circulation, so that the clinical picture is not the simple effect of fluid loss. The question of haemolytic power of vibrios has so long remained an academic luxury of the laboratory man. It will now justify more fully the attention it has received, and will excite an equal if not a greater interest in the clinician.

Summary and Conclusion

The occurrence of haemoglobinuria, haemoglobinaemia, and hyperbilirubinaemia is described in a case of cholera in which the patient died later of post-choleric uraemia. The strain isolated from the stool was a typical *Vibrio cholerae* (Ogawa type) with no evidence of haemolytic activity against sheep and human erythrocyte in either the 8-hours or the 72-hours culture. The possible roles of anhydraemia and acidosis, of alterations in the red blood corpuscles, and of sulphonamides in bringing about the intravascular haemolysis are discussed. It is suggested that endohaemolysin of *V. cholerae* is absorbed into the circulation and brings about the destruction of red blood cells.

Our thanks are due to Dr. A. K. Dutta Gupta, superintendent, for permission to publish the case, and to Dr. M. Ghose, Dr. K. Bhattacharyya, and Dr. H. Saha for their co-operation.

REFERENCES

Awny, A. (1948). *J. roy. Egypt. med. Ass.*, **31**, 351.
Bernard, P. N., Guillerm, J., and Gallut, J. (1939a). *C.R. Soc. Biol., Paris*, **130**, 23.
—— —— —— (1939b). Ibid., **130**, 228.
Chatterjee, H. N. (1947). *Trans. roy. Soc. trop. Med. Hyg.*, **40**, 905.
Cooray, G. H., and De, S. N. (1949). *J. Path. Bact.*, **61**, 541.
De, S. N., Sarkar, J. K., and Tribedi, B. P. (1950). *Proc. First Annual Conference Ind. Ass. Path.*, p. 64.
—— —— —— (1951). *J. Path. Bact.*, **63**, 707.
—— and Sengupta, K. P. (1951). *Lancet*, **2**, 1100.
—— —— Bhattacharyya, K., and Konar, N. R. (1951). *J. Indian med. Ass.*, **21**, 95.
Doorenbos, W. (1936a). *C.R. Soc. Biol., Paris*, **121**, 128.
—— (1936b). Ibid., **121**, 130.
Gilligan, D. R., Altschule, M. D., and Katersky, E. M. (1941). *J. clin. Invest.*, **20**, 177.
Goyle, A. N. (1939). *Indian J. med. Res.*, **26**, 611.
Harkins, H. N., and Hastings, A.B. (1931). *J. biol. Chem.*, **90**, 565.
Liu, S. H., Fan, C., and Wang, S. H. (1933). *Proc. Soc. exp. Biol., N.Y.*, **30**, 419.
Macgraith, Brian (1948). *Pathological Processes in Malaria and Black Water Fever*, p. 94. Oxford.
Manson-Bahr, P. H. (1950). *Manson's Tropical Diseases*, 13th ed. London.
Oliver-Gonzáles, J. (1944). *Proc. Soc. exp. Biol., N.Y.*, **57**, 25.
Rogers, L. (1921). *Bowel Diseases in the Tropics*, p. 92. London.
Smith, F., and Evans, R. W. (1943). *British Medical Journal*, **1**, 279.
Stone, J. D. (1947). *Austr. J. exp. Biol. med. Sci.*, **25**, 137.
Vassiliadis, P. C. (1937). Cited by G. S. Wilson and A. A. Miles in Topley and Wilson's *Principles of Bacteriology and Immunity*, 1948, 3rd ed., p. 518. London.
Wintrobe, M M. (1946). *Clinical Haematology*, 2nd ed., p. 476. Philadelphia.

MASSIVE HAEMORRHAGE FROM THE ACUTE GASTRIC ULCER

BY

V. J. DOWNIE, D.S.O., O.B.E., *M.C.*, F.R.C.S.Ed.
Chief Assistant in Surgery, Barnet General Hospital; Surgeon, Finchley Memorial Hospital

In a proportion of cases of gastro-duodenal haemorrhage submitted to laparotomy no ulcer can be detected after a thorough external examination of the stomach and duodenum. Three courses are open to the surgeon; he can close the abdomen, he can open the stomach, or he can perform a wide gastric resection. Until recently I belonged to the first school of thought, believing that an invisible and impalpable ulcer was an acute lesion which might be expected to heal under conservative treatment. I was fortified in this belief by two cases in which the patients' recovery after simple laparotomy had been uneventful.

The following six cases, five of them occurring in rapid succession within a seven-weeks period, have driven home the lessons that an acute ulcer can kill in spite of liberal transfusion, and that the lesion may be so small as to make its detection by a finger within the stomach extremely unlikely. These six patients had severe and repeated haemorrhages, but in all of them careful examination of the stomach and duodenum with the lesser sac widely open failed to reveal any outward evidence of active ulceration.

