FIG. 2.—Left: Isolated rat kidneys 4 days after administration of ethyleneimine. Kidneys were removed and x-rayed after animal had received diatrizoate by intravenous injection. Right: Transected kidney from same animal. Note how area of opacification in radiograph corresponds to area of necrosis in renal pyramid.

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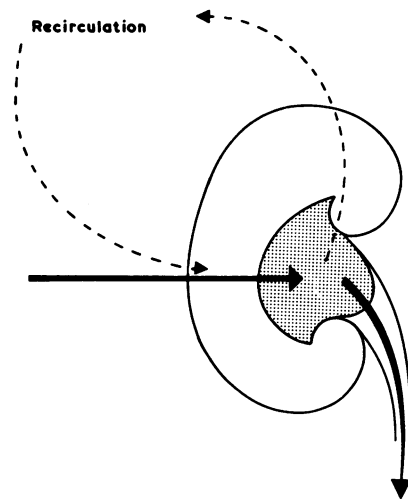
FIG. 3.—Diagram showing continuous recirculation of proportion of contrast medium excreted by kidney after it has "leaked" from damaged tubules in necrotic renal papilla.

REFERENCES

Chamberlain, M. J., and Sherwood, T. (1967). Nephron, 4, 65.
Chrispin, A. R. (1968). In Paediatric Urology, ed. D. Innes Williams, p. 533. London, Butterworths.
Chrispin, A. R., Hull, D., Lillie, J. G., and Risdon, R. A. (1970). British Medical Journal, 1, 410.
Emmett, J. L. (1964). Clinical Urography, 2nd edn., Vol. 1, p. 26. Saunders, Philadelphia.
Fry, I. K., and Cattell, W. R. (1970). British Journal of Hospital Medicine, 3, 67.
Mahaffy, R. G., Caridis, D. T., and Matheson, N. A. (1968). In Dialysis and Renal Transplantation: Proceedings of the 4th Conference of the European Dialysis and Transplant Association, ed. D. N. S. Kerr, p. 437. Amsterdam, Excerpta Medica.
Schwartz, W. B., Hurwit, A., and Ettinger, A. (1963). New England Journal of Medicine, 269, 277.

Medical Memoranda

ⓑ Polyuria after Cardiac Surgery

Diabetes insipidus is of interest as a rare disease which may be difficult to diagnose. Study of this condition in man and animals has contributed to our understanding of water balance. We present a case of what is believed to be transitory diabetes insipidus.

CASE REPORT

The patient was born in January 1954 after a normal pregnancy and delivery. At age 1 she was found to have a cardiac murmur. Subsequent investigation confirmed the diagnosis of acyanotic Fallot's tetralogy with a right to left shunt of 2:1. On 6 May 1969 she was admitted to Guy's Hospital for further investigation and surgery.

On examination she was short (height on the 3rd percentile), with normal secondary sexual characteristics but a rather distinctive facies with an absent epicanthic fold. The only other abnormal findings were those consistent with Fallot's tetralogy. Investigations were: haemoglobin 13·0 g./100 ml.; platelets 133,000/cu. mm.; coagulation normal; 24-hour urine volume 500 ml., with corrected creatinine clearance of 64 ml./min.; skull x-ray picture normal.

Operation was performed on 13 May under normothermic 36—37°C.) cardiopulmonary bypass (3·5-8 1./min.). The total bypass time was 58 minutes. The diagnosis was confirmed, the ventricular septal defect closed, and the stenosis relieved. There was immediate restoration of normal cardiac rhythm with a negligible gradient between right ventricle and pulmonary artery. Post-

operatively the blood pressure was 90/70. The hypotension was not thought to be hypovolaemic as there was a difference of only 2°C. between the peripheral and central temperatures, and the central venous pressure of 10 cm. of water was adequate.

After 10 hours isoprenaline was infused at 0·4 μg./min. This produced a satisfactory rise of blood pressure. Subsequent attempts to wean her off the isoprenaline were unsuccessful, and at 36 hours the combination of a falling blood pressure and urine output with a rising central venous pressure led to a diagnosis of cardiac tamponade. This was relieved by one of us with immediate return of blood pressure, urine output, and peripheral circulation to normal limits.

There were no further complications until the sudden onset of a diuresis on the afternoon of the fifth postoperative day. This continued for 42 hours during which time she passed about 10 litres of urine with a specific gravity of 1000-1008. Diuretics had not been given. She was not hypokalaemic, hypercalcaemic, or hyperglycaemic. There were no signs of adrenal failure. She was otherwise symptomless. The blood urea was 18 mg./100 ml. and the urea clearance 59 ml./min. At this time the differential diagnosis lay between hysterical polydipsia (or its iatrogenic counterpart of excessive intravenous therapy causing a solute and/or water diuresis) and failure in production of antidiuretic hormone.

