cytes, and 80 monocytes). Urine, N.A.D. Chest x-ray examination; both diaphragms greatly raised; no definite evidence of pleural effusion or lung lesion seen.

After the paracentesis abdominis she said she felt perfectly fit. There was never any sign of reaccumulation of fluid. A month later her E.S.R. was 20 mm. after one hour (Westergren), and three months later 8 mm. after one hour, when her haemoglobin was 85% (12.3 g. per 100 ml.). When last seen (October, 1954) she had remained in excellent health.

Case 2

A 22-year-old unmarried woman was referred to me in September, 1954, by Dr. H. Auger with a history that for six weeks she had had upper abdominal pain which came and went for no apparent reason, with some nausea and anorexia. In the same period she had noticed abdominal enlargement. On examination there was obvious ascites; otherwise nothing of importance was found. She was admitted and 5 pints (2.8 litres) of colourless fluid was withdrawn. She was afebrile apart from temperatures of 99° F. (37.2° C.) twice and 100° F. (37.8° C.) once.

Investigations.—Peritoneal fluid: clear and colourless; cells, debris only seen; Gram, no organism seen; Ziehl-Neelsen, acid-fast bacilli not found; protein, 30 mg. per 100 ml.; urea, 16 mg. per 100 ml.; culture, no growth. E.S.R., 5 mm. after one hour (Westergren). Blood count: Hb, 100% (14.8 g. per 100 ml.); W.B.C., 10,000 (7,000 polymorphs, 1,800 lymphocytes, 800 monocytes, and 400 eosinophils). Catheter specimen of urine, N.A.D. Occult blood test on stools, negative. Alkaline phosphatase, 5 units. Serum bilirubin: direct, less than 1 mg. per 100 ml., total 0.3 mg. per 100 ml. Thymol turbidity, 1; thymol flocculation, 0; serum colloidal gold, 0; bromsulphthalein test, no dye retained after 30 minutes; Mantoux test (1 in 10,000) negative. Chest x-ray examination, N.A.D. Barium-meal x-ray examination, stomach and duodenal cap outlined normally.

After the paracentesis abdominis she seemed well in every way, there was never any sign of reaccumulation of fluid, and her abdominal pain disappeared and did not return. When last seen (September, 1955) she said she had remained very well.

Discussion

These two patients were similar in that both were young women who developed transient ascites which did not recur. They differed mainly in the composition of the ascitic fluid. In Case 1 it contained 2,200 mg. of protein per 100 ml., and 100 white cells and 2,000 red cells per c.mm.; in Case 2 it contained 30 mg. of protein per 100 ml., and no cells. Case 1 also had moderate fever for a week and an E.S.R. of 30 mm. after one hour (Westergren); Case 2 had hardly any fever and an E.S.R. of 5 mm. after one hour.

The cause of the ascites is a matter of speculation. If the differences between the two cases represented a difference in aetiology (which is perhaps by no means certain) the picture in the first case suggests that some inflammatory process may have been responsible. The ascitic fluid in the second case, on the other hand, had the appearance and composition of a transudate. I can hazard no guesses at the nature of an inflammatory process which behaved in this manner. But a possible cause of a temporary peritoneal transudate seems to be thrombosis of the portal vein or one of its large tributaries.

In *Medicine* (1953)—and a similar view is expressed in other textbooks—it is stated that: "Thrombosis of the portal vein, which may be rapid and complete or slow and incomplete, usually occurs as a complication of cirrhosis of the liver and cancer of the liver. It is found at post-mortem more often than it is diagnosed clinically. A sudden complete thrombosis of the portal vein causes diarrhoea, severe gastro-intestinal haemorrhage and death. With lesser

degrees of obstruction diarrhoea and haemorrhage may be followed by rapidly developing ascites and splenomegaly. With slow and incomplete thrombosis a collateral circulation may form and various degrees of ascites, splenomegaly and anaemia may result. This is another variety of Banti's syndrome." No doubt the proved cases of thrombosis of the portal vein have been accompanied by the grave disorders just described, because the means of proof is a necropsy. Yet venous thromboses are well known to occur in, say, the axillary vein or the superior vena cava of otherwise apparently healthy people, and are merely responsible for temporary oedema of an arm or of the upper part of the body. It seems reasonable to suppose that similar thromboses might occur in the portal vein or its tributaries and cause no more than transient ascites. It is true that, according to Volwiler *et al.* (1950), ligation of the portal vein in dogs causes ascites only if plasmapheresis is also carried out. But animal experiments do not necessarily give sure indications about natural disease processes in man.

