known that in Addison's disease there is a greatly increased glucose tolerance due to unbalanced action of insulin, and that this state of hypoglycaemia is not corrected by injections of desoxycortone acetate.

It is stated by some authorities that "anaemia may occur in Addison's disease but is not specific" (Whitby and Britton, 1942). According to Goldzieher (1939), "anaemia, originally mentioned as a feature by Addison, is actually not a symptom of the disease. The red cell count is not infrequently increased, and the haemoglobin higher, as the result of the inspissation of the blood." Other sources (Leyton and Simpson, 1936-41) tentatively suggest that the adrenal cortical hormones might be needed for adequate haemopoiesis, but still favour failure of iron absorption, possibly due to achlorhydria, as the more likely cause.

Brown and Roth (1925) found definite anaemia in one of 17 cases of Addison's disease they observed. It showed haemoglobin 50% and 53% (Dare scale) and R.B.C. 2,960,000 and 3,280,000. The patient died three days after admission. They quote a case observed by Neumann with a red cell count of 1,120,000. The lowest figures given by Goldzieher (1939) are Hb 67%, R.B.C. 3,450,000; and by Rowntree and Snell (1931) Hb 30% and 39% (Dare scale), R.B.C. 1.5 and 1.9 millions.

The rapid response to desoxycortone acetate in the present case suggests that this substance is here essential for the normal formation of red cells. That intestinal insufficiency, as indicated by the high fat content of the stools, was not the cause of the anaemia is shown by the persistent colour index of 1.0 to 1.1 (excepting one reading of 1.3); this argues against the absence of the pernicious anaemia haemopoietic factor being the primary cause, as also does the absence of therapeutic response to liver. The normal colour index suggests that the absence of iron is not the primary cause. As there was no achlorhydria, this too could not have participated in the causation of the anaemia. Indirect evidence of the specific action of the cortical hormones in haemopoiesis is that polycythaemia occurs in suprarenal cortical excess, due to cortical hyperplasia, adenoma, or carcinoma.

It is probable, therefore, that in this case of Addison's disease the anaemia is due to the direct specific effect on the bone marrow of an absence of suprarenal cortical hormone. This may often occur in Addison's disease, but, being slight, may be hidden. In addition, diminished bowel absorption may add the features of iron- or "liver"-deficiency anaemia. To these factors must be added the effect of dehydration (probably absent in this case), acidosis, and ionic change on the individual and total red cell volume. A variable blood picture may thus result. A normocytic anaemia of this degree of severity will, in view of the failure of response to all the usual haematinics, resemble "idiopathic aplastic anaemia."

The presence of "one primitive megaloblast" in the earliest blood count does not make the diagnosis a macrocytic anaemia, as none have been seen since then. The 8% eosinophils on first admission to this hospital may have been due to the injections of liver before admission, though eosinophilia has been reported in Addison's disease by Zondek (1944).

Melanuria is rare in Addison's disease, and it is not more common or more pronounced in cases of the disease with deeper pigmentation. It is said to occur in severe cases (Karsner, 1938). This suggests that cell permeability is not dependent upon the same cortical hormone the absence of which results in pigmentation. A severe alteration in cellular permeability may therefore occur with slight or no pigmentation, and vice versa. Non-pigmented cases of Addison's disease have been described by Conybeare and Millis (1924) and White and James (1927). A severe alteration in cellular permeability might result in the liberation of melanin, and its immediate precursor melanogen, into the circulation; the melanin is then reduced to melanogen in the serum (Karsner, 1938), and excreted in the urine, where it is reoxidized to melanin.

In the present instance there was no direct relation between exacerbation of the syndrome and of the stomatitis, which, however, improved when the clinical condition responded to desoxycortone acetate.

Methods Used

Faecal Fats.—The Soxhlet extraction method, substantially the same as that of Holt, Courtney, and Fales, and referred to in

Harrison's *Chemical Methods in Clinical Medicine*, 2nd edition, pp. 469 and 470.

Blood Chlorides.—Whitehorn's method (*J. biol. Chem.*, 1920-1, 45, 449).

Serum Sodium.—Modification of method of McCance and Shipp described in Harrison's *Chemical Methods in Clinical Medicine*, pp. 379 and 380.

Serum Potassium.—Method described by Jacobs and Hoffman (*J. biol. Chem.*, 1931, 93, 685).

The serum sodium and serum potassium methods are colorimetric methods, and a "Spekker" absorptiometer has been used in these estimations.

Summary

A case simulating Addison's disease with severe normocytic normochromic anaemia, steatorrhoea, and melanuria, and its treatment, are described.

A tentative explanation of the underlying mechanism of the syndrome anaemia, steatorrhoea, and melanuria is put forward.

The differential diagnosis is discussed.

