six remissions, four occurred within the first year. Three patients belonged to the group who were adequately treated.

Though the treated patients showed a lower incidence of hypertension at the start of the trial, this immunity did not last; of the 20 patients whose blood-pressures were initially normal, nine developed hypertension during the period of the trial. The introduction of effective hypotensive agents, which were not available during the period covered by the untreated series, may well have prolonged the survival of some of the cortisone-treated patients.

There was evidence of renal involvement at the time of initial muscle biopsy in 13 treated patients; seven of these were alive after three years, including one in whom an initial blood urea of 100 mg./100 ml. had fallen to 26 mg./100 ml. Complete remission of active disease has occurred in two cases with initial renal involvement; in one there was no proteinuria and a normal blood-pressure four years later; the other had hypertension. Recovery from renal polyarteritis nodosa in the absence of treatment is extremely rare.

Of the 11 deaths, nine were from active arteritis and two from the late effects. In two patients there was fatal uraemia; another died from perforation of a polyarteritic ulcer of the jejunum; two died of haemorrhage due to polyarteritis of the bowel, associated in one with subarachnoid bleeding; one had acute cerebral polyarteritis; one, suffering from peripheral neuropathy, died of pneumonia; one died of heart failure, probably attributable to coronary polyarteritis; and one died from cachexia. Of the later deaths, one was due to chronic pyelonephritis and the other to malignant hypertension; both had previously had renal polyarteritis nodosa.

Discussion

The limitations of this trial, which were fully discussed in our first report, arose through the impossibility of having concurrent randomly selected controls. At 12 months the results in the treated group are distinctly superior, though this difference between the two groups is reduced if patients entering the trial with hypertension are excluded. At three years the advantage enjoyed by the treated group has diminished and disappears entirely if patients with initial hypertension are excluded. By four years, though the numbers available for comparison had fallen, the percentage survival in the two groups is the same, but exclusion of hypertensive patients reveals a bias in favour of the untreated group, amongst whom no further deaths had taken place after the first year of the disease.

In view of the differences between treated and untreated groups it is probably safer to avoid further comparison. The start of treatment certainly brought speedy and often dramatic improvement to most patients. On the other hand, it later brought distressingly unpleasant complications to a few. The trial has not produced evidence that cortisone, in the dosage given to these patients, improved the chances of ultimate recovery. Possibly it postponed death.

Summary

Twenty-one cases of histologically proved polyarteritis nodosa have been treated with cortisone, and their course has been followed in each instance for at least three years. At the end of this period eight were dead, five had remitted, and eight still needed steroid therapy.

The Panel would like to express its gratitude to its secretary, Dr. J. J. R. Duthie, for keeping this and other trials in progress; to Dr. Geoffrey Rose for preparing this and the previous report; to Dr. Graham Wakefield for his report to the Panel; and to the physicians who gave of their time and interest to keep the records which made the trial possible.

REFERENCE

Medical Research Council (1957). *Brit. med. J.*, 1, 608.

MYOCARDIAL INFARCTION AFTER ACUTE GASTRO-INTESTINAL HAEMORRHAGE REPORT OF 3 CASES

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The occurrence of electrocardiographic changes suggesting myocardial ischaemia after acute haemorrhage is well recognized, both in patients (Aschenbrenner, 1934; Flaum and von Jagić, 1935; Marchal *et al.*, 1935; Bloch, 1938; Szekely, 1940; Scherf *et al.*, 1941; Rasmussen and Foss, 1942; Scherf and Klotz, 1944; Ørning *et al.*, 1946; Master *et al.*, 1947, 1950; Dack *et al.*, 1951) and in experimental animals (Büchner, 1932; Radnai, 1935; Marchal *et al.*, 1938; Izquieta and Paster-nack, 1946). The incidence of such changes depends upon the frequency with which they are sought, and this probably accounts for most of the variation in different published series—for example, 26 of 74 cases (35%) (Ørning *et al.*, 1946), 33 of 58 cases (57%) (Rasmussen and Foss, 1942), 59 of 103 cases (57%) (Master *et al.*, 1950), 11 of 16 cases (69%) (Scherf *et al.*, 1941), 24 of 28 cases (85%) (Dack *et al.*, 1951), and 14 of 15 cases (93%) (Scherf and Klotz, 1944). Myocardial infarction after acute haemorrhage is much less common, but its true incidence is difficult to assess, as the diagnosis is usually made at necropsy. Non-fatal cases may be missed, since routine electrocardiography is rarely employed in patients with haemorrhage, but, even so, the electrocardiographic changes may be transient (Boyd and Scherf, 1940), and hence be misinterpreted as due to myocardial ischaemia only. In one series of 103 patients with acute gastro-intestinal haemorrhage there were 22 fatal cases, of which 13 came to necropsy and four showed myocardial necrosis (Master *et al.*, 1950). Haemorrhage was found to be the precipitating factor in 7 out of 48 myocardial infarcts without coronary occlusion (Master *et al.*, 1941) and in 4 out of 25 cases of subendocardial necrosis examined after death (Horn *et al.*, 1950). On the other hand, in a necropsy study of 300 cases of myocardial infarction Bean (1937) found only two associated with acute loss of blood. Isolated case reports have also appeared (McLaughlin *et al.*, 1940; Master and Jaffe, 1940; Blumgart *et al.*, 1941, Case 2; Kinney and Mallory, 1945), and the condition has been demonstrated in experimental animals (Büchner, 1932; Büchner and von Lucadou, 1934; Melcher and Walcott,