She was given one unit of the short-acting aqueous preparation of vasopressin injection (Pitressin). This suppressed the urine flow rate from 8·8 to 0·5 ml./min. for one hour. The urine flow rate then increased again to 8·4 ml./min. until the long-acting oily preparation was given (see Fig. 1). This effect lasted for 24 hours during which time she was allowed unrestricted access to oral fluids and received a further 2·5 litres of fluid intravenously. At no time did she show any signs of water intoxication, as might have been the case had she been suffering from compulsive polydipsia. For the next four days she received 5 units of the preparation in oil daily. Her requirements subsequently rapidly
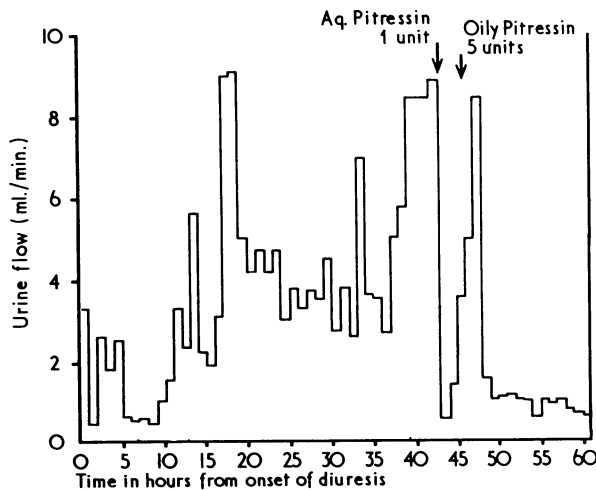
Fig. 1.—Urine flow rate.

declined and she received only 10 units over the following four days, and thereafter no further therapy was necessary. On 25 May the first fluid deprivation test (see Fig. 2) was performed, the results of which are discussed below.

An insulin stress test of pituitary function revealed a pre-insulin cortisol level of 17·5 μg./100 ml. with a rather shallow rise to 25·5 μg./100 ml. at two hours. The growth hormone response was normal. A fortnight after her last injection of vasopressin a second fluid deprivation test was normal. Her recovery was unmarred by any further complication.

COMMENT

The diagnosis of diabetes insipidus, as opposed to excess solute or water intake, rests on the response to vasopressin and the interpretation of the fluid deprivation test. Fluid deprivation sufficient to cause a loss of 3% body weight is sufficient stimulus for maximal production of antidiuretic hormone in normal subjects (Jones and de Wardener, 1956). Provided the renal concentrating mechanism is intact, a further increase in urine osmolality on injection of vasopressin
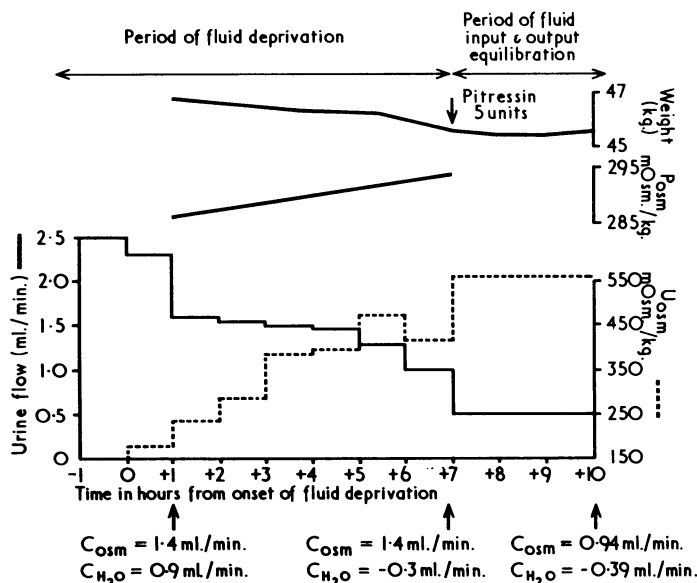

Fig. 2.—Results of first fluid deprivation test.

after an adequate period of fluid deprivation provides a clear separation of compulsive water drinking from failure in production of antidiuretic hormone (Barlow and de Wardener, 1959). In the former case there will be no further rise in urine osmolality as maximal antidiuretic hormone production is already occurring; in the latter case there will be a further rise as endogenous antidiuretic hormone is insufficient to cause maximum urine osmolality. In the present patient a rise from 417 to 553 mOsm./kg. was obtained.

During a period of fluid deprivation the urine osmolality may rise to some extent, even in the absence of antidiuretic hormone, if the glomerular filtration rate falls (Berliner and Davidson, 1957). We have no data for the glomerular filtration rate at the beginning and end of fluid deprivation in this patient. The osmolar clearance, however, was unchanged during this period in the face of a falling urine flow rate, and this latter was therefore probably not due to a decreased solute load.

After the injection of vasopressin her weight remained unchanged as she was allowed to drink a volume of water equal to the volume of urine she subsequently passed. It is therefore reasonable to assume that during this period her glomerular filtration rate and plasma osmolality remained constant and the further rise in urine osmolality was not due to a further fall in the glomerular filtration rate. During this period only one sample of urine was obtained (100 ml. three hours after administration of oily vasopressin), and some of this would have been elaborated before the exogenous antidiuretic hormone took effect. Thus the highest urine osmolality achieved after vasopressin may have exceeded the value of 553 mOsm./kg. measured in the mixed specimen.

In summary the responses to fluid restriction and antidiuretic hormone administration indicate that the patient was probably suffering from diabetes insipidus. We propose that the rise of 136 mOsm./kg. during the second half of the fluid deprivation test—which was performed during a period of only partial antidiuretic hormone dependence—represents incomplete rather than total antidiuretic hormone failure. If the test had been performed shortly after the onset of her polyuria it is likely that a much greater rise would have been obtained.

Review of the literature has failed to disclose any documented cases of diabetes insipidus as a complication of bypass surgery. It is reasonable to suppose that a partial failure in production of antidiuretic hormone may occur more commonly than has been hitherto suspected after major surgery of this kind.

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REFERENCES

Barlow, E. D., and de Wardener, H. E. (1959). Quarterly Journal of Medicine, 28, 235.
Berliner, R. W., and Davidson, D. G. (1957). Journal of Clinical Investigation, 36, 1416.
Jones, R. V. H., and de Wardener, H. E. (1956). British Medical Journal, 1, 271.