The pathology of chronic oesophagitis¹

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EDITORIAL SYNOPSIS This paper reports the results of a histological study of severe chronic oesophagitis. The material consisted of 46 specimens removed by oesophagogastrectomy. Two distinct types of lesion were found: chronic superficial oesophagitis and localized penetrating ulceration, the latter being found closely related to intrusions of gastric cardiac type (mucosa) into the lower oesophagus.

Although the first histological study of the lesions of peptic oesophagitis was made as long ago as 1879 by Quincke, the chronic form of the disease was regarded as a rarity by clinicians and pathologists alike until quite recent years. Jackson (1929) was the first to appreciate that the condition might have some clinical importance, but it was not until the development of thoracic surgery that it began to be recognized as a relatively common and occasionally disabling disease. Since 1939 the clinical and radiological aspects of the condition have received much attention in the literature, but there have been few reports of comprehensive pathological studies, and these have been concerned chiefly or wholly with post-mortem material.

Early writers stressed the close morphological resemblance between chronic ulcerative lesions of the lower oesophagus and those occurring in the stomach and duodenum (Tileston, 1906; Stewart and Hartfall, 1929). Lyall (1937) was the first to suggest that such lesions in the oesophagus were not of uniform structure. He described two types: a superficially ulcerative lesion associated with gross fibrous thickening, resembling the 'congenital' oesophageal stenosis of children reported by Kelly (1930; 1936), and a more sharply localized and deeply penetrating ulcer, associated with heterotopic gastric mucosa. Lyall noted, however, that even in the otherwise superficially ulcerative lesions there were sometimes areas of deeper penetration, leading to actual perforation of the oesophagus and erosion of adjacent structures in some cases. Others have since reported generally similar findings (Allison, 1946, 1948; Peters, 1955).

Barrett (1950), after studying the cases reported by Tileston (1906), by Stewart and Hartfall (1929), and

by Lyall (1937), suggested that the localized ulcers described and illustrated by these writers were in all cases situated in gastric mucosa which extended upwards into the lower oesophagus from the stomach. The occurrence of such a mucosal anomaly, distinct from the heterotopic patches in the upper oesophagus described by Taylor (1927), was quickly confirmed by others (Bosher and Taylor, 1951; Allison and Johnstone, 1953; Peters, 1958) but the lesions reported by these authors in association with the anomaly varied considerably in morphology.

The present investigation was prompted by the fact that as yet no systematic study of fresh surgical material has appeared in the literature.

MATERIAL AND METHOD OF EXAMINATION

Forty-six specimens removed by oesophago-gastrectomy were studied. The ages of the patients ranged from a few months to 75 years. In most cases resection had been undertaken for the relief of oesophageal obstruction, due most commonly to actual fibrous stenosis but occasionally to gross fibrosis of the oesophageal wall, which caused severe derangement of function though only relatively slight narrowing of the lumen. A hiatus hernia of the stomach of Allison's sliding type (1951) had been demonstrated clinically in 44 of the 46 cases, and in a few cases resection had been performed because oesophageal fibrosis had prevented the reduction and repair of the hernia.

Each specimen (Fig. 1) comprised approximately the lower half of the oesophagus together with a part of the stomach, in most cases the proximal half, in a few cases less. The specimens were fixed with the least possible distortion in formol acetate (4.5% sodium acetate in 10% formalin). After thorough fixation, the oesophageal part of each specimen and the upper part of the segment of stomach attached were divided transversely at intervals of approximately 0.5 cm. In as many cases as possible a thread of braided nylon was passed through one side of each of the blocks so made, to act as a

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FIG. 1. A typical specimen ($\times 0.75$).

marker by which the sections could subsequently be orientated. Each block was then dehydrated by a mixture of *n*-butyl alcohol and ethyl alcohol (Lendrum, 1941), cleared in chloroform, and embedded in paraffin wax. The upper face of each block was sectioned on a base sledge microtome, and the sections were mounted in the usual way. Sections from every block were stained with haematoxylin and eosin and with haematoxylin and van Gieson's mixture; further sections were stained by other methods, including Mallory's phosphotungstic acid haematoxylin, Weigert's elastic tissue stain, and Southgate's method or the periodic acid-Schiff technique for mucin, when required. The sections were examined microscopically and by projection upon a screen at a magnification of about 30 diameters.

