with present methods of treatment. In some patients renal-clearance values remained unchanged, and it was thought that further deterioration in these patients may have been prevented by hypotensive treatment.

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HYPERALDOSTERONISM

REPORT OF A CASE

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The syndrome of hyperaldosteronism is now being recognized with increasing frequency. The clinical picture is characterized by polyuria and polydipsia in addition to hypertension. The case reported here is of interest in that polyuria was absent and observations on the patient suggest a possible mechanism for the polyuria of this syndrome.

Case History

An assistant production manager aged 28 had previously enjoyed perfect health. When first seen in April, 1959, his sole complaint was some recent loss of visual acuity, noticed only while playing tennis. This led him to consult an optician, who referred him to hospital as a case of hypertensive retinopathy. On direct questioning he admitted to some recent tiredness but strenuously denied any polyuria, nocturia, or thirst. A routine medical examination in 1955 had shown a "high normal blood-pressure." There was no family history of hypertension.

On examination he appeared fit and active, with normal muscular power. His blood-pressure was 190/130. Both fundi showed haemorrhages, exudates, and arterial narrowing but no papilloedema.

Investigations.—A chest x-ray film showed slight cardiac enlargement and an E.C.G. showed left ventricular prepon-

derance. The blood urea varied from 24 to 39 mg./100 ml. and the creatinine clearance was 90 ml./min. After 5 units of "pitressin" the urinary specific gravity rose to 1018. Urinary catechol amines were 90 µg./24 hours. The plasma electrolytes showed a consistently abnormal pattern of hypokalaemia and alkalosis. There were seven pre-operative measurements of potassium, with a mean of 3.1 mEq/l. (range 2.8 to 3.4 mEq/l.), and six measurements of bicarbonate, with a mean of 35.6 mEq/l. (range 33 to 38 mEq/l.). This suggested the possibility of an aldosterone-secreting tumour of the adrenal cortex, and a search was made to demonstrate a tumour but with negative results. Intravenous pyelogram, aortogram, and presacral insufflation of oxygen failed to show any abnormality.

Aldosterone and Electrolyte Excretion.—The sodium intake was varied to assess changes in aldosterone excretion and the patient's ability to get into sodium balance. The Chart shows the results of these investigations. When the sodium intake was 110 mEq/day the aldosterone excretion varied between 24 and 76 µg./day (normal values 4 to 15 µg./day on free sodium intake); the 24-hour-urine volume was then between 1.9 and 2.3 l. Reduction in sodium intake to approximately 10 mEq/day was followed by a sharp fall in sodium excretion, so that the new balance was established in three days. This was achieved without a change in aldosterone excretion but was associated with a marked reduction in urine volume (and water consumption). There was some decrease in potassium excretion during this time. When the sodium intake was raised to 210 mEq/day a new balance was again established in three days, and this time there was a rise in the urine volume (and water intake) but no depression of aldosterone excretion. During this period the potassium excretion rose considerably. After the operation, when all replacement therapy had been withdrawn and the patient was on a normal sodium intake the aldosterone excretion was extremely low (1.4 µg./day). Four months later, under the same conditions, the aldosterone excretion was 1.3 and 1.2 µg./day.

Operation.—On June 8 a laparotomy was performed. A small tumour was palpated in the left adrenal and the whole gland was removed. During this procedure cardiac arrest occurred, requiring massage for three minutes, after which ventricular fibrillation began. After one shock with the defibrillator normal rhythm was restored.

Histology.—The tumour was a spherical cortical adenoma 10 mm. in diameter. The histology showed cells with an eosinophilic cytoplasm like those of the zona reticularis, but a few were filled with lipid. In places the cells showed a tubular formation with a clearly defined fibrous stroma.

Post-operative Course.—During the first two days the patient was mentally confused, but after this his mental and intellectual functions appeared to be completely normal. The blood-pressure remained at pre-operative levels for three days and then gradually fell, and by the fourteenth day was down to 140/90. A week after operation the plasma potassium had risen to 4.6 mEq/l. and the bicarbonate was then 35 mEq/l. Three days later the potassium was 5.0 mEq/l. and the bicarbonate 31 mEq/l.

Subsequent Course.—The blood-pressure has remained at normal levels since operation, the average pressure four months after being 140/90. However, although the blood-pressure and plasma electrolytes remained normal, the blood urea rose to 50 mg./100 ml. a month after the operation, confirmed on two occasions. This was interpreted as indicating renal damage, and he was readmitted in September for reassessment. This time the creatinine clearance was 130 ml./min., and after pitressin the urinary specific gravity rose to 1022. The blood urea during this stay in hospital varied between 26 and 43 mg./100 ml. A renal biopsy was attempted but no tissue was obtained. However, the white-cell excretion in the urine after an artificially induced fever did not rise outside normal limits and all urinary cultures were sterile, so there appeared to be no evidence of chronic pyelonephritis. Ten months after operation the blood-

pressure was 130/85, plasma potassium 3.7 mEq/l., plasma bicarbonate 29 mEq/l., and blood urea 37 mg./100 ml.

Discussion

The diagnosis of hyperaldosteronism was suggested by the finding of hypertension, hypokalaemia, and alkalosis. The urinary pH was between 6.7 and 7.2, as has been previously reported in this condition (Milne *et al.*, 1957). The high aldosterone excretion, which was not depressed by salt-loading, confirmed the diagnosis. It is of great interest that this patient was able to achieve sodium balance very rapidly both on a low-salt intake and on a high one, despite the unchanged aldosterone excretion. That patients with excess aldosterone of endogenous or exogenous origin can escape from the sodium-retaining effect of the hormone is well known. This "escape mechanism" has been studied by August *et al.* (1958) and involves changes in water turnover (Mills and Robinson, 1960). The results presented here show that when sodