These clinical and laboratory findings suggested a reduction in his blood-volume due to extravasation of plasma and made renal tubular necrosis unlikely. The transfusion of a further three bottles of plasma restored the pulse and blood-pressure to normal and reversed the peripheral vasconstriction. Shortly afterwards there was a marked diuresis, with a corresponding fall in body weight and reduction in the circumference of his swollen limbs (see Fig. 2). His haemoglobin fell to 92% while his plasma protein concentration remained unchanged. Serial electrocardiograms showed transient ST and T-wave changes, but there was at no time evidence of jugular or pulmonary congestion.

Four weeks after admission to hospital he was discharged, having apparently made a complete recovery.

Fig. 2.—Changes in body weight and circumference during recovery.

**COMMENT**

The compression and decompression routine followed in this patient before his collapse was in accordance with normal industrial practice, and it cannot be established when extravasation of plasma began. In retrospect it seems possible that the clinical course might have been more favourable if the third decompression had been halted at 25 ft. (7.6 m.) (11 lb./sq. in.–0.77 kg./sq. cm.) when symptoms recurred.

While it is clear that plasma infusion was life-saving in this patient, the part played by decompression in enabling him to recover from the hypovolaemic shock is uncertain. In the cases described by Brunner et al. (1964) decompression was probably unimportant. Nevertheless if similar cases are encountered it will probably be wise to treat them with simultaneous decompression and plasma transfusion. It should be pointed out that the most suitable air inlet for the transfusion bottles is a needle or glass tube long enough to project above the surface of the plasma when the bottle is inverted, and this must be inserted before compression.

If the above recommendations are adopted, it will be necessary to provide transfusion equipment and a supply of plasma or a plasma expander wherever work is carried out in compressed air. A liberal supply should be available, since in our case no fewer than six bottles of plasma were required to reverse the hypovolaemia.

We should like to acknowledge the help of the nursing staff of the Royal Portsmouth Hospital, and to thank the Medical Director-General, Royal Navy, for permission to publish information in this article.

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Unusual Case of Multiple Spontaneous Perforation of Small Bowel

Br. med. J., 1966, 2, 155-156

Spontaneous perforation of the small bowel is a rare occurrence. Though the diagnosis has on occasion been made before operation (Funderburk and White, 1962), a presumptive diagnosis of gastro-duodenal perforation or ruptured appendix is made in most cases. This is due to the lack of specific distinguishing features of this much rarer form of alimentary tract perforation, and to its protein etiology. The following is yet another case with its own unique and interesting features.

**Case History**

A man aged 50 developed sudden severe central and upper abdominal pain while walking. He vomited, and as severe pain persisted he was admitted to hospital six hours later.

He had a 12-year history suggestive of peptic ulceration of increasing severity, but a barium-meal examination repeated four weeks before admission had shown no ulceration. Apart from his dyspepsia he regarded himself as a fit man, well able to pursue his occupation as a bricklayer. There had been no history of drug therapy other than antacids, and at no time had there been diarrhoea.

Examination on admission revealed generalized abdominal tenderness and muscle-guarding. The abdomen was scaphoid and silent, and there was loss of liver dullness. A diagnosis of gastro-duodenal perforation was made and laparotomy performed.

**Operative Findings.**—No lesion was found in the stomach or duodenum, but there was a series of six lesions of the jejunum and ileum beginning about 3 ft. (91 cm.) from the duodeno-jejunal flexure. These lesions appeared as discrete haemorrhagic areas ½ in. (1.25 cm.) in diameter and situated 2 to 4 in. (5 to 10 cm.) apart. One of the lesions had perforated, causing a round punched-out perforation ½ in. (1.25 cm.) in diameter. The wall of the jejunum was thickened but otherwise appeared normal. A considerable amount of fibrinous exudate was present. There was no enlargement of the mesenteric lymph nodes. The spleen was of normal size, but showed purple mottling. At the site of the highest lesion the lumen of the jejunum was reduced by thickening of the wall; this short segment was resected. The perforation was closed and the other lesions were oversewn with seromuscular catgut sutures.

