Annals of the Royal College of Surgeons of England (1977) *vol* 59

Role of ischaemia in the initiation of peptic ulcer

Christopher Piasecki MB BS PhD

Senior Lecturer, Department of Anatomy, Royal Free Hospital School of Medicine, London

Summary

Study of intramural vascular pathways to the human gastroduodenal mucosa shows that some persons have patches of mucosa supplied by end arteries. These patches occur only in the ulcer-bearing areas. It is suggested how this may contribute to the initiation of peptic ulceration and explain some of its features.

Introduction

The factors which are still attracting most research into the cause of peptic ulceration—namely, acidity and the mucous barrier[1-3]—do not explain what are perhaps the most outstanding features of the disease. These are: (1) the fact that peptic ulcers usually occur singly and (2) the remarkable localization of gastric ulcers to the lower parts of the lesser curvature and of duodenal ulcers to the anterior and posterior walls of the first 2 cm of the pylorus. This suggests the operation of strong localizing factors, but these have not received much attention.

Many local mechanisms have been suggested, but they lack anatomical and clinical evidence of their real involvement. These include local pressure[1], effect of a 'pyloric jet'[4], islets of ectopic mucosa[1, 2], and the association of ulcers with the fundopyloric mucosal boundary and well-defined muscle bundles[5].

However, a local vascular factor—always to be suspected when localization is prominent—is supported by two pieces of evidence. Clinically, it is well known that pulling the stomach downwards and to the left at operation results in pallor on the anterior wall of the first 2.5 cm of the duodenum (known as 'Mayo's anaemic spot'). This pallor disappears on relaxing the tension[4, 6]. Anatomically, postmortem injection studies in man reveal a poor vascularity in the lesser curvature and first part of the duodenum relative to other areas[7-9]. (This has recently been highlighted by the occasional occurence of ischaemic necrosis of the lesser curvature following highly selective vagotomy.) This evidence stimulated a more detailed study of the vascular patterns in man.

Methods

The stomach, duodenum, and pancreas were removed en bloc post mortem from subjects with no history of gastrointestinal disorder. The arterial system was injected with ink and gelatin, the tissues dehydrated and cleared, and the vessels examined by stereomicroscopy.

Results

The poverty of vascularity in the lesser curvature, found by previous authors, was shown to be localized still further. In the lesser curvature there was a quantitative decrease in number and calibre of anastomosing vessels from the cardiac to the pyloric parts. In the duodenum there was a similar decrease from the second part to the pylorus—that is, the plexus was least dense in the pyloric third of the lesser curvature and in the first 2.5 cm of the duodenum.

A still more significant finding was that in the areas of the stomach wall with the poorest plexus there were small areas with no plexus at all. The mucosa underlying these patches was supplied by mucosal arteries which arose outside the stomach wall, pierced the main muscle, and did not communicate with the submucous plexus. They might be named 'mucosal end arteries of extramural origin'. Such patches were found in 5 out of 9 stomachs and were commonest in the pyloric third of the lesser curvature ($\chi^2 = 0.001$). Although the sample was small, these findings do show that some individuals possess

Based on an Arnott Demonstration presented on 4th May 1976

patches of gastric mucosa with this pattern of supply (95% confidence limits for 5/9 are 19–81% of the population). Such patches were also found in 7 out of 10 duodenal specimens (95% confidence limits 26–96%) and all were confined to the first 1.5 cm of the organ, where they were randomly distributed about the circumference. Figure 1 (*a*) shows the distribution of these patches and it is evident that this closely mimics the distribution of chronic peptic ulcers.

Suggested mechanism of ulceration based on local factors

Since the occurrence of patches of mucosa supplied by end arteries is the closest known association between an anatomical feature and the site of ulceration it must be strongly suspected of being involved in the aetiology. Hence the following mechanism is proposed. Figure 1 (*b*) shows the basic pattern of blood supply to such a patch. On principle, contraction of the muscle at 'A' would not affect the blood flow to the mucosa at 'a' owing to the collateral circulation in the submucous plexus. However, contraction at 'B' involving an end artery of extramural origin might, if prolonged, result in ischaemia in a local area of mucosa at 'b'. Once ischaemia was sufficiently prolonged to lower the vitality of the mucosa the acid secreted by the adjacent fundic mucosa would attack the ischaemic area and prevent it from healing.