LEVEL OF THE OESOPHAGO-GASTRIC JUNCTION The line of division between the stomach and the oesophagus is arbitrary, and cannot be related to any known anatomical landmark. In normal circumstances the external configuration may be a fairly reliable guide, but in the presence of a small hiatus hernia of the stomach of the so-called sliding type it is not reliable, since the herniated

part of the stomach tends to be more or less tubular in form and of a circumference not much greater than that of the lower oesophagus. The transition from the oesophageal to the gastric type of musculature, both in the major laminae and in the muscularis mucosae, is not abrupt but gradual. The line of the mucosal junction, though normally sharply defined, is most irregular in contour, so that the two epithelia normally interdigitate in a zone of considerable length; moreover, extensive intrusions of gastric mucosa into the oesophagus, even as high as the aortic arch or higher, are now known to occur. There appears, however, to be no record of downward extension of the squamous epithelium of the oesophagus into the stomach. The submucosal mucous glands are generally accepted by histologists as essentially oesophageal structures, though according to Schumacher (1927) they do not normally extend downwards as far as the line of junction of the two mucous membranes.

In this study it seemed desirable to adopt a reference point of some sort to indicate, however approximately, the level at which the oesophageal part of each specimen merged into the gastric part. The lowest level at which submucosal mucous glands or their ducts could be demonstrated, or in their absence the lowest level at which squamous epithelium was found, was selected for this purpose. The level thus indicated is probably somewhat above the true physiological level of the junction. In only one case, in which both submucosal glands and squamous epithelium had been completely destroyed, was it impossible to estimate the level of the junction in this way.

OBSERVATIONS

The inflammatory lesions which were found in these cases were of two quite distinct types, though in more than half of the cases both types were present in combination.

CHRONIC SUPERFICIAL OESOPHAGITIS The 16 specimens which exhibited this type of lesion alone showed no significant abnormality of external contour, but the lesion was palpable as a sausage-shaped zone of increased firmness. The transverse sections revealed more or less concentric thickening and fibrosis of the oesophageal wall and, in all but one case, narrowing of the lumen. The stenosis was commonly severe, and varied in length from 1.0 cm. to more than 4.0 cm.

Histologically, the essential features were superficial erosion of the mucosa and fibrosis, with varying degrees of actual tissue destruction, in all layers of the oesophageal wall. The mucosal erosion was usually most extensive at a level somewhat above the line of junction between the squamous and gastric mucosae (Figs. 2, 3); it was always superficial, never extending in depth beyond the level of the muscularis mucosae. The interstitial fibrosis of the deeper tissues was always maximal in the submucosa, in which there was usually complete

FIG. 2. *Chronic superficial oesophagitis. A section through the region of the junction between squamous and gastric mucosae, showing much intact squamous epithelium. Haematoxylin and eosin $\times 9$.*

FIG. 3. *Same case as Fig. 2. A section at a higher level shows complete destruction of the squamous epithelium, which is replaced by granulation tissue. Haematoxylin and eosin $\times 16$.*

FIG. 4. Chronic superficial oesophagitis, showing extensive destruction of the muscularis mucosae and interstitial fibrosis of the other layers, with gross stenosis, but complete mucosal regeneration. Haematoxylin and van Gieson $\times 10$.

destruction of the mucous glands. In the muscular laminae there were varying degrees of loss of muscle fibre, often surprisingly slight, but even in the most severely damaged specimens there was never a complete and cleanly cut breach in either muscular lamina (Fig. 4). The neural structures of Auerbach's plexus, though often damaged, could be identified easily in almost every case. Fibrosis was often considerable in the peri-oesophageal connective tissue, tending to be maximal around the longitudinally running bundles of the vagus nerves. Blood vessels in the submucosa and in the peri-oesophageal connective tissue showed only minimal subintimal fibrosis.

In one case the level of the oesophago-gastric junction could not be estimated. In the remaining 15 cases there were small extensions of gastric mucosa of cardiac type into the lower oesophagus, to a distance of 2.0 cm. or less in all but two cases. Mucosa of fundal type extended into the oesophagus in six cases, but these extensions were exceedingly small.

LOCALIZED PENETRATING ULCERATION This could be chronic or subacute.