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1951). The importance of preventing this serious complication of acute haemorrhage prompts us to report the following three fatal cases.

Case 1

A man aged 66 was admitted to hospital as an emergency case on February 1, 1958. He had vomited about 1 pint (568 ml.) of altered blood on each of the three preceding days, but had first noticed dark stools on January 24 and had felt weak subsequently. Intermittent dyspepsia had been present for 10 years. No chest pain or discomfort had been noticed.

On examination his temperature was 96.8° F. (36° C.). He was an extremely pale collapsed man in peripheral circulatory failure. Pulse was impalpable at the wrists; heart rate 90 a minute, regular; B.P. unrecordable. Heart: not clinically enlarged; sounds faint but normal. Respiratory system: respirations 24 a minute; chest—diffuse rales and sibilant rhonchi. Abdomen: slight epigastric tenderness only. C.N.S. normal.

Investigations.—Blood count: Hb, 11.5 g./100 ml.; P.C.V., 32%; W.B.C. 30,000/c.mm. (polymorphs 80%, lymphocytes 16%, monocytes 3%, basophils 1%). E.C.G.: Q in Lead I, QS with absent R in V₄, S-T elevation in Leads I, II, V₂, and V₄. Interpreted as recent anterior myocardial infarction.

Treatment.—Oxygen by inhalation. Intravenous infusion of L-noradrenaline in dextrose N/5 saline (8 ml./litre) begun immediately. A separate intravenous transfusion of whole blood begun 45 minutes later. B.P. rose to 80/40 mm. Hg and was maintained, but the patient died four hours after admission, having received 1 pint (568 ml.) of blood during that time.

Post-mortem Examination

The relevant post-mortem findings were as follows. Stomach: A chronic gastric ulcer 3 cm. in diameter was present on the lesser curvature 5 cm. from the gastro-oesophageal junction, and in the base four vessels plugged with thrombi were seen. Heart: A large recent infarct with yellow discoloration in the anterior wall of the left ventricle was present. The coronary arteries were extremely atheromatous, with calcification and narrowing of the lumina, but no thrombus was found.

Histology.—The gastric ulcer with thrombosed vessels in its base was confirmed. The heart showed muscle necrosis with marked cellular infiltration consisting mainly of lymphocytes with a few polymorphs, macrophages filled with pigment, fibroblasts, and fairly numerous young capillaries. There was also some extracellular basophilic material present between the muscle fibres. Estimated age of infarct, 4 to 10 days.

Case 2

A man aged 75. Emergency admission on November 14, 1957. He had had intermittent dyspepsia, relieved by food and alkalis, for 10 years, and exertional chest pain for three years, relieved by glyceryl trinitrate. Two weeks before admission he had constant epigastric pain unrelieved by food. One week before admission (November 7) he began to pass black tarry stools, and continued to do so but did not vomit. On November 11 he felt faint and had a profuse melaena, after which he developed retrosternal pain lasting three hours.

On examination he was a pale obese man, with a temperature of 100.4° F. (38° C.), pulse 120 and regular, B.P. 150/75 mm. Heart: not clinically enlarged; sounds normal; no cardiac failure. Respiratory system was normal. Abdomen: obese; no localized tenderness. Per rectum: confirmed melaena stool; no mass. C.N.S. normal.

