BLOOD VESSELS OF A GASTRIC ULCER

BY

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[WITH SPECIAL PLATE]

It is no new idea that acute ischaemia can cause necrosis of the gastric mucosa. Disse (1904) claimed that the arteries supplying the gastric mucosa in man are virtually end-arteries, each one supplying 4 mm.^{2} of surface mucosa, and that blockage of one or more of these vessels would cause local necrosis. Various theories have been suggested by Hofmann and Nather (1921) on how such a block may be produced. They present a long list from which to choose, including compression from without by the muscle coats of the stomach, spasm of the arteries, and thrombosis and embolism.

Recent work by Barlow (in the press) shows, however, that the vessels of the gastric submucosa and mucosa are not anatomical end-arteries, but that they anastomose freely with each other. Examination of Barlow's dissections suggests that it would require an extensive vascular block to render a patch of mucosa ischaemic. It is true, nevertheless, that the vessels in and around a chronic gastric ulcer show widespread occlusions; but in an acute ulcer this is by no means the case.

During the past year specimens of gastric ulcer removed at operation in our department have been examined by the methods of microangiography, designed to show the vessels without impedance of other tissues.

Method

On removal of the resected portion of stomach at operation the specimen is placed in a saline bath at 37° C., and kept there until injection is completed. A suitable artery, usually the left gastric or right gastro-epiploic, and the largest of the accompanying veins are cannulated. The cut ends of the stomach are clamped or oversewn to prevent leakage. A 10% solution of colloidal silver iodide is then injected into the artery at a pressure of not more than 160 mm. Hg, 70–90 ml. being the average quantity entering the vessels. In some cases 10–15 ml. of a solution of 10% bismuth oxychloride ("chlorbismol") is injected after the silver iodide. Bismuth oxychloride has a particle size too large to enter the capillaries, and it outlines the larger vessels very clearly. The vessel used for injection is then ligated, the stomach filled with cotton-wool to limit distortion, and the whole specimen immersed in 10% formalin for 24 hours.

After fixation the stomach is cut along the greater curvature, opened out mucosa uppermost, and radiographed. A further period of formalin fixation follows for 24–48 hours. Transverse sections 400 μ thick are made through the ulcer itself and for varying distances proximal and distal to it along the line of the lesser curve. These sections are then examined by the technique of microarteriography (Barclay, 1947).

Results

Normal Stomach

A transverse section of the normal healthy stomach in the line of the lesser curve shows a characteristic vascular pattern (Plate, Fig. 4), so regular and well defined that it is easy to detect the normal anatomical layers of the stomach wall. The vessels of the mucosa are rich and complex and those of the underlying submucosa present a typical ordered pattern. The vessels supplying the external muscle coats are seen as a longitudinal band running across the zone near the bottom of the section.

Chronic Ulcer

Figs. 1 and 2 show radiographs of two stomachs each containing a chronic gastric ulcer of the lesser curve, the first situated at the centre of the lesser curve and the other approximately 5 cm. from the pylorus. In both there is a well-marked ischaemic zone around the ulcer in which the larger vessels do not outline. Elsewhere the vessels of the stomach show normal filling.

Section through the ulcer itself shows that the floor of the ulcer consists of bloodless tissue throughout the whole thickness of the stomach wall; in this area there is a complete absence of all but very fine vessels (Fig. 3). The vessels of the mucous membrane, on the other hand, are normally filled right into the edge of the ulcer. The general picture contrasts sharply with that seen in a transverse section of the normal stomach.

Furthermore, sections taken from the lesser curve some distance from a chronic ulcer show that these changes extend as an ischaemic core quite deep to the mucosa for 1–2 cm. along the lesser curve. Fig. 6 shows such a section taken 1.6 cm. from the edge of a chronic gastric ulcer. There is a well-marked ischaemic zone in the centre of the section, with absence of all but very fine vessels. The overlying submucosa shows a marked derangement of the normal vascular pattern, although the vessels of the mucous membrane are normally filled. The appearances are those of an ischaemic process deep in the stomach wall, which undermines and eventually destroys the mucosa.

This method of microradiography demonstrates vividly the old pathological teaching, extensive vascular block and derangement around a chronic ulcer. It has been a constant finding in all the specimens of chronic gastric ulcer examined.

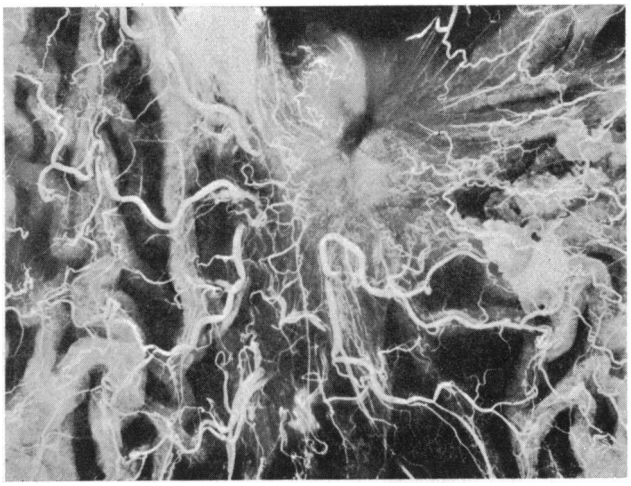
Such an ischaemic state might well explain why an ulcer does not heal, but rather persists and even spreads. It is not, however, evidence that such a vascular condition caused the original ulcer. It is indeed much more probable that the vascular block is the result of a chronic inflammatory state producing changes not dissimilar to those found in chronic ulceration elsewhere in the body.

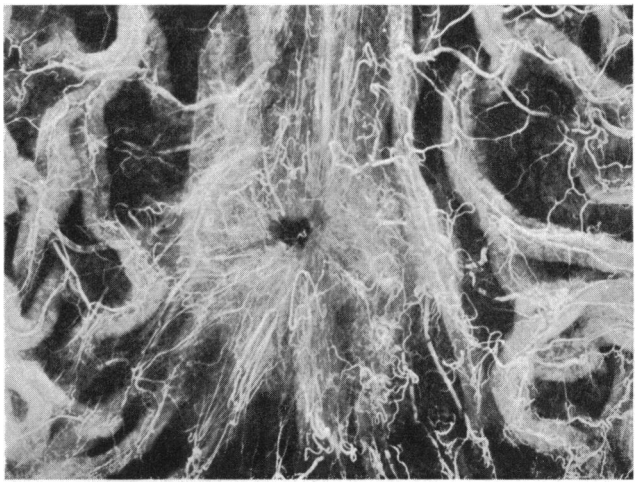
An attempt was made, therefore, to find and examine small acute ulcers. Such ulcers are not commonly found in gastrectomy specimens, but occasional ones were detected remote from chronic ulcers and also in specimens removed urgently for haematemesis.

Acute Ulcer

The typical appearance of a transverse section through an acute ulcer is shown in Fig. 5. There is a marked increase in the blood vessels in the submucosa. Their arrangement is haphazard and diffuse, and the alteration of vascular pattern in the submucosa extends widely beyond the point where the mucosa is breached. The mucosal vessels themselves are well filled and appear to be normal right into the ulcer defect, and at one edge the mucosa has collapsed almost as if the foundations on which it rests have been disrupted.

The normal pattern (Fig. 4) contrasts as vividly with the vessels of an acute ulcer as it does with the appearances in a chronic ulcer, but in a very different way. The acute ulcer presents a picture of an acute vascular disturbance





FIGS. 1 and 2.—Surface radiographs (× 78) of two specimens of chronic gastric ulcer situated on the lesser curve. The vessels have been injected with colloidal silver iodide. Each ulcer shows a well-marked surrounding ischaemic zone with absence of all but fine vessels.

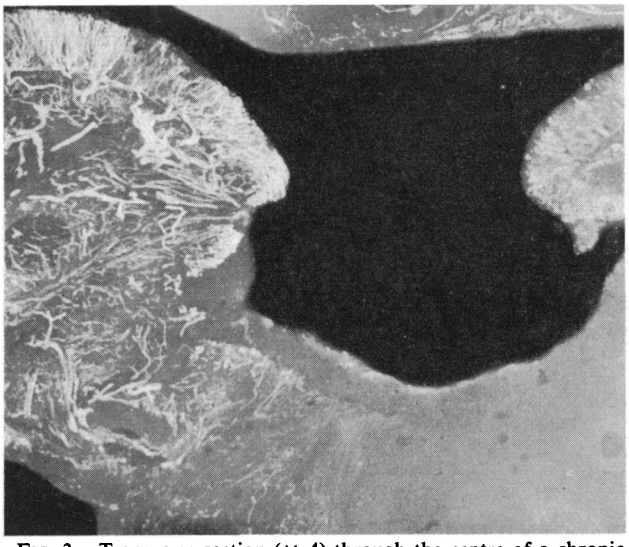


FIG. 3.—Transverse section (× 4) through the centre of a chronic gastric ulcer showing the well-marked ischaemic base, with only an occasional fine vessel running through it, and the normal filling of the vessels of the mucous membrane right up to the ulcer edge. (Section 400 μ thick.)