Chronic Two specimens presented this type of lesion in its pure form, each containing two lesions. In one, the site of each lesion was marked by an external dimple. In each case the lesions were situated opposite one another, and in the one case in which orientation of the specimen was possible they lay in the anterior and posterior walls respectively. Appreciable stenosis was present in both, reaching a length of 0.5 cm. in one and 3.5 cm. in the other; in each case, owing to the presence of two opposing lesions, the narrowed lumen was approximately central in position (Fig. 5). The ulcers appeared to be roughly oval in form, measuring up to 3.0 cm. in length and up to about 1.5 cm. in transverse diameter.

Histologically, this type of lesion resembled exactly a chronic peptic ulcer of the stomach. Each ulcer was localized and well defined, and the tissue destruction in its floor extended through the full thickness of the oesophageal wall, producing a

FIG. 5. *Pure chronic localized penetrating ulceration. Two lesions are present, one anterior and one posterior, involving principally sections 5 and 6, in which the dimples produced in the external surface of the specimen are visible. The lumen is narrowed but only slightly displaced from the mid-line ($\times 0.35$).*

cleanly cut breach in the muscular laminae, the severed edges of which tended to turn inwards towards the lumen (Fig. 6). The granulation tissue which replaced the mucosa in the floor of the lesion showed superficial fibrinoid necrosis. There were severe obliterative changes in the adjacent arterial branches in the submucosa and peri-oesophageal connective tissue.

Gastric mucosa of cardiac type extended to the top of each specimen; in one it had completely replaced the normal epithelium, and in the other small islands of squamous epithelium remained in the upper part only. The gastric mucosa, which was distorted and showed intestinal metaplasia, lined the circumference of each lesion, and showed regenerative activity which had resulted in the re-epithelialization of one crater (Fig. 7). Mucosa of fundal type projected upwards to a maximum height of 1.0 cm. in each specimen, but did not reach the immediate neighbourhood of any of the ulcers.

Subacute Localized penetrating ulcers of subacute type were found in three cases, in one of which chronic penetrating ulcers were also present at a different level. The subacute ulcers were small, up to 0.7 cm. in diameter. They produced no visible alteration in the external or internal contour of the oesophagus, and did not cause any significant narrowing or asymmetry of the lumen. In the one specimen which could be orientated accurately the subacute lesion was in the posterior wall.

Histologically, these lesions resembled exactly subacute ulcers of the stomach. The sharply circumscribed mucosal erosions did not penetrate beyond the depth of the muscularis mucosae, but there was moderately severe and extensive fibrosis of the underlying submucosa, and slight interstitial fibrosis of the circular muscular lamina. The granulation tissue lining the floor of the ulcer showed superficial fibrinoid necrosis. There were no vascular lesions.

In each specimen gastric mucosa of cardiac type reached a considerable height, up to 6.0 cm. above the estimated level of the oesophago-gastric junction, and each lesion was surrounded entirely by this type of mucosa. Small upward projections of fundal mucosa were present in two cases.

COMBINED LESIONS In 26 of the 46 specimens localized penetrating ulceration was found in combination with chronic superficial oesophagitis; the penetrating component was chronic in 23 and of subacute type in three cases. In all the cases the lesions were contiguous, the localized penetrating component being found at the lower end of the zone of chronic superficial oesophagitis.

In only one case did the external appearance of the specimen furnish a clue to the presence of a penetrating lesion; in this, a fairly deep external dimple marked the site of a chronic penetrating ulcer (Fig. 8). In all the other specimens the externally visible and palpable changes were identical with those found in cases of uncomplicated chronic superficial oesophagitis. When the transverse sections were prepared, eccentricity of the lumen in the region of

FIG. 6. *Pure chronic localized penetrating ulceration in a lower oesophagus lined extensively by gastric mucosa. Complete destruction and fibrous replacement of the muscular laminae in the floor of the ulcer. Haematoxylin and van Gieson $\times 12$.*

FIG. 7. *Pure chronic localized penetrating ulceration, healed. Submucosal mucous glands are present on each side of the healed ulcer. Haematoxylin and van Gieson $\times 12$.*

FIG. 8. Chronic localized penetrating ulceration combined with chronic superficial oesophagitis. The external dimple marking the site of the penetrating ulcer is seen posteriorly in section 6 ($\times 0.55$).

a chronic penetrating ulcer was sometimes obvious, and the actual crater could occasionally be made out with the naked eye. In the great majority, however, the presence of penetrating ulceration was recognized only during histological examination of the specimen; in these cases, owing to inversion of the edges of the severed muscle and fibrous thickening of the floor, the crater of the localized ulcer had been almost completely obliterated, and the narrowed lumen was still more or less centrally placed in the

oesophagus. None of the subacute ulcers could be recognized with the naked eye.

Seventeen of the 23 cases showing chronic penetrating ulceration contained a single penetrating lesion. Eleven of these specimens could be orientated accurately; in nine the lesion was in the posterior wall and in two it was situated anteriorly. In six cases two penetrating lesions were found; in each of these the lesions lay opposite one another, and in the five specimens which could be orientated one lesion was in the posterior and the other in the anterior wall. The greatest diameter of the chronic penetrating lesions appeared to be about 2.0 cm., and to lie in the longitudinal axis of the oesophagus.

In each of the three cases showing subacute penetrating ulceration the penetrating lesion was single. Only one of these specimens could be orientated accurately, and in this the penetrating component was situated posteriorly.

Appreciable stenosis was present in 23 of the 26 cases in this group. In nearly every case both lesions appeared to contribute to the narrowing, long stenosis in general being due chiefly to the chronic superficial component.

The histological appearances of the component lesions were similar to those already described. When the changes were followed from above downwards in consecutive transverse sections the typical picture of chronic superficial oesophagitis was first seen (Fig. 9). The earliest suggestion of the presence of the penetrating component was the appearance of a zone of fibrinoid necrosis in the granulation tissue and exudate replacing the eroded mucosa. In further sections at lower levels the full picture of the localized lesion was found (Fig. 10). In the lesions classified as subacute the ulceration penetrated only into the submucosa, but in the chronic lesions there was the characteristic localized destruction of the musculature, which was replaced by scar tissue. In 13 cases this localized destruction was complete, involving the full thickness of the circular and longitudinal laminae; in 10 cases the destruction was complete only in the circular lamina, the longitudinal lamina showing gross interstitial fibrosis but only partial destruction of muscle fibres. Obliterative changes in the adjacent arteries were common, being found in 14 of the cases with chronic ulceration and in one of those with subacute ulceration.

In two cases the floor of a chronic penetrating lesion had been completely covered by squamous epithelium. In several other cases attempts at healing were seen, usually by the regeneration of simple columnar epithelium. In every case the lower part of the circumference of the penetrating lesion, whether subacute or chronic, was lined by mucosa of gastric cardiac type. The upper part of the cir-

FIG. 9. *Chronic localized penetrating ulceration combined with chronic superficial oesophagitis. This section, from the upper part of the specimen, shows partly healed chronic superficial oesophagitis, with some stenosis. Haematoxylin and van Gieson $\times 5$.*

FIG. 10. *Same case as Fig. 9, section at a lower level, adjacent to junction between squamous and gastric mucosae. Gross stenosis and disorganization of structure produced by chronic localized penetrating ulcers in anterior and posterior walls. The defect towards the lower right hand edge is an artefact. Haematoxylin and van Gieson $\times 5$.*

FIG. 11. *Same case as Figs. 9 and 10, but at a lower level still, showing the oesophagus lined wholly by gastric mucosa. Submucosal mucous glands are present. There is little or no sign of inflammation at this level. Haematoxylin and van Gieson $\times 5$.*

cumference was devoid of epithelium, merging into the granulation tissue of the chronic superficial lesion above, and the lateral margins were lined sometimes by squamous and sometimes by glandular epithelium. The gastric mucosa which abutted upon the margin of the penetrating lesion in all cases was of cardiac type; fundal mucosa, though occasionally present at the same level in the oesophagus, was never seen in close proximity to the edge of the ulcer. Below the level of the penetrating lesion there was in almost all cases a short segment of oesophagus, lined by gastric mucosa but in other respects practically normal, containing submucosal mucous glands and showing only slight fibrosis, if any, in the submucosa and peri-oesophageal connective tissue (Fig. 11). The greatest height in the oesophagus reached by gastric mucosa in these cases was about 4.0 cm., but in the majority the height was 2.0 cm. or less.