The relevant literature is reviewed.

I wish to thank Dr. Osman for his kind help and advice in treating this case and writing this paper; Dr. Jones, of the County Laboratory, Maidstone, and Dr. Taylor and Mr. Rogers, of the Pathological Department, County Hospital, Pembury, for the haematological and biochemical investigations. Also Dr. Grasby, medical superintendent, and Dr. Ponder, County Medical Officer, for permission to publish this report; the military authorities for sending data obtained in a military hospital; and the medical superintendent of the West Glamorgan County Hospital, Neath.

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THREE CASES OF CLOSED INTRA-ABDOMINAL INJURY

BY

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Cases of visceral injury resulting from blunt injuries to the abdomen are interesting clinically. The exact mechanisms by which the lesions are produced have occasioned some discussion. The following are case notes of three such injuries produced by three different types of trauma. All three cases were seen within the latter six months of 1944.

Case I: Propeller Accident; Ruptured Jejunum

The patient—a young man of the Fleet Air Arm—gave a history of having been struck in the abdomen by the blades of a propeller, against which he was leaning when the engine of the aircraft was started. The only visible external injuries were abrasions in the right groin and over the lower half of the sternum. At first he appeared to have no serious internal injuries, and his general condition was good. He was admitted to R.A.F. sick quarters, where the medical officer wisely put him to bed and made half-hourly records of his pulse rate and blood-pressure readings. His pulse rate continued to be normal and constant, but he vomited twice. In view of his abdominal symptoms, the M.O. was not happy about him.

When I first saw the patient there were guarding and tenderness of the upper abdomen, but peristalsis was audible through the

stethoscope. In view of this observation, his general condition, the pulse rate, and blood-pressure readings, it was thought at first that his pain and guarding were probably not due to a ruptured jejunum. However, a "straight" radiograph of his abdomen (taken with a portable machine while the patient was sitting erect) showed free gas under both cupolas of the diaphragm, and clinched the diagnosis. Conditions prevailing at the time prevented his admission to our mobile hospital, and he was transferred to the Royal Victoria and West Hants Hospital.

Laparotomy, performed by Mr. Markaby, showed a laceration about the size of a threepenny-bit in the jejunum, 10 in. from the duodeno-jejunal flexure. Prolapsed mucosa had effectively plugged the laceration. There was a small quantity of free fluid in the peritoneal cavity, and some local peritonitic reaction, with flakes of lymph round the site of laceration. The small gut was dilated proximally. Distally it was empty. The laceration was closed with a purse-string suture, reinforced with a row of Lembert sutures.

Mr. Markaby, in a letter, says that recovery was uneventful, and that the patient was discharged from hospital three weeks later. Up to the time of operation (16 hours after the accident) the pulse rate had been constant and the blood pressure had remained at 135/75 mm. Hg.

Case II: Crush Injury; Ruptured Stomach

This patient was crushed between two heavy lorries, the bumper of the moving vehicle striking him in the epigastrium. His abdomen was compressed with considerable force in an antero-posterior direction. Severe abdominal pain and distress immediately followed the injury. He was admitted to our hospital two hours after the accident.

On examination he was evidently in great pain. His whole abdomen was rigid. Resuscitative measures were begun forthwith, and these were continued during the operation. The abdomen was opened through a generous left upper paramedian incision. The left upper rectus abdominis was found to be partially ruptured. The peritoneal cavity contained blood and food debris in quantity. The stomach was ruptured 3 in. from the pylorus. The rent was about 7 in. long and ran at right-angles to the long axis of the viscus, extending from the anterior surface across the greater curvature on to the posterior surface. The transverse mesocolon was split in a vertical direction from its colonic attachment to near its root. The lesser sac was thus freely opened and contained blood and food debris. The colon was not injured and its blood supply not impaired. There was an extensive retroperitoneal haematoma in the region of the pancreas.

No other injuries were found. The rents in the stomach and mesocolon were repaired. As much as possible of the blood and food debris was removed from the peritoneal cavity. The retroperitoneal haematoma was left untouched, and the abdomen was closed without drainage. Gastric suction through a nasal tube was begun immediately. The intravenous drip was kept going with glucose-saline. Penicillin, sulphathiazole, and anti-gas-gangrene therapy was started as a prophylactic measure against infection of the retroperitoneal tissues.

The patient's condition next morning was satisfactory. He said that he was in no pain but felt thirsty. He had passed urine. Twenty-four hours after the operation, however, signs of cardio-respiratory failure set in relatively suddenly, and he died four hours from the onset of symptoms. During these four hours his colour remained fairly good and his extremities were not cold. His pulse was rapid and weak. His respiration became rapid and gasping, but there was no "bubbling" in the lungs and he did not complain of pain in the chest.

