The results are shown in Table VII. The changes in urine osmolality, urine flow, and solute output between the morning and afternoon periods in the control dehydrated group were not significantly different from the changes which occurred in the group who were dehydrated, given pitressin, and then drank 1.5 litres of water at midday.

It is concluded that in dehydration there is some factor, other than the level of circulating antidiuretic hormone (A.D.H.), which causes the urine of subjects who have been dehydrated to be more concentrated than that of subjects who have been given pitressin when hydrated. It is also evident that this factor does not disappear within two to three hours of drinking a quantity of fluid sufficient to produce a diuresis in a dehydrated subject not given pitressin.

Discussion

The finding that in normal subjects the urine concentration following dehydration is greater than after a subcutaneous injection of 5 units of pitressin tannate in oil confirms the observation made in convalescent subjects (Miles *et al.*, 1954). This difference does not appear to be dependent on the rate of solute output, for, though dehydration was accompanied by a lower solute output, the correlation between osmolality and solute output is not significant, and in two subjects given identical meals during dehydration and during administration of pitressin the concentration of the urine was greater after dehydration, though the solute outputs were the same in both experiments. The same lack of correlation is apparent from inspection of the illustrations of West *et al.* (1955), which are derived from the results of similar experiments to those reported here.* Neither is the lower concentration obtained with pitressin due to an inadequate or excessive quantity of pitressin, for the results were the same after 2.5, 5, and 10 units; nor for the following reason does it appear to be due to any qualitative difference that may exist between circulating A.D.H. and pitressin. The administration of 1.5 litres of water to dehydrated subjects who had been given pitressin was without effect on the concentration of the urine, but if given to dehydrated subjects who had *not* been given pitressin there was a prompt and substantial diuresis with a fall in urine osmolality. This diuresis was due presumably to a decrease in the level of circulating A.D.H., a decrease which must have occurred in both groups, but as there was no fall in urine concentration in those who were given pitressin it is deduced that, following dehydration, pitressin is as effective as A.D.H. in producing a concentrated urine.

It follows that dehydration is associated with some factor which, when combined with either A.D.H. or pitressin, causes the urine to be more concentrated than when pitressin is given in the hydrated state. The existence of such a factor is also implicit from the results of Shannon's (1942) experiments on dogs with diabetes insipidus, in which it is reasonable to suppose there was little or no capacity to produce A.D.H. He found that if the animals were normally hydrated the administration of pitressin did not lower the urine flow, or raise the creatinine U.P. ratio to the same level as if it were given after a period of dehydration, when the values were comparable to those obtained in normal dogs following a period of severe dehydration. Similar results have been obtained in human diabetes insipidus (Leaf *et al.*, 1952).

It is concluded that the urine following dehydration is more concentrated than after pitressin because of a mechanism which allows a greater proportion of tubular water to be reabsorbed independently of the rate of solute output, and which works equally well in the presence of either A.D.H. or pitressin.

*Since the completion of the experiments described here West *et al.* (1955) have reported their findings in a similar investigation, and some of their results have been referred to above. The procedures they used, however, and the number of experiments they performed differ so widely from those reported here that it is difficult to make many justifiable comparisons, but in general the data from both investigations are in agreement.

Summary

The urine osmolality, urine flow, and solute output after 48 hours' dehydration are compared with the values obtained after a subcutaneous injection of 5 units of pitressin tannate in oil.

The urine osmolality is greater, and the urine flow and solute output are less, after dehydration.

The difference in osmolality does not appear to be dependent on the rate of solute output or on the quantity or quality of the pitressin administered.

It is concluded that during dehydration there is a factor, other than the antidiuretic hormone or the rate of solute output, which is concerned with concentrating the urine.

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BENIGN ASCITES

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Most cases of ascites are probably due to cardiac failure, cirrhosis of the liver, or malignant disease involving the peritoneum or obstructing the portal vein. Other causes are states associated with depletion of the blood proteins, such as the nephrotic syndrome and starvation, grave anaemia, tuberculous peritonitis, the syndrome of "polyserositis," ruptured ovarian cyst, and thrombosis of the portal vein. The last-named is described as occurring as a complication of cirrhosis of the liver and of malignant disease.

According to the textbooks none of these causes of ascites is benign, and only when the condition is due to grave anaemia, starvation, tuberculous peritonitis, and the nephrotic syndrome is there said to be hope of complete recovery. I have been unable to discover in the literature any reference to ascites appearing in otherwise apparently healthy patients and later clearing up completely. The following case reports indicate that such ascites can occur.

Case 1

A married woman aged 22, referred by Dr. R. Woollett, was admitted to the Farnham Hospital in June, 1949, with the history that for two weeks she had had increasing enlargement of the abdomen. She denied having any symptoms, apart from some effort dyspnoea and nausea, and there was nothing relevant in her previous history. On examination the abdomen was greatly distended with fluid; otherwise she appeared healthy and no signs could be found. Twelve pints (6.8 litres) of yellow fluid was drained off. She ran an irregular temperature up to 100° F. (37.8° C.) for the first week, but had no fever subsequently.

Investigations.—Peritoneal fluid: slightly cloudy and yellow; R.B.C., 2,000 per c.mm.; W.B.C., 100 per c.mm. (mainly lymphocytes); protein, 2,200 mg. per 100 ml.; no acid-fast or other organisms seen; no growth on culture. E.S.R. 30 mm. after one hour (Westergren). Blood count: Hb, 75% (11.1 g. per 100 ml.); R.B.C., 4,400,000 per c.mm.; W.B.C., 4,000 per c.mm. (3,400 polymorphs, 520 lympho-

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