DISCUSSION

The changes described here under the heading of chronic superficial oesophagitis resemble in all respects those reported by Stewart (quoted by Allison, 1946; 1948) in two of the earliest cases of chronic oesophagitis to be treated by surgical excision. The most distinctive features were the superficiality of the actual ulceration, which in no case penetrated beyond the depth of the muscularis

mucosae, the extent and severity of the interstitial fibrosis in the deeper layers of the oesophageal wall, and the absence of any significant changes in the blood vessels. Notable also was the extensive destruction of the submucosal mucous glands. The absence of any deep penetration of the ulceration into the oesophageal wall contrasts with the findings of Lyall (1937) and of Peters (1955), both of whom reported that in a small proportion of their otherwise similar cases deeply penetrating ulceration occurred.

It is probable that this type of oesophageal lesion is the direct result of constant or repeated reflux of gastric secretions into the oesophagus, due in its turn to incompetence of the mechanism which normally prevents such reflux. The nature of this normal mechanism has not been determined, and indeed is the subject of considerable dispute. The histological findings in these cases do not throw any direct light upon this question: there was no anatomical evidence of a sphincter at any level in any case. The fact that in many cases squamous epithelium had survived intact immediately above the level of the oesophago-gastric mucosal junction, though the mucosa at higher levels had been destroyed over a length of several centimetres, may be of some significance in this connexion. This phenomenon was noted by Stewart (Allison, 1946; 1948) and also by Peters (1955), who attributed it to the pinchcock action of the diaphragm, by which the

walls of the oesophagus were held in close apposition, thus preventing the accumulation of regurgitated gastric secretions at this level. In the great majority of the present series of cases the area in question was well above the level of the diaphragmatic hiatus owing to the presence of a hiatus hernia of Allison's sliding type (1951), and thus could not have been protected in this way. A similar effect could, however, have been produced by the action of an intrinsic oesophageal sphincter which, though unable to prevent gastro-oesophageal reflux, might be adequate to prevent the accumulation of regurgitated secretions in the actual zone of its activity. Evidence for the existence of such a sphincteric action in the lower oesophagus has been accumulating steadily in recent years (Dornhorst, Harrison, and Pierce, 1954; Creamer, 1955a and b; Fyke, Code, and Schlegel, 1957; Botha, Astley, and Carré, 1957; Atkinson, Edwards, Honour, and Rowlands, 1957; Creamer and Pierce, 1957; Botha, 1958).

In their pure forms, the subacute and chronic lesions of localized penetrating type which were encountered exactly resembled peptic ulcers of the stomach itself; every important feature in the classical descriptions of Hurst and Stewart (1929) could be matched in these oesophageal lesions. It is of course impossible to determine whether the ulceration occurred originally in mucosa of squamous or of gastric type; in each case, however, mucosa of gastric cardiac type lined a part at least of the edge of the crater. The chronic ulcers of this kind, which were associated with considerable extensions of gastric mucosa into the lower oesophagus, were clearly similar to those described by Tileston (1906), by Stewart and Hartfall (1929), and by Lyall (1937). Barrett (1950) was the first to recognize their true nature. Subacute ulceration similar to that described here, occurring in an oesophagus lined extensively by gastric mucosa, has been reported by Bosher and Taylor (1951), by Allison and Johnstone (1953), and by Peters (1958). The extensive submucosal fibrosis which accompanied these quite small and shallow erosions, and the architectural irregularity of the surrounding mucosa, were probably the result of the healing of other and perhaps more widespread lesions of this type. Despite its extent, the fibrosis accompanying these lesions did not appear to cause any appreciable narrowing of the oesophageal lumen.

Of the 46 specimens examined, no less than 26 showed chronic superficial oesophagitis in combination with one of the varieties of localized penetrating ulceration; in 23 cases the penetrating ulcer was chronic. The penetrating components of these combined lesions were in all cases similar in their pathological anatomy to those seen as solitary

lesions, save that the upper margin of each penetrating ulcer blended with the ulceration of superficial type which extended upwards from it, and thus lacked any mucosal lining. Gastric mucosa lined a part, and in most cases a considerable proportion, of the edge of the penetrating ulcer, even in the two cases in which the floor of the ulcer had become covered by squamous epithelium.

The finding of chronic localized penetrating ulcers, alone or in combination with chronic superficial oesophagitis, in as many as 25 out of the 46 cases examined is in striking contrast to the reports of other writers. Thus Lyall (1937) recognized only one such lesion among the eight cases of chronic oesophageal inflammation which he described, and Allison and Johnstone (1953) concluded from their investigations that penetrating ulcers could be expected in roughly 10% of cases of chronic oesophagitis with stenosis. Peters (1955), though he gave no indication of the actual proportion of his cases which showed penetrating ulceration, made it clear that he regarded such lesions as unusual. It is evident, however, from Lyall's description that in some of his cases of the more diffuse type of oesophagitis there was in some part of the eroded area a small zone of deep penetration. It seems probable that these, and the cases of similar type reported by Peters, were in fact examples of the combined lesion described here. The lesion which Wolf, Som, and Marshak (1953) called 'marginal' or 'oesophago-gastric ulceration', though not fully studied from the pathological standpoint, showed many similar features and was probably also of this combined type. There is no doubt that the penetrating component in many of the cases in the present series would not have been found had the histological examination been less searching, owing to the tendency for the crater of the penetrating ulcer to become obliterated in the manner which has been described. It may well be that the discrepancy between the observations recorded here and those of other writers is due principally to differences in the method of examination employed.

It is most unlikely that the sharply localized penetrating components of these combined lesions, involving only a relatively small segment of the oesophageal circumference, and usually or invariably confined to the posterior and anterior walls, were due solely to the erosive action of regurgitated gastric secretions. It is generally accepted that other factors, at present unidentified, are involved in the production of structurally identical lesions in the stomach and duodenum, and it seems probable that similar factors are necessary for the development of such lesions in the oesophagus. There can be no doubt, nevertheless, that gastric secretions do play an

important part in their production. Since the intrusions of fundal mucosa into the oesophagus in these cases were never sufficiently large to have produced a significant quantity of acid and pepsin, it must be assumed that these secretions were derived from the stomach by reflux. Incompetence of the oesophago-gastric sphincter must therefore have preceded the development of the oesophageal lesion, and cannot have been caused by it in the manner suggested by Dey, Gilbert, Trump, and Roskelley (1946).

It has been suggested that the degree and length of the stenosis in chronic oesophagitis may be a reliable guide to the nature of the underlying lesion. Barrett (1950) noted that clinical evidence of oesophageal narrowing had not been present in most of the cases of penetrating ulceration reported in the literature. Wolf *et al.* (1953) stated that severe and long stenosis of the oesophagus was associated with persistent vomiting or long-continued gastric intubation, whereas short stenosis was found in cases of hiatus herniation with reflux, and in association with the lesion which they called marginal or oesophago-gastric ulceration. In the present series of cases no correlation was found between the length of the stenosed segment and the type of oesophageal ulceration present. Long stenosis, though rather more common in association with chronic superficial oesophagitis, also occurred in a case of pure chronic penetrating ulceration, and was not uncommon in cases showing combined lesions. Conversely, short stenosis occurred in some cases of pure chronic superficial oesophagitis.

The glandular mucosa which extended into the lower part of the oesophagus in almost every case in the series, though showing architectural distortion and often intestinal metaplasia with goblet cell formation, appeared to be essentially similar to that normally found in the cardia of the stomach; there seemed to be no justification for regarding it as a persistence of the embryonic columnar epithelial lining of the oesophagus (Barrett, 1958). Allison and Johnstone (1953) suggested that the anomaly might be due to upward growth of mucosa from the cardia during the healing of ulceration in the lower oesophagus. The minimal submucosal fibrosis and the presence of undamaged submucosal mucous glands in this zone in most cases argue strongly against any such explanation in the present series, and it therefore seems probable that these mucosal anomalies are of congenital origin. Although mucosa of this type may to some extent protect the oesophagus against the erosive action of regurgitated gastric secretions, it is evident that under appropriate circumstances it may undergo penetrating ulceration as severe as that occurring in the stomach.

CLINICAL FEATURES

A study of the clinical records of all the cases failed to reveal any distinct pattern of symptoms whereby the different pathological types of lesion might be distinguished clinically. A few general observations are perhaps worth recording.