The only other recognized cause of ascites which might possibly produce only transient ascites appears to be cirrhosis of the liver (which does not always run a progressive course and is not always accompanied by other clinical and biochemical evidence of liver disease). But this seems a less likely explanation than portal-vein thrombosis.

I have been unable to find any reference in the literature to examples of benign ascites similar to these, yet, as two cases have come to my notice in six years, I find it difficult to believe that other cases of this kind have not occurred. Perhaps those who have seen such cases have cautiously refrained from publishing accounts of them because of the obscurity of their aetiology. It is partly in the hope of stimulating the description of similar cases that I submit this report.

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Medical Memoranda

A Case of "Menorrhagia"

The following case of feigned menorrhagia is described because of its unusual features, and in the hope that others in charge of the health of hospital staff may not be similarly misled.

CASE REPORT

A nurse aged 27 began duty at the hospital in July, 1954. She gave a history of appendicectomy at the age of 18, cholecystectomy at 24, and removal of an ovarian cyst at 25. Her menstrual history was normal, and physical examination revealed no abnormality except three operation scars on the abdomen.

At the time of her August period she complained of excessive loss and was unable to stay on duty. She was treated by rest in bed with injections of progesterone, and when, after four days, these had no effect, with stilboestrol. As this brought no benefit she was admitted to the gynaecological ward of a general hospital. It was noted that at the time of her transfer she did not look as exsanguinated as might have been expected, and the haemoglobin was 93%. An examination under anaesthesia and a dilatation and curettage revealed no abnormality. The bleeding soon stopped and the patient returned to the hospital where she was employed and was put back on duty. It was

decided to start a three-weeks course of ethinyloestradiol, 0.01 mg. three times daily, from the start of the next period.

When the September period came round the nurse again complained of embarrassing loss and said she could not remain on duty. Rest in bed and the hormone therapy advised produced no benefit after two days, and at the same time it was reported that two bottles of blood were missing from the refrigerator. A nurse who had cleared up the "embarrassing loss" remarked that it did not seem like menstrual discharge, and those in charge of the patient remarked that while there was plenty of blood in her bed there was little direct evidence of vaginal loss. A search of the nurse's room in her presence revealed one of the missing bottles, half full of blood, hidden in a cupboard. When confronted with this evidence she admitted stealing the blood and using it to simulate a menorrhagia.

Further inquiry revealed that at the time of the previous episode a bottle of blood had been missed, but, as it was not certain that it had not been used normally, its loss was not reported at the time. When taxed with taking this bottle and using it in the way she had used the others the patient denied any knowledge of the first bottle, but its loss remains unexplained.

At a subsequent interview the nurse said that she had lost one brother in the war, her father, of whom she was very fond, had died unexpectedly, and she did not get on well with her mother. Just before the September episode she said she had received news that her fiancé had been killed in a road accident, and she lost control of herself.

There is a note in her National Health insurance records that in 1952 she had "an attack of being unable to see," which was considered to be hysterical.

This would seem to have been a case of a woman of unstable personality, with an unhappy family background, who devised this ingenious method of malingering for some reason which was not discovered. She refused our offer to have suitable investigation and treatment arranged.

R. L. MIDGLEY, M.D., M.R.C.P.

Dissecting Aneurysm of the Aorta

During the past year two cases of dissecting aneurysm of the abdominal aorta have occurred in my practice. This leads one to suspect that the condition may not be so rare as is generally believed, and must be considered when attending any elderly patient presenting with acute pain in the abdomen or back.