Post-mortem Examination.—There was a quantity of blood-stained fluid in the peritoneal cavity. The suture lines were sound. There was an extensive retroperitoneal haematoma extending from the region of the pancreas along the line of the great vessels to the pelvic brim. The quantity of blood lost into the haematoma was not sufficient to account for death. There was considerable haemorrhage around and a little haemorrhage into the pancreas. No areas of fat necrosis were observed. The kidneys were very congested, but were unruptured. There were no injuries to the spleen, liver, or intestines. The suprarenals showed no gross abnormality. There was a tear in the internal coats of the abdominal aorta, but this had not been the source of the haematoma. The heart appeared normal. Only post-mortem clot was found in the main branches of the pulmonary artery. The lungs showed no gross abnormality. The diaphragm was not ruptured.

Case III: Kick on Left Lower Ribs; Ruptured Spleen

An airman aged 23 was kicked in the left lower ribs while playing football. He was "winded," but after a few minutes continued with the game. Later he returned to his unit by motor coach. He noticed nothing much amiss, and after a good night's sleep arose as usual next morning. At 11 a.m.—he had been injured at 4.30 p.m. the previous day—he was straining at stool, when he was seized with

acute pain in the left hypochondrium. He also noticed pain in his left shoulder. He was emphatic that these pains had not previously been experienced. His unit medical officer observed him for a short time and sent him in as a case of ? ruptured spleen.

On admission his pulse rate was 80 a minute and blood pressure 120/70. His haemoglobin was 90%. He was sweating and in pain. On examination he was tender over the whole abdomen, but especially so in the left hypochondrium and left loin. There was no rigidity. The area of splenic dullness was increased and there was shifting dullness. When the patient was turned on one side he complained of abdominal pain on that side. He was tender in the pelvic pouch. The urine was normal. Blood transfusion was started, and was continued during the operation. The abdomen was opened through an adequate upper left paramedian incision. The peritoneal cavity contained a quantity of free blood. The spleen was surrounded by a mass of blood clot and presented two large lacerations on its convex surface and on its upper and anterior border. Splenectomy was performed, and after removing the clot and free blood so far as was possible the abdomen was closed without drainage. The temperature did not settle for 10 days, but the patient felt and looked fit. Fourteen days after his operation the white cells numbered 21,000 per c.mm. (neutrophils 71%, lymphocytes 23%, monocytes 3%, eosinophils 1%, basophils 2%). He was evacuated to England, and I have no further information regarding him. The patient had been stationed in the Middle East for two years, but had never had malaria or any other illness. Histological examination of the spleen showed no pathology apart from the lacerations.

Discussion

In Case I the jejunum was ruptured as the result of a sharp glancing blow on the relaxed abdomen. The lesion produced was a small partial tear 10 in. from the duodeno-jejunal flexure. The appearance of the bowel and the near-by tissues was not such as to suggest that the bowel had been crushed against the spine by the blade of the propeller (personal observation). If this had been the mechanism responsible then one would have expected to find considerable bruising of all the tissues so forcibly compressed, and not only an isolated tear of the small gut.

The site of jejunal tears in cases of "subcutaneous" injuries is reported to be fairly constant ("near the fixed portions of the gut"). If the rupture is due to a localized crushing of the bowel, then it must follow that only those injuries in which the force is applied to these sites produce rupture, and in cases of blows elsewhere on the abdomen rupture does not occur. Can this view be accepted? (At present I have not access to the relevant literature.)

In Case II the force was of a powerful crushing nature, relatively slowly applied. Here it was the thick-walled full stomach which was ruptured, and there was extensive bruising of the tissues on the posterior abdominal wall, but no rupture of the other viscera and, in spite of the great compression of the abdomen, no rupture of the diaphragm.

In Case III the force was a sudden kick on the elastic ribs overlying the spleen. The ribs were not fractured, but they transmitted the sudden force to the organ, which was ruptured in two places. The mechanism here would seem to be the creation of a pressure wave in the semi-fluid pulp of the spleen and its contained blood, sufficient to burst open the capsule of the viscus. The length of the interval between the injury and the first onset of symptoms was striking.

In Case I the absence of any marked constitutional symptoms even 16 hours after the bowel had been ruptured was interesting.

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W. M. Johnson and O. T. Davis (*South. med. J.*, 1945, **38**, 373), who record their observations on 22 cases of acute pancreatitis, come to the following conclusions: (1) Pancreatitis is far commoner than is generally thought. (2) Pancreatitis can often be suspected clinically, and can be diagnosed almost invariably by the serum amylase test properly performed. (3) The aetiology of pancreatitis is still questionable; it may be due to a number of factors. (4) The fact that pancreatitis is far more often a medical than a surgical condition makes its recognition exceedingly important.