and normal saline into the bronchi. In the next year they also published an analysis of cases of pulmonary abscesses, including those which were complicated with bronchitis.

Ewart Martin (Edinburgh), in 1924, published his experiences of bronchoscopy in relation to lung diseases, and gave results of treatment, some cases having extended over one and a half years.

Howarth, in 1925, published, in association with Cassidy, a case of abscess of the lung and bronchiectasis treated by bronchoscopic aspiration and lavage.

Owing to successive researches and inventions, these methods have now become much simplified, and are part of the usual routine of the laryngologist. Success in this country has been largely associated with becoming accustomed to using the largest endoscopic tubes which can be safely employed in each case. Endobronchial therapy now offers a wide and expanding field for further research work.

THE ETIOLOGY OF DIVERTICULA OF THE INTESTINE.

BY

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IN the interesting paper on intestinal diverticula, published in the *BRITISH MEDICAL JOURNAL* of January 23rd (p. 130), a prediverticular state of local inflammation of the colon is described which, it is suggested, is responsible for the formation of the diverticula.

The etiology of diverticula has for a long time been of special interest to me, and of the various theories put forward to account for them none have seemed very convincing. I have for some years believed and taught that inflammation is a probable factor in producing the condition, at any rate in some of the cases in which the colon is affected, and I therefore particularly welcome the paper as supplying confirmatory evidence. My reasons for believing diverticula of the colon to be due to an inflammatory cause were two in number:

1. The most common seat of diverticulosis is one at which evidence of chronic inflammation in the shape of peritoneal adhesions is very often found—that is, the lower end of the sigmoid flexure. Clinically also patients are frequently seen in whom the sigmoid is hard and tender, suggesting that a mild degree of inflammation is present. The obvious criticism will perhaps be made that if these signs of inflammation have anything to do with the liability of this part of the colon to form diverticula, diverticula should be still more common in the caecal appendix, where inflammation is far more frequent than anywhere else in the gut. The answer to this is that the appendix is prevented from being a common seat of diverticulosis by the greater relative thickness of its muscular covering, and also by the fact that its lumen is rarely distended sufficiently to stretch its walls, and therefore the final factor in the production of diverticula—internal pressure—is wanting.

2. The second reason was the outcome of an investigation which I made some years ago of a number of specimens of gut removed either by operation or after death from cases of diverticulitis, by which I hoped to be able to trace some definite causal relationship between the fat of the appendices epiploicae and the occurrence of diverticula. I discovered nothing that seemed to me at that time sufficiently convincing for publication, but my suspicions were aroused by the fact that in several of these specimens I found in the wall of the gut, extending for a distance of some inches from the inflamed diverticula, localized patches of inflammation, some of which were evidently of long standing, in the bases of attachment of the appendices epiploicae, whereas in places where the muscle fibres were in direct contact with the peritoneum and had no fatty covering there were no signs of inflammatory change.

It seemed to me probable that these inflammatory patches might be accounted for by a lack of free lymphatic drainage at the points where the intestinal muscle was coated with a thick layer of fat, and that there might thus be a twofold cause which determined the onset of diverticulosis—

first, an abnormally heavy deposit of fat in the appendices epiploicae; and secondly, an inflammatory process in the deepest portion of the fat, which in the course of time so weakened the muscle that it eventually gave way to the pressure exerted by the intestinal contents and allowed herniation of the mucous membrane to occur.

But whatever part inflammation may play in the production of the diverticula it must, I think, be assumed that the intestinal fat is in some way primarily responsible, for not only do the diverticula invariably occur at points where the intestinal wall is normally covered by fat and never where it is covered by peritoneum alone, but also an abnormal degree of adiposity is an accompaniment of diverticulosis to which nearly every writer on the subject has drawn attention, and in fact might almost be said to be a *sine qua non*. My experience has been limited to cases in which inflammation of the diverticula has occurred; of seventeen cases at Guy's Hospital of which I have notes, only two were in people not unusually stout, and both of these had passed through a period of obesity before the symptoms of diverticulitis manifested themselves. Therefore no theory of etiology can be held to be satisfactory which does not make the fat the starting-point in the process of which the herniation of the mucosa is the final outcome.

Three possible explanations occur to me, each of which I think is reasonable in that a parallel can be found for it in pathological processes occurring elsewhere in the body. They are as follows:

1. The subperitoneal fat weakens the intestinal wall by inducing a simple atrophy of the muscle from pressure.
2. The fat invades and separates the muscle fibres, and so produces weak spots. In this case the natural line of invasion would be along the vessels which penetrate the muscular coat, so that these would be the most likely points for herniae to occur.
3. The fat becomes infected, probably by the intestinal contents, and the muscle in contact with it is weakened by an inflammatory process.

In the light of the x-ray evidence now added to the reasons which I have given above there would seem to be good grounds for believing that this last alternative is the true explanation of most of the diverticula occurring in the colon, though some in the colon and perhaps most of those in the small intestine, where infection of the fat is less likely to happen, may be ascribed to one or other of the non-inflammatory causes I have suggested.

FISH AS THE SOURCE OF CERTAIN COCCIDIA RECENTLY DESCRIBED AS INTESTINAL PARASITES OF MAN.

BY

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For some time the attention of the authors has been devoted to the study of protozoal parasites in the organs of fish, which, since they are a common item in the human dietary, were readily obtained from various retail fish shops in London. While the present communication is to be regarded as a preliminary note on this subject, there have been made already certain observations which appear to prove conclusively that three recently described coccidia, up till now presumed to be true intestinal parasites of man, are merely parasites of fish passed unaltered through the human alimentary tract.

Wenyon (1916) discovered a coccidian belonging to the genus *Eimeria* in human faeces, but, rightly doubting that it was not a true parasite of man, refrained from creating a new species until something more was known of its habitat and life-cycle. Dobell (1919), however, named it *Eimeria wenyoni*. It will be seen later that this organism is morphologically identical with *Eimeria clupearum* Thélohan, 1894.