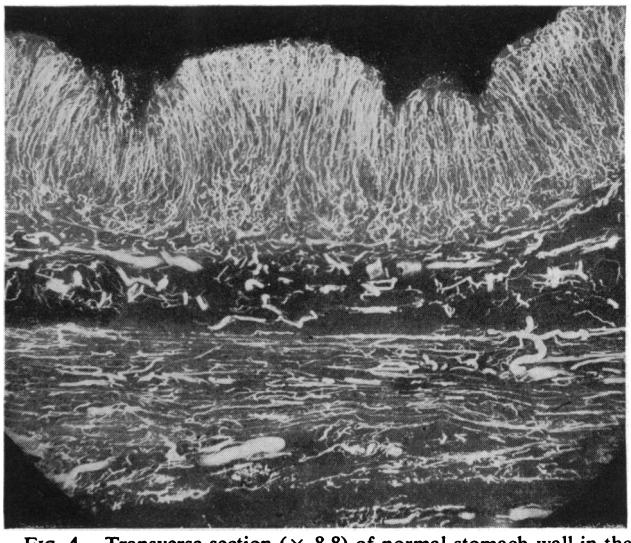


FIG. 4.—Transverse section (× 8.8) of normal stomach wall in the centre of the lesser curve. The vessels in the mucosa are well filled and the normal vessel pattern in the underlying submucosa is clearly seen. The fine vessels running longitudinally near the bottom of the section are those of the external muscle coats. (Section 400 μ thick.)

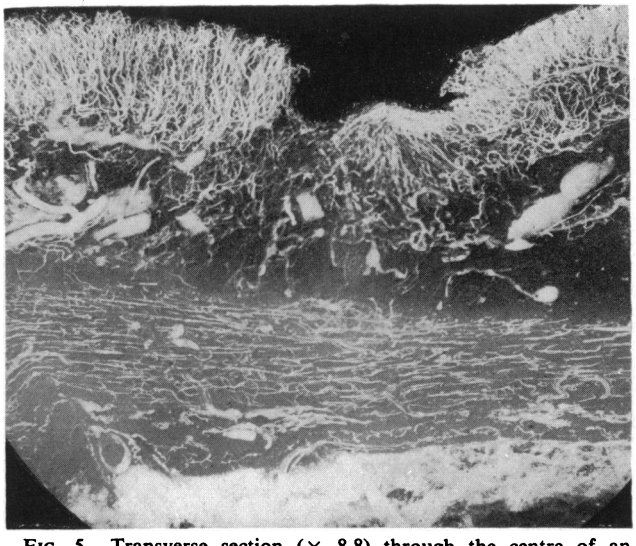


FIG. 5.—Transverse section (× 8.8) through the centre of an acute ulcer showing an increase in the size and number of vessels. Their arrangement is haphazard and diffuse and extends widely beyond the point where the mucosa is breached. (Section 400 μ thick.)

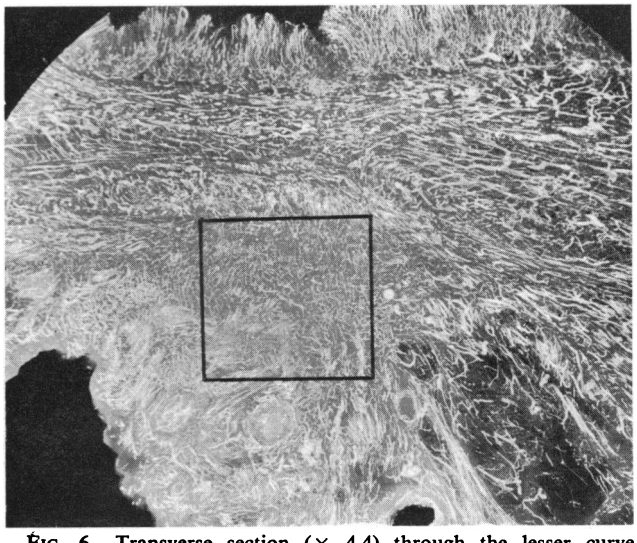


FIG. 6.—Transverse section (× 4.4) through the lesser curve 1.6 cm. from the edge of a chronic gastric ulcer. The outlined ischaemic area deep in the muscle wall is shown containing only fine vessels. (Section 400 μ thick.)

occurring chiefly just under the mucous membrane—a hypervascularity and derangement, an increase in vessels, and not an ischaemia.

Discussion

The method of studying vascular patterns in simple gastric ulcer by microradiography lends support to the hypothesis that in peptic ulcer an acute vascular disturbance occurs in the submucosa close to the mucous membrane, and that the breach of the mucosa itself results from this underlying vascular derangement.

Such a hypothesis is of interest when compared with the observations of Wolf and Wolff (1943) on their patient, Tom, who had a large gastric fistula. They noted that in states of acute emotion the exposed gastric mucosa became engorged and turgid, and finally produced here and there an acute erosion which bled from its congested edges.