Case Reports

Case 1 (Simple Laparotomy).—Mrs. A., aged 75. History of indigestion for many years. Presenting symptom was haematemesis. Died six days after operation from repeated massive haematemeses, and in spite of the transfusion of 11 pints (6.3 litres) of blood. Necropsy revealed a shallow acute ulcer on the posterior wall of the body of the stomach.

Case 2 (Billroth I Gastrectomy).—Mrs. B., aged 76. History of indigestion for seven years. Presenting symptom was melaena, followed later by haematemesis. The resected specimen contained a shallow acute ulcer on the lesser curve of the stomach: the ulcer was irregular in shape, about the area of a sixpence, and there was a small vessel, plugged with recent clot, in its base. The scar of a healed ulcer was visible on the posterior wall of the stomach. Her post-operative condition was good for three days, after which it rapidly deteriorated, and she died from peritonitis on the fifth day. The necropsy had certain features of interest, and the findings are discussed below.

Case 3 (Polya–Hofmeister Gastrectomy).—Mr. C., aged 71. No history of previous indigestion. Presenting symptom was melaena, followed later by haematemesis. The resected specimen contained a shallow ulcer high on the lesser curve. No open vessel could be seen and the ulcer was little more than an erosion. This patient had repeated severe melaena, and received 13 pints (7.4 litres) of stored blood in four days. Whilst awaiting his anaesthetic in the theatre he had a massive haematemesis of about 2 pints (1.14 litres), followed by a convulsion and profound collapse. He was revived by artificial respiration and forced transfusion by the anaesthetist (Dr. J. Rochford), and the operation was begun as soon as the systolic pressure reached 90. It is clear that this patient was bleeding furiously from an acute ulcer, and that his only hope lay in urgent surgery. He made an excellent recovery, but his convalescence was somewhat delayed by threatened disruption of the abdominal wound. This complication was averted by conservative treatment with strapping.

Case 4 (Polya–Hofmeister Gastrectomy).—Mrs. D., aged 50. Two years' history of indigestion, the last attack starting three days before admission. Presenting symptom was melaena, followed later by haematemesis. The resected specimen contained a shallow acute ulcer with an open vessel in its base. This patient had two attacks very suggestive of pulmonary embolism on her sixth post-operative day. She was treated with intravenous heparin, and her subsequent recovery was uneventful.

Case 5 (Polya–Hofmeister Gastrectomy).—Miss E., aged 58. History of three months' indigestion, last attack starting 24 hours before admission. Presenting symptom was haematemesis. The specimen contained a shallow acute ulcer with an open vessel in its base. Convalescence was uneventful, except for an infected haematoma of the abdominal wound.

Case 6 (Polya–Hofmeister Gastrectomy).—Mr. F., aged 50. History of two years' indigestion, last attack starting two weeks before admission. Presenting symptom was melaena, followed later by haematemesis. The specimen contained part of an acute ulcer high on the lesser curve; the remainder of the ulcer had been crushed by a clamp and was included in the Hofmeister valve. Recovery was uneventful.

Four of these six patients were admitted under the medical care of Dr. G. Riddell Royston, and the other two (Cases 4 and 5) were admitted under Dr. Bruce Williamson. Cases

2 and 3 were transferred from Finchley Memorial Hospital, where the operating theatre was temporarily out of commission.

Discussion

The post-mortem examination in Case 2 showed that there had been no attempt at healing of the gastro-duodenal anastomosis, which was surrounded by a band of necrosis 1 cm. wide, involving both the duodenum and the stomach. The actual leak was not at the anastomosis, but in the necrotic suture line which had closed off the redundant lesser curve moiety of the gastric incision. The catgut sutures were easily identifiable and were intact.

These findings were regarded as of some importance, and were thought to be related to the patient's age and to the fact that she had received 9½ pints (5.4 litres) of stored blood. It is of interest that a similar case occurred recently in the same hospital. In this instance a man aged 61 underwent partial gastrectomy of the Polya type for gastro-duodenal haemorrhage. He had been desperately ill, and had received 12 pints (6.8 litres) of stored blood. General peritonitis supervened after the lapse of a few days, and a secondary operation revealed almost complete dehiscence of the anastomosis, which in this case had been fashioned with thread sutures. There was also some evidence of delayed healing, in the shape of threatened disruption of the wound, in Case 3.

