, 618.