The mechanism of prolonged contraction is likely to be different in gastric and in duodenal ulcers. In the stomach contractions are most prolonged in the lesser curvature, where the 'standing wave' is seen radiologically in the incisural region. In the duodenum the same mechanism cannot be postulated since ulcers do not usually underly the pyloric muscle, and the patches of mucosa supplied extramurally are randomly distributed around the circumference of the first 1.5 cm while ulcers are mainly on the anterior or posterior wall. The clue to the mechanism in the duodenum is provided by the phenomenon of Mayo's anaemic spot. In this manoeuvre the downward pull of the stomach will be resisted by an opposing force in the hepatoduodenal ligament (Fig. 2). This tug-of-war of opposing forces will be transmitted across the anterior and posterior duodenal walls. The diffuse attachment of the hepatoduodenal ligament to the anterior and posterior walls will minimize the force transmitted by the superior wall, while the inferior wall would escape as it is not caught in the line of opposing forces. Mucosal arteries of extramural origin on the anterior and posterior walls would therefore be most vulnerable to the effects of tension. Thus localization of the resultant ischaemia would be correlated exactly with the sites of duodenal ulceration. This mechanism is supported by the pale appearance of Mayo's 'spot' and by Kirk's finding[4] of palpable tension at this site. In everyday life this mechanism might be active when the stomach moves to the left and downwards when overfilled by an unusually large meal, as occurs in overeating caused by worry (or from sheer gluttony!).

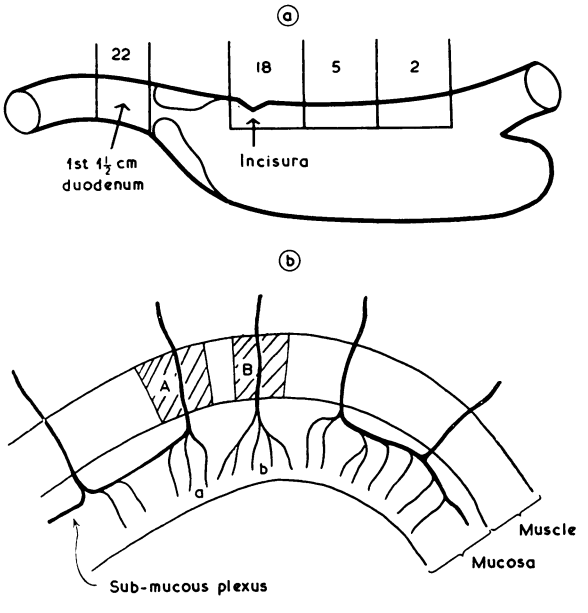

FIG. 1 (*a*) *Scheme showing approximate position of 25 gastric and 22 duodenal mucosal arteries of extramural origin (total from 7 specimens. (b) Scheme showing a mucosal artery of extramural origin (centre) passing through muscle, having no connection with the submucous plexus, and supplying a patch of mucosa 'b'. (Reproduced by permission from the* Journal of Anatomy[8]).