Investigations.—Blood count: Hb, 10 g./100 ml.; P.C.V., 28%; W.B.C. 12,000/c.mm. (polymorphs 78%, lymphocytes 20%, monocytes 2%). E.C.G.: Q in Leads I and V₄. Inverted T in Leads I, II, V₂, V₄, and V₇. R absent from V₄. No S-T elevation. Interpreted as anterior myocardial infarction of uncertain age.

Treatment.—Copious oral fluids. Intravenous blood transfusion of 4 pints (2.3 litres) during a period of 12 hours. Pulse rate was then 90 a minute and B.P. 160/90 mm. Hg. Blood count: Hb, 11.9 g./100 ml.; P.C.V., 36%. There was no further melaena, but 24 hours after admission acute pulmonary oedema developed suddenly and the patient died within 30 minutes despite the administration of morphine, intravenous aminophylline, and oxygen.

Post-mortem Examination

The relevant post-mortem findings were as follows. Stomach and duodenum: A chronic ulcer 1 cm. in diameter was present in the duodenum. Heart: A small amount of turbid fluid was present in the pericardial sac, with pericarditis. The anterior lateral wall of the left ventricle showed extensive fibrosis with a more recent infarct, haemorrhagic and necrotic, in the anterior wall at the apex. The coronary arteries were opened up in their entirety, showing moderate atheroma with calcification but no thrombus.

Histology.—The chronic duodenal ulcer was confirmed. Heart: The old infarct showed well-formed fibrous tissue, while in the more recent one the muscle fibres were hyaline and eosinophilic, with extensive infiltration by inflammatory cells, mainly polymorphs, but a few lymphocytes and a few fibroblasts could be seen. Estimated age of infarct, four to eight days.

Case 3

A married woman aged 74 years. Emergency admission, April 9, 1955. From relatives it was learnt that she had vomited 1 pint (568 ml.) of fresh blood 12 hours before admission, and a coffee-ground vomit two hours before admission, after which she complained of persistent precordial pain. Exertional chest pain for one year, and occasional epigastric pain, relieved by vomiting, for two years.

On examination she was pale, slightly dyspnoeic, confused and disorientated, and unable to give any account of her symptoms. Her temperature was 99.5° F. (37.5° C.), pulse 110 and regular, and blood-pressure 200/90 mm. There was slight distension of neck veins. Heart: moderate left ventricular enlargement; sounds normal, respiratory system: respiration 22; basal rales in chest. Abdomen: no localized tenderness. C.N.S.: no abnormality elicited.

Investigations.—Blood count: Hb, 8.9 g./100 ml.; R.B.C., 3,000,000/c.mm.; P.C.V., 30%; W.B.C., 17,700/c.mm. (polymorphs 76%, lymphocytes 21%, monocytes 3%); M.C.V., 82 cubic microns; M.C.H.C., 30%. Urine: S.G. 1018; trace albumin only. Blood urea: 60 mg./100 ml.

Treatment.—Oral fluids. Transfusion of 4 pints (2.3 litres) of blood during the first 12 hours; Hb rose to 12.1 g./100 ml., P.C.V. 40%, but confusion persisted. On April 12 6 oz. (170 ml.) of fresh blood was vomited. Pulse was 120 and blood-pressure 180/90 mm. Hg. Blood count: Hb, 8.2 g./100 ml.; P.C.V., 26%; R.B.C., 3,250,000/c.mm.; M.C.V., 81 cubic microns; M.C.H.C., 31%; W.B.C., 14,900/c.mm. A further 3 pints (1.7 litres) of blood was transfused over a period of 12 hours, but signs of congestive heart failure increased. Blood count: Hb, 11.3 g./100 ml.; P.C.V., 36% after transfusion. On April 14 there was further melaena and the patient became comatose, dying within three hours despite a further transfusion of 1½ pints (852 ml.) of blood. The latest blood count showed Hb 9 g./100 ml.; P.C.V., 28%.

The E.C.G. 24 hours before death showed sinus rhythm, left ventricular preponderance, T wave inversion in Leads I, II, and V₇, flattened T waves in Leads III and V₄, and S-T depression in Leads II, V₂, and V₇. This was interpreted as evidence of left ventricular hypertrophy and ischaemia.