AGE INCIDENCE The findings in this relatively small series of cases do not justify any firm conclusion as to the age incidence of the various types of lesion. Of the 16 cases of pure chronic superficial oesophagitis, however, 11 had symptoms dating from infancy or childhood, whereas of the 25 cases showing chronic penetrating ulceration, alone or combined, 19 first had symptoms of an oesophageal lesion at or over the age of 40 years. In this series the great majority of adults showed chronic penetrating ulceration: in the children the pure chronic superficial lesion alone was usually present.

SYMPTOMS The major symptoms were pain, regurgitation and vomiting, and difficulty in swallowing. Haemorrhage was uncommon. Pain was rather more common in cases showing penetrating ulceration than in pure chronic superficial oesophagitis, but did not invariably accompany even severe chronic penetrating ulceration. In its site and distribution there was no clue to the nature of the underlying lesion. Regurgitation of food and fluid was common with all types of lesion. Vomiting also was common, but was a late symptom in the majority of cases, evidently due to the development of oesophageal obstruction. This contrasts with the findings of certain American authors, notably Wolf *et al.* (1953), who maintained that chronic oesophagitis of the type producing long stenosis is very rare except as a sequel to prolonged and persistent vomiting. Gastric and duodenal ulceration, noted by these and other writers as common concomitants of this type of oesophagitis, were not demonstrated in any of the cases of pure chronic superficial oesophagitis in this series, nor was there a history of prolonged gastric intubation (Lodge, 1955) in any case.

Difficulty in swallowing was a common complaint whatever the type of the underlying lesion. It was almost always associated with considerable narrowing of the oesophageal lumen by fibrosis, but in a few cases in which there was no stenosis it was presumably due either to muscular spasm or more probably to impairment of the peristaltic function of the organ by severe interstitial fibrosis. Haemorrhage was noted in 13 cases. In nine of these it was very slight and occasional. In three cases only was there a history of severe and acute haematemesis, and in each of these there was a chronic penetrating

lesion, one in its pure form, the other two in combination with chronic superficial oesophagitis.

SUMMARY

Specimens removed by oesophago-gastrectomy from 46 cases of severe chronic oesophagitis were examined histologically. Two distinct types of lesion were found: chronic superficial oesophagitis and localized penetrating ulceration.

Chronic superficial oesophagitis was characterized by irregular but invariably shallow ulceration, associated with oedema, inflammatory cellular infiltration and fibrosis in all layers of the oesophageal wall, but especially in the submucosa.

Localized penetrating ulceration was found in subacute and chronic forms. These lesions in their histological structure closely resembled subacute and chronic peptic ulcers of the stomach, and were in all cases closely related to intrusions of mucosa of gastric cardiac type into the lower oesophagus.

The two basic types of lesion were found in combination in 26 of the 46 cases. In 23 of these, localized penetrating ulceration of chronic type was combined with chronic superficial oesophagitis.

The high proportion of cases showing chronic localized penetrating ulceration, alone or in combination with chronic superficial oesophagitis, is in marked contrast to the findings of other writers. It is suggested that the method of examination employed in this study permitted the detection of chronic penetrating ulceration of gastric type in the oesophagus in many cases which would otherwise have been regarded as examples of pure chronic superficial oesophagitis, and that penetrating ulceration is in fact far more common in the oesophagus than is generally appreciated.

Examination of the clinical records did not reveal any clear-cut pattern of symptoms by which the two types of pathological lesion could be distinguished clinically. Massive haemorrhage occurred only in the presence of localized penetrating ulceration. Pure chronic superficial oesophagitis was found more commonly in children and in those whose symptoms dated from childhood than was chronic localized penetrating ulceration, which was found chiefly in patients whose symptoms first appeared at or over the age of 40 years.