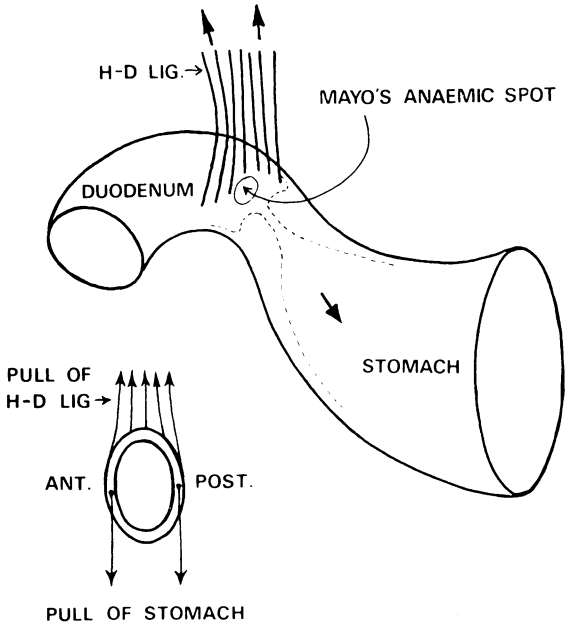

FIG. 2 *Scheme showing forces involved during production of Mayo's anaemic spot. H–D lig: hepatoduodenal ligament. (Reproduced by permission from the* British Journal of Surgery[9]).

Explanation of other features of the disease

It is evident, therefore, that local anatomico-physiological factors are capable of explaining the initiation of chronic peptic ulceration. No single factor alone is responsible, and it seems that the coexistence of a patch of mucosa supplied by a mucosal end artery of extramural origin with prolonged contraction of muscle is necessary. These two features also explain the singleness of most ulcers as well as the occurrence of 'kissing' ulcers in the duodenum. The fact that only some individuals have such end arteries and that coexistence of both features at one site is necessary for ulceration explains why most of us do not suffer from the disease in spite of exhibiting other features such as stress and hyperacidity.

The same argument might account for the familial, genetic, and geographical features of peptic ulceration, because the possession of a particular anatomical pattern of vessels (and psychosomatic reaction) is most likely to be genetically determined (just as are external facial features in different families and races). This is supported by the fact that certain genetically determined features are known to be associated with ulceration. Thus in persons of Blood Group O the incidence of duodenal ulceration is increased threefold over the population at large (while Group A is associated with hypoacidity and gastric cancer)[10, 11]; persons whose saliva lacks certain genetically inherited mucopolysaccharides, the ABH substances, have an abnormally high incidence of peptic ulcer[12]; and Fodor *et al*[13] have demonstrated an additive effect of Group O, lack of salivary ABH, and hypersecretion.

From the absence of the disease in animals it would be expected that the causal factor operative in man is absent in animals. So far it has been shown that dogs' stomachs do not have any areas of poor vascularity and there are no extramural end arteries[14]. On the theory outlined above the absence of this one feature would suffice to explain the absence of the disease in dogs and it will be interesting to see the vascular patterns in the stomachs of other species.

I would conclude that this multiple aetiology of localizing factors accounts for the initiation and localization of peptic ulcers and for most of the salient features of the disease while not precluding the influence of stress and acidity.

References

1 Ivy, A C, Grossman, M I, and Bachrach, W H (1950) *Peptic Ulcer.* London, Churchill.
2 Spira, J (1956) *Gastroduodenal Ulcer.* London, Butterworth.
3 Baron, J H (1972) in *Chronic Duodenal Ulcer,* ed. Wastell, MS, ch. 2. London, Butterworth.
4 Kirk, R M (1968) *Gut, 9,* 414.
5 Oi, M, *et al.* (1969) *Gastroenterology, 57,* 280.
6 Mayo, W J (1908) *Surgery, Gynecology and Obstetrics, 6,* 600.
7 Piasecki, C (1973) Ph D Thesis, University of London.
8 Piasecki, C (1974) *Journal of Anatomy, 118,* 295.
9 Piasecki, C (1971) *British Journal of Surgery, 58,* 660.
10 Bralow, S P (1969) *American Journal of Digestive Diseases, 14,* 655.
11 Menguy, R (1970) *American Journal of Surgery, 120,* 282.
12 Yager, J, and Weiner, H (1971) in *Advances in Psychosomatic Medicine,* vol. 6, ed. Weiner, H, p. 40. Basel, Springer-Karger.
13 Fodor, O, *et al.* (1968) *American Journal of Digestive Diseases, 13,* 260.
14 Piasecki, C (1975) *Journal of Anatomy, 119,* 133.