Post-mortem Examination

Relevant post-mortem findings were as follows. Stomach: Midway along the lesser curvature were two chronic gastric ulcers each 1 cm. in diameter with an eroded vessel in the base of one of them. The entire gastro-intestinal tract was filled with blood. Heart: There was a small patch of fibrosis

on the left border of the left ventricle towards the base, and also a large area of recent infarction with yellow discoloration involving the greater portion of the base of the anterior and left lateral borders of the heart. The coronary arteries were opened up along their entire length and showed numerous patches of calcifying atheroma, but no thrombus was found. Brain: A recent infarct was present in the right occipital lobe involving the tip and an area of 4 sq. cm. of the adjacent cortex. Cerebral vessels grossly atheromatous, but no thrombus present.

Histology.—Stomach: The two chronic gastric ulcers were confirmed. Heart: The fibrotic patch was confirmed. In the more recent infarct the muscle fibres had a ground-glass hyaline appearance, with swollen irregular nuclei, and were separated by numerous polymorphs and extracellular basophilic material. Estimated age of infarct, three to four days.

Comment

Pathogenesis

Post-haemorrhagic electrocardiographic changes are thought to indicate a relative inadequacy of the coronary blood flow ("acute coronary insufficiency"), which, if severe or protracted, may lead to myocardial necrosis (Master *et al.*, 1947, 1950). A 35% reduction in coronary flow has been shown experimentally to be the critical level for the appearance of electrocardiographic changes (Wégria *et al.*, 1949). Coronary artery spasm has been suggested as the responsible mechanism (Scherf *et al.*, 1941; Rasmussen and Foss, 1942), but the coronary vascular resistance actually falls after a severe haemorrhage (Opdyke and Foreman, 1947; Vowles *et al.*, 1957). Szekely (1940), who found no correlation between the haemoglobin level and the occurrence of electrocardiographic changes in non-haemorrhagic anaemia, suggested that reduction in the circulating blood volume is responsible for the inadequate coronary flow after haemorrhage, and this has been demonstrated in experimental animals (Opdyke and Foreman, 1947; Corday *et al.*, 1949; Vowles *et al.*, 1957).

The coronary flow returns to normal when the circulating blood volume is restored (Corday *et al.*, 1949; Vowles *et al.*, 1957) and experimental post-haemorrhagic cardiac failure has been reversed by mechanically augmenting the left coronary flow without increasing the circulating blood volume or raising the arterial pressure (Sarnoff *et al.*, 1954b). Prolonged post-haemorrhagic coronary insufficiency may produce irreversible myocardial damage, so that restoration of the circulating blood volume fails to reverse cardiac failure (Wiggers and Werle, 1942; Wiggers, 1945, 1947) even though the coronary flow may reach four times the pre-haemorrhage level (Opdyke and Foreman, 1947). Myocardial infarction has been described in this state (Melcher and Walcott, 1951), though it is not invariably present. Since myocardial infarction *per se* may produce a fall in circulating blood volume of 16–20% (Agress *et al.*, 1950, 1951) a vicious-circle mechanism may be initiated.

The available evidence therefore suggests that post-haemorrhagic cardiac disturbances are due to an inadequate coronary blood flow secondary to the reduced circulating blood volume, and that myocardial necrosis may occur if the changes are unduly great or prolonged. The cerebral infarct of Case 3 was probably the result of a similar disturbance of the cerebral circulation (Vowles and Howard, 1958).

Pathology

Master *et al.* (1950) emphasized the absence of coronary thrombosis and the subendocardial localization

of the infarction in their cases. Post-haemorrhagic coronary thrombosis does occur, however (Blumgart *et al.*, 1941; Kinney and Mallory, 1945), and the myocardial lesion may be indistinguishable from the usual infarct (Gross and Sternberg, 1939). Our cases showed no coronary thrombosis, but the myocardial infarcts were identical with those which follow coronary occlusion. Coronary thrombosis was, however, absent at necropsy in 57% of 2,351 published cases of myocardial infarction (Evans, 1954). Master *et al.* (1950) found no correlation between post-haemorrhagic coronary insufficiency and age or sex, but it was related to hypotension, tachycardia, and a fall in haemoglobin of more than 30%. It was twice as common when there was pre-existing cardiac disease, though myocardial infarction was found in a 19-year-old girl with normal coronary arteries. Dack *et al.* (1951) found the condition more often in the elderly, and half their patients had pre-existing cardiac disease. Two of our patients had pre-existing effort angina, and all three were found to have marked coronary atheroma. A given fall in arterial blood-pressure produces a much greater reduction in blood flow through narrow rigid arteries than those of normal calibre and elasticity (Vowles and Howard, 1958), and the exaggerated effects of loss of blood on areas of myocardium supplied by narrowed vessels has been demonstrated in experimental animals (Corday *et al.*, 1949).