This aspect of the matter was discussed at length with Dr. Royston, and it was agreed that elderly patients should be submitted to operation as soon as their condition permitted, and before they had received the equivalent of a replacement transfusion of stored blood. The part which vitamin C plays in wound healing is not understood fully, but it may be of significance that Case 3 had received 7 g. of vitamin C (added to the pre-operative blood transfusion in the proportion of 1 g. to the pint), whereas Case 2 had received only 2 g.

The knowledge that fatal haemorrhage can occur from a stomach which is outwardly unblemished does not absolve one from the duty of carrying out a careful laparotomy whenever the possibility of a lower lesion exists—for example, where there has been melaena but no haematemesis. The following case, operated on during the seven-weeks period mentioned above, is a bizarre reminder of the need for caution before proceeding to gastrectomy.

Mrs. X, aged 64, was admitted under the care of Dr. Royston with clinical features suggesting either mild cholecystitis or appendicitis. Whilst under observation she had a massive melaena. At laparotomy the stomach and duodenum appeared normal, but a small mass could be felt in the ascending colon. The colon was full of blood, but there was none proximal to the ileo-caecal valve. The mass showed signs of recent inflammation, and was thought to be a solitary diverticulum, but, as carcinoma could not be excluded, right hemicolectomy was performed. Recovery was uneventful except for superficial thrombophlebitis of the left leg. Subsequent examination of the specimen showed that an inflamed solitary diverticulum consisting of mucous membrane and its muscularis had penetrated the main muscle coat, and had eroded a large artery lying immediately external to its deepest point. A post-operative barium enema showed no evidence of diverticulosis in the remaining colon. I cannot find any record of a similar case in the literature.

Summary

Six cases of massive haemorrhage from acute gastric ulceration are described, together with a contrasting and unusual case of diverticulitis of the ascending colon.

It is suggested, not without a qualm of misgiving at the extent of the operation as compared with that of the lesion, that wide gastric resection should be the operation of election, and that the interference with wound healing which may follow massive transfusion of stored blood is an additional argument in favour of early operation on elderly patients.

I am grateful to Dr. Royston and Dr. Bruce Williamson for entrusting me with the surgical care of these patients, and to Dr. Ronald Welch for his reports on the necropsies on Cases 1 and 2, and his report on the histopathology of the colonic diverticulum.

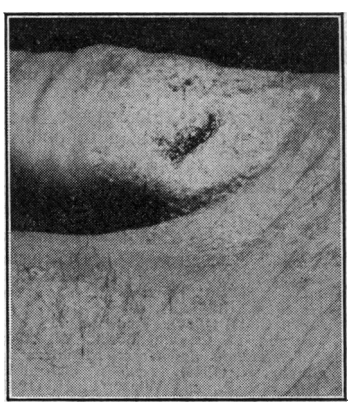
Medical Memoranda

Norwegian Scabies

During the past 50 years there have been only a few reports of the occurrence of Norwegian, or crusted, scabies in English-speaking countries: in England Calnan (1950) described four cases seen in a mental defectives' institute, Beatty (1913, 1915) saw two cases in Dublin, and two more have been reported in the American literature (Anderson and Stout, 1940; Sweitzer and Winer, 1941). It was first described by Danielssen and Boeck (1848) in leprous patients, and has since been a subject much discussed by European dermatologists. I recently saw the following case at the Royal Infirmary, Sheffield.

CASE REPORT

A railway plate-layer aged 55, of average intelligence and maintaining, from his appearance, a moderately high standard of personal hygiene, complained of an eruption over the whole body of one month's duration. Itching was only slight in degree, but was worse at night. He gave no previous history of any skin disease. He was living with a family of daughters and grandchildren, and always slept by himself. The family have a cat, but he had otherwise no close contact with animals.



Lesion on finger.

On examination he showed bluish friable hyperkeratotic plaques on the fingers (see illustration), wrists, elbows, over the thyroid cartilage, tips of the ears, buttocks, thighs, and soles of the feet. The axillary folds were studded with large papules. Burrows were noticed on the penis and, on closer examination, also on the fingers and wrists. A live acarid was removed from one of the burrows and the diagnosis of scabies confirmed. The hyperkeratotic lesions were scraped with a curette and the scales examined microscopically; these revealed large numbers of acarids of all sizes in a state of great activity and resembling in every way Mellanby's pictures (1943). Physical examination, including detailed examination of the nervous system, showed no other abnormality.

The routine treatment by baths and benzyl benzoate emulsion for two days was prescribed, and 11 days later the patient's skin was clear except for some residual hyperkeratosis of the soles, which responded in a few days to a 5% salicylic acid ointment. A month later the skin was completely normal. At that time, some three months after the onset, there were still no reports of further cases in the family.

COMMENT

The name crusted scabies is preferred by many to Norwegian scabies, but it may cause confusion by suggesting an impetiginous form of eruption. Histological reports in