The nature of the vascular disturbance found in association with acute ulcers has not so far been determined, but further studies by microradiography, and by coloured intravascular injections of arteries and veins, show that venous stasis is a predominant feature. That such a type of acute vascular reaction can produce ulceration elsewhere in the body is common knowledge. The surgeon called upon to operate on strangulation of the bowel often sees it.

It should be of interest to pursue the study of the vascular derangement of the stomach mucosa.

Summary

The vascular pattern of the normal lesser curve of the stomach, of a chronic ulcer, and of an acute ulcer, as shown by microradiography, are compared.

In a chronic ulcer there is widespread vascular occlusion in the base of the ulcer and extending along the line of the lesser curve deep to the mucosa.

In an acute ulcer there is an acute vascular disturbance occurring chiefly just under the mucous membrane—a hypervascularity and not an ischaemia.

It is a pleasure to acknowledge the continued advice of Professor F. H. Bentley in this investigation, and the assistance of Mr. J. E. Rogers, Mr. W. G. Leslie, and Mr. J. B. Wilson, of the departmental laboratories, in the preparation of specimens and microradiographs.

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The shock of the two colliery disasters, at Knockshinnoch and at Creswell, which took place within three weeks of each other in September, may have obscured the fact that mining is actually getting safer in this country. Accident statistics recently issued by the Ministry of Fuel and Power in its *Statistical Digest for 1948 and 1949* display very clearly that, though between the wars there was little or no improvement in the safety record, since 1941 every year except 1947 has shown a further decrease in deaths and reportable injuries. Thus the numbers killed in 1920, 1930, and 1940 were 1,103 (or 0.88 per 1,000 men employed), 1,013 (1.07 per 1,000), and 923 (1.20 per 1,000) respectively, whereas in 1945 the figure had dropped to 550 (0.76), and in 1948 and 1949 had sunk to 460 (0.62)—less than half the average pre-war value. The reportable injuries show a similar though less striking trend: 4,287 (3.4 per 1,000) in 1920, 3,237 (4.2) in 1940, but only 2,180 (2.9) in 1949.

OBSERVATIONS ON BLOOD FLOW IN HUMAN INTESTINE

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In the past most of our information concerning the visceral circulation has been derived from animal experiments. It is difficult, however, to apply the results of such experiments direct to man, for anaesthesia, operative interference, and species difference are incalculable factors. There is urgent need for the better understanding of circulatory responses in human viscera, especially as regards their relation to the control of peripheral resistance. Indirect studies have been made in such structures as the liver (Bradley *et al.*, 1945) and the kidney (Goldring *et al.*, 1940).

Friedman and Snape (1946) and Shoshkes (1948) observed colour changes in colostomies and used them mainly in the investigation of local applications of drugs. Almy and Tulin (1947) made direct proctoscopic observations in young adults to demonstrate colonic responses to stress, but the role of the intestinal blood vessels in the integration of the circulation as a whole is still inadequately understood.

In a previous investigation of the problem I also used patients on whom the operation of colostomy had been performed, employing the temperature and colour of the intestinal protrusions surrounding the abdominal stoma to detect intestinal blood-flow change in these subjects. It was shown that vascular responses in the skin were often accompanied by opposite reactions in the bowel (Grayson, 1949a). Thus cutaneous vasodilatation produced by warming the body was accompanied by a pronounced diminution in bowel blood flow, whereas during the cutaneous vasoconstriction produced by cooling a limb an increase in bowel blood flow occurred. Again, during the spontaneous fluctuations which occasionally occur in the skin of a subject at rest, opposite fluctuations were usually observed in the bowel. In other experiments the intravenous administration of the dihydrogenated alkaloids of ergot—"hydergine" or dihydroergocornine—which act mainly as cutaneous vasodilator drugs, was followed by a marked diminution in bowel blood flow which accompanied the rise in skin blood flow (Grayson and Swan, 1950).

Although such opposed vascular responses have been a striking feature of the previous investigations, the skin and bowel circulations do not inevitably show opposite responses to all stimuli. For example, intravenous adrenaline or *nor*adrenaline produces vasoconstriction in both skin and bowel (Grayson and Swan, 1950). Again, where prolonged heating of the body produces a rise in rectal temperature the initial diminution in bowel blood flow already referred to is followed by an increase. Similarly, in experiments where the rectal temperature was lowered a diminution in bowel blood flow occurred (Grayson, 1949b). In these examples the blood flow changes in skin and bowel were similar and not opposite.

The present paper, however, is concerned only with the opposed reactions that take place between skin and bowel and their possible significance in the maintenance of blood pressure.

Methods

The temperature of any animal tissue exposed to an environment at a temperature different from that of the blood depends in part on the blood flow through the organ and can be used to detect blood-flow change. This