Clinical Features

Acute coronary insufficiency alone may produce retrosternal pain, congestive heart failure, and acute pulmonary oedema (Master *et al.*, 1947, 1950), but fever, leucocytosis, raised E.S.R., and a raised serum transaminase suggest the occurrence of myocardial infarction. Prolonged retrosternal pain occurred in two of our patients, but Case 1 gave no history of pain. Cases 1 and 2 developed pulmonary oedema, and Case 3 congestive heart failure. Cases 2 and 3 were pyrexial, but Case 1 had a subnormal temperature. All three patients showed a polymorph leucocytosis.

Electrocardiographic Features

The usual post-haemorrhagic electrocardiographic changes are S-T depression with flattening or inversion of T waves in one or more leads. Master *et al.* (1950) emphasized the rarity of Q waves and S-T elevation, though deep Q waves were recorded in three of their fatal cases. Only one of these came to necropsy, when widespread subendocardial necrosis was found. The electrocardiographic changes of acute coronary insufficiency are usually transient and maximal in the initial record; persistent changes suggest myocardial infarction of varying degrees of severity (Master *et al.*, 1947). The E.C.G.s of two of our patients showed Q waves, one with S-T elevation; but, in the third, Q waves were absent and the changes were interpreted as due to left ventricular hypertrophy and ischaemia without definite infarction.

Treatment

Master *et al.* (1947) urge early and adequate intravenous blood transfusion to eliminate coronary insufficiency and prevent myocardial infarction, even though cardiac failure may be present. They still recommend blood transfusion when myocardial infarction has occurred after acute haemorrhage, since they believe that early restoration of coronary flow will restrict the area of myocardial necrosis to a minimum. Delay in transfusion may lead to irreversible myocardial failure (Wiggers, 1945, 1947).

Intra-arterial transfusion has been shown experimentally to be no more effective than intravenous transfusion in correcting insufficient coronary flow after severe haemorrhage (Case *et al.*, 1953).

Intravenous L-noradrenaline has been used to combat hypotension both in severe haemorrhage (Goldenberg *et al.*, 1949) and in spontaneous cardiac infarction (Miller and Moser, 1959). As it increases coronary blood flow (Burn and Hutcheon, 1949) its use in the treatment of haemorrhage not responding quickly to blood transfusion would help to protect the myocardium (Vowles *et al.*, 1957). The latest pressor drug metaraminol bitartrate ("aramine"; laevo-1-(hydroxyphenyl)-2-amino-1-propanol hydrogen d-tartrate) has similar clinical effects (Stechel *et al.*, 1956; Besterman, 1959), and its intravenous administration has been shown to reverse the effects of induced coronary insufficiency in dogs (Sarnoff *et al.*, 1954a).

Summary

The clinical features and necropsy findings of three cases of myocardial infarction occurring after acute gastro-intestinal haemorrhage are presented. The pathogenesis of the condition is discussed, and the importance of early restoration of the blood volume after haemorrhage, especially in the elderly, is emphasized as a means of preventing this dangerous complication.

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CLINICAL ASSESSMENT OF INTESTINAL FAT-ABSORPTION USING RADIOACTIVE FAT

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When intestinal malabsorption is suspected in clinical practice a chemical fat balance is a standard method of investigation. Such a balance study requires admission to hospital, is time-consuming, and involves the unpopular and often inadequate collection and mixing of faeces. There is a need for a simple and rapid alternative to collection of faecal fat in the study of fat-absorption.

Thannhauser and Stanley (1949) pioneered the combined study of blood levels and excretion of radioactivity after an oral dose of natural unsaturated fat labelled with radio-iodine. In most of the subsequent reports on the use of this method to measure fat-absorption, attention has been paid almost entirely to the blood concentration and the faecal excretion of the isotope (Stanley and Thannhauser, 1949; Baylin *et al.*, 1955; McKenna *et al.*, 1957; Shingleton *et al.*, 1957a, 1957b; Grossman and Jordan, 1958; Lubran and Pearson, 1958; Ruffin *et al.*, 1958; Spector *et al.*, 1958; Berkowitz *et al.*, 1959). Other workers have described an analogous method in which the iodine present in the urine after an oral dose of lipiodol is estimated chemically (Groen, 1948; Silverman and Shirkey, 1955). In this communication we record our experience of the assessment of fat-absorption by giving radioactive

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