The ratio of aneurysm of the abdominal to that of the thoracic aorta was stated by Osler (1905), when reporting 16 cases, to be about 1:10. Arteriosclerosis is the most important aetiological factor according to Neely (1937), who reported five cases in 1937. Goldowsky (1952), in reporting 15 cases, also stated that arteriosclerosis was a marked feature in each case. The youngest patient in his series was 55. He believes that there is an increasing incidence of ruptured abdominal aortic aneurysm.

Clinical Picture.—The clinical picture arising depends upon the abdominal organ principally involved by the extravasated blood. Abdominal pain is the most frequent symptom, occurring in all five of Neely's cases, and in 14 out of 15 cases reported by Goldowsky. The pain is almost always sharp and agonizing, the distribution depending on the point of rupture of the aorta. If extravasation occurs mainly into the retroperitoneal space the pain may be confined to the back and radiate down the legs. If the kidney and ureter are involved the symptoms suggest severe renal colic. Less commonly, if rupture occurs high in the abdominal aorta the picture may resemble acute cholecystitis, or acute intestinal obstruction, or perforated peptic ulcer as in the case reported by Manson (1937). Vomiting and gaseous eructations also occur with some frequency, as in the two cases here reported. Shock is always present and progressive, the

degree depending upon the extent of the initial tear. In the two cases reported here the blood pressure was found to drop gradually and steadily, thus contrasting with the sudden drop usually associated with coronary-artery thrombosis. The blood pressure and the volume of the pulse in the lower limbs are generally less than those found in the upper limbs.

CASE REPORTS

Case 1.—A man of 83, healthy apart from slight attacks of asthma over many years, went to bed on the night of December 22, and twenty minutes later was seized with agonizing pain in the small of his back which radiated down his legs. There was at no time any pain in his abdomen or chest. He vomited once. When I saw him twenty minutes later he was rolling about on his bed in agony, his face grey and sweating. His blood pressure was 170/120. The pulse was of poorer volume in the right than in the left anterior tibial artery. There were no other positive physical signs, and a tentative diagnosis of a leak in the abdominal aorta was made. After an injection of morphine the patient became comfortable, and his colour gradually returned to normal. One hour later his blood pressure was 140/100. He passed a quiet and comfortable night, but when seen again at 9 a.m. the next day he had some return of pain, but his condition was fair and he was cheerful. His blood pressure was now 100/90. Morphine was again given. Three hours later another severe attack of pain occurred in his back and legs, as before. His blood pressure fell to 90/70, and he again showed signs of shock. Morphine was repeated. After this his condition slowly deteriorated. At 4.30 p.m. his blood pressure was 60/50, and he gradually lapsed into a coma, dying at about 5.30 a.m. on December 24. After death the abdomen was observed to be a dark red-black and somewhat distended. Whether the condition was due to intraperitoneal bleeding or to extra-peritoneal spread could not be decided, as there was no post-mortem examination.

Case 2.—A man of 64 was seized with acute abdominal pain while attending a wedding. His previous health had been fairly good, but he had attended hospital eighteen months previously with epigastric pain. The present attack of abdominal pain was most severe in his right loin, passing downwards to the testicle. He vomited several times. When seen he was rolling about with pain, and there was some pallor and sweating. The blood pressure was 170/130. Renal colic was tentatively diagnosed, and the pain was controlled by morphine. One hour later he was seized with another attack of pain more severe than the first, during which he became shocked. His blood pressure fell to 120/80. He complained of weakness and pain in his legs, particularly the right, and the pulse in the right popliteal artery was found to be much diminished. One hour later his blood pressure was 80/60, and he gradually lapsed into a coma and died three hours later. The hospital in which the patient had been investigated was contacted, and it was found that they had diagnosed an abdominal aortic aneurysm.

CONCLUSIONS

(1) The diagnosis of dissecting aneurysm of the aorta must be considered in any elderly patient presenting with an acute abdominal crisis. (2) Abdominal pain or pain in the back is the only constant complaint. (3) Since rupture appears to have taken place in two stages—the tear involving first the inner tunics and eventually the external—death may be delayed for many hours.

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