Histological examination revealed oedema, congestion, and marked eosinophilic infiltration of the submucosa, with sloughing of the mucosa (Fig. 1). The appearances suggested a non-specific enteritis.
Progress.—In the immediate post-operative period the patient progressed fairly well. Stool culture, blood cultures, and repeated Widal reactions were negative. The haemoglobin was 13.8 g./100 ml., P.C.V. 47%, M.C.H. 30%, W.B.C. 14,600/c.mm.; the film showed neutrophilia, with a normal eosinophil count. On the sixth post-operative day he again developed severe abdominal pain and signs of peritonitis. At a second laparotomy multiple perforations similar to the original one were discovered. However, in addition, much of the intestine was intensely congested and reddened in colour, suggesting infarction. A 6-in. (15-cm.) segment of small bowel, bearing several perforations, was resected and four other perforations were oversewn. After this operation he remained ill, his condition gradually deteriorated, and he died three days later.

Post-mortem Examination.—At necropsy a large segment of intensely congested and almost gangrenous ileum was found. The superior mesenteric artery was occluded completely by organized thrombus extending down the vessel for a distance of 10 cm. The aorta showed severe atheroma with areas of ulceration and calcification in its lower part. The spleen showed multiple small areas of infarction of several weeks' duration, and the left kidney had a small haemorrhagic infarct 1 cm. in diameter at its lower pole. Histology:—Examination of sections of jejunum and ileum revealed that in some areas the mucosa had ulcerated and was replaced by granulation tissue showing a non-specific inflammatory reaction as in Fig. 1. In other areas the mucosa was intact but the villi were broadened, stunted, and atrophic (Fig. 2). The inflammatory cellular infiltration was mainly of plasma cells and eosinophils. There was no evidence of focal arteritis. These features were compatible with a combination of an acute ischaemic episode superimposed on chronic intestinal ischaemia.

**COMMENT**

Apart from trauma, the commonest cause of small-bowel perforation is lymphosarcoma (Markowitz, 1960), but occasionally metastatic tumours such as bronchogenic carcinoma are responsible (Funderburk and White, 1962). Other causes include regional ileitis and specific infections such as typhoid fever and tuberculosis. In other cases anatomical abnormalities such as duplication of small bowel (Levack, 1962), multiple jejunal diverticula (Herrington, 1962), and especially Meckel's diverticulum may predispose to ulceration and perforation. Finkbiner and Decker (1963) have described perforations of the small bowel in association with certain collagen disorders such as lupus erythematosus, rheumatoid arthritis, and periarteritis nodosa. Focal ischaemia due to necrotizing arteritis is the cause in these cases.

Recently there have been reports of perforating ulcers of the small intestine due to thiazide derivatives or accompanying potassium supplements (Lindholmer et al., 1964; Baker et al., 1964).

Sometimes, in the absence of any other obvious pathological condition, a diagnosis of primary non-specific ulcer of jejunum or ileum is made. The cause of such cases is not established, but local ischaemic necrosis from segmental vascular occlusion has been suggested by Gaum and Devereux (1960) and by Teicher et al. (1963).

In the present case necropsy revealed evidence of extensive thrombosis of the superior mesenteric artery but without any evidence of arteriolitis or local segmental vascular occlusion.

It may be that the focal intestinal lesions observed in this patient were the result of multiple emboli of the end-arteries of the intestinal wall superimposed upon chronic ischaemia due to occlusion of the superior mesenteric artery; focal infarction being produced because of the initial pre-existing ischaemic changes in the gut. The infaracts of the spleen and left kidney suggest the possibility that similar embolization had occurred from the atheromatous aorta.

I wish to thank Mr. R. B. Wright for permission to publish this case; Mr. H. I. Tankel for constant prompting and advice; Mr. J. H. Levack, who performed the second operation; and Dr. A. Dick for the pathological reports.

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