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REFERENCES

- Allison, P. R. (1946). Peptic ulcer of the esophagus. *J. thorac. Surg.*, **15**, 308-317.
- (1948). Peptic ulcer of the oesophagus. *Thorax*, **3**, 20-42.
- (1951). Reflux esophagitis, sliding hiatal hernia, and the anatomy of repair. *Surg. Gynec. Obstet.*, **92**, 419-431.
- , and Johnstone, A. S. (1953). The oesophagus lined with gastric mucous membrane. *Thorax*, **8**, 87-101.
- Atkinson, M., Edwards, D. A. W., Honour, A. J., and Rowlands, E. N. (1957). Comparison of cardiac and pyloric sphincters. A manometric study. *Lancet*, **2**, 918-922.
- Barrett, N. R. (1950). Chronic peptic ulcer of the oesophagus and 'oesophagitis'. *Brit. J. Surg.*, **38**, 175-182.
- (1958). The lower oesophagus lined by columnar epithelium. In *Modern Trends in Gastro-enterology* (2nd ser.), ed. F. Avery Jones, pp. 147-162. Butterworth, London.
- Bosher, L. H., and Taylor, F. H. (1951). Heterotopic gastric mucosa in the esophagus with ulceration and stricture formation. *J. thorac. Surg.*, **21**, 306-312.
- Botha, G. S. M. (1958). Mucosal folds at the cardia as a component of the gastro-oesophageal closing mechanism. *Brit. J. Surg.*, **45**, 569-580.
- , Astley, R., and Carré, I. J. (1957). A combined cineradiographic and manometric study of the gastro-oesophageal junction. *Lancet*, **1**, 659-662.
- Creamer, B. (1955a). Oesophageal reflux. *Ibid.*, **1**, 279-281.
- (1955b). Oesophageal reflux and the action of carminatives. *Ibid.*, **1**, 590-592.
- , and Pierce, J. W. (1957). Observations on the gastrooesophageal junction during swallowing and drinking. *Ibid.*, **2**, 1309-1312.
- Dey, F. L., Gilbert, N. C., Trump, R., and Roskelley, R. C. (1946). Reflex shortening of the esophagus in the experimental animal with the production of esophageal hiatus hernia. *J. Lab. clin. Med.*, **31**, 499-506.
- Dornhorst, A. C., Harrison, K., and Pierce, J. W. (1954). Observations on the normal oesophagus and cardia. *Lancet*, **1**, 695-698.
- Fyke, F. E. Jr., Code, C. F., and Schlegel, J. F. (1957). The gastro-oesophageal sphincter in healthy human beings. In *Proceedings of the International Congress of Gastroenterology*, London, 1956, pp. 3-18.
- Hurst, A. F., and Stewart, M. J. (1929). *Gastric and Duodenal Ulcer*. Oxford University Press, London.
- Jackson, C. (1929). Peptic ulcer of the oesophagus. *J. Amer. med. Ass.*, **92**, 369-372.
- Kelly, A. B. (1930). Congenital stenosis of the oesophagus in children, associated with diaphragmatic hernia of the stomach. *Proc. roy. Soc. Med.*, **23**, 1521-1526.
- (1936). Some oesophageal affections in young children. *J. Laryng.*, **51**, 78-99.
- Lendrum, A. C. (1941). The preparation of tissues for paraffin embedding. *J. Path. Bact.*, **52**, 138-142.
- Lodge, K. V. (1955). The pathology of non-specific oesophagitis. *Ibid.*, **69**, 17-24.
- Lyall, A. (1937). Chronic peptic ulcer of the oesophagus: a report of eight cases. *Brit. J. Surg.*, **24**, 534-547.
- Peters, P. M. (1955). The pathology of severe digestion oesophagitis. *Thorax*, **10**, 269-286.
- (1958). The congenital short oesophagus. *Ibid.*, **13**, 1-11.
- Quinke, H. (1879). Ulcus oesophagi ex digestionem. *Dtsch. Arch. klin. Med.*, **24**, 72-79.
- Schumacher, S. (1927). "Die Speiseröhre." In *Handbuch der mikroskopischen Anatomie des Menschen*, ed. W. von Möllendorff, Vol. V, Pt 1, pp. 301-336. Springer, Berlin.
- Stewart, M. J., and Hartfall, S. J. (1929). Chronic peptic ulcer of the oesophagus. *J. Path. Bact.*, **32**, 9-14.
- Taylor, A. L. (1927). The epithelial heterotopias of the alimentary tract. *Ibid.*, **30**, 415-449.
- Tileston, W. (1906). Peptic ulcer of the oesophagus. *Amer. J. med. Sci.*, **132**, 240-265.
- Wolf, B. S., Som, M., and Marshak, R. H. (1953). Short esophagus with esophagogastric or marginal ulceration. *Radiology*, **61**, 473-495.