should perhaps have led to the diagnosis of visceral scleroderma. The patient's age, however, was unusual, most of the reported cases occurring in patients aged 40 to 60.

I thank Dr. Geoffrey Steed for permission to publish this report, and Dr. J. D. N. Nabarro for his help and encouragement in writing it. The barium-meal examination was carried out by Dr. C. G. Whiteside, radiology department, Middlesex Hospital, and the histological sections were prepared by Dr. John Arthur, Bland-Sutton Institute of Pathology, Middlesex Hospital.

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Acute Left Ventricular Failure after Successful Surgical Treatment of Bleeding Gastric Ulcer

This paper describes the case of a patient with oligæmic shock due to hæmorrhage who, immediately after arrest of the hæmorrhage, developed left heart failure with pulmonary oedema. The main purpose is to demonstrate that such a new development can take place with dramatic suddenness and that the treatment should be altered diametrically to correspond.

CASE REPORT

A married woman aged 76 was admitted to hospital on February 8, 1961, with a history of having vomited a quantity of bright red blood after experiencing a sudden sharp pain in the upper part of her abdomen. She vomited blood repeatedly and started passing fresh blood per rectum. Her past history revealed that she had suffered "a heart attack" around Christmas, 1960, but otherwise had always been fit and active.

On examination the patient was a thin, pale woman, mentally very active. The pulse was 96 a minute, regular, and of good volume; blood-pressure 160/50. There was evidence of mitral stenosis and aortic regurgitation: not in failure clinically. An E.C.G. showed left ventricular hypertrophy with strain. There was some tenderness in the epigastrium. Dark red blood was passed per rectum. On admission the hæmoglobin was 55%.

On account of the history and the evidence of free aortic regurgitation, conservative treatment was decided upon. During the next 18 hours 12 pints (6.8 litres) of blood were given, but repeated hæmatemeses and melaena were of such extent that she barely held her ground. At this stage it was decided to deal with the problem surgically.

She was taken to the theatre on her bed, with transfusions set up in both arms. Digoxin 0.25 mg. was given before she went to the theatre. On arrival in the anaesthetic room she vomited bright red blood and passed blood and clots per rectum. Rapid induction was performed on the table, and almost immediately she suffered a severe collapse, the carotid pulses being impalpable. The abdomen was quickly

opened through an upper midline incision. The stomach was found to be full of blood clot. A high partial gastrectomy was performed in just under 30 minutes, and at the end of operation her condition appeared to be satisfactory in the circumstances. During the operation a total of 1 pint (570 ml.) of blood was transfused. Examination of the specimen revealed an eroded artery in a subacute gastric ulcer. A disturbing factor noted at the conclusion of the operation was the degree of jugular venous distension. A few minutes later, just before her return to the ward, cyanosis developed. Frothy, pinkish fluid appeared at the corners of her mouth and the chest became full of moist sounds. B.P. at the time was 190/60. Oral suction was performed and then a bronchoscope was passed and suction of bronchi continued by this route for 20 minutes—fluid forming as quickly as it was removed.

In view of this acute left and right ventricular failure, aminophylline 0.25 g. was given intravenously and both transfusions were taken down. Esmarch bandages were applied on the suggestion of Dr. K. D. Keele to all four limbs to impede the venous return, thus performing a physiological venesection. At the end of 20 minutes the bronchoscope was removed and suction continued via the oropharynx. After 45 minutes from the onset of symptoms there was a noticeable improvement in the patient's condition. This continued steadily over the next 30 minutes, during which time the necessity for suction became less and the jugular venous engorgement disappeared. The total amount of bronchial aspirations was 2½ pints (1,420 ml.) of frothy blood-stained fluid. The patient was then returned to the ward and placed in an oxygen tent. The limb bandages were released and reapplied at intervals of 30 minutes over the next four hours, until they were finally taken off one at a time, the effect on jugular venous pressure being noted.

The patient made a satisfactory recovery. It is interesting to note that on the first post-operative day her hæmoglobin was 87% and hæmatocrit reading 47; transfusion, therefore, had not been excessive. Her subsequent post-operative progress has been uneventful.

COMMENT

This case raises a number of interesting points: (1) It illustrates a remarkable and dramatic situation where successful surgical treatment of severe hæmatemesis in a patient with a gross cardiac lesion precipitated an equally serious medical emergency. (2) It demonstrates the swift change from exsanguination to left ventricular failure requiring opposite modes of treatment. (3) It also shows the success of combining bronchial aspiration and medical venesection in the emergency treatment of acute left ventricular failure. (4) With increasing boldness of surgical approach in the treatment of hæmatemesis in the elderly, particularly those with cardiac lesions, this type of case may not be uncommon.

We thank Dr. K. D. Keele, under whose care this patient was originally admitted, and Mr. O. D. Morris, who performed the operation, for permission to publish this case.

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A scholarship in memory of the late Mr. S. I. Levy has been established by his widow at the Medical School of the Hebrew University of Jerusalem. Mr. Levy was for many years surgeon to the East Ham Hospital and to the Westminster Hospital, and for about 30 years senior surgeon to the London Jewish Hospital. The scholarship will be granted annually to a graduate of the Hebrew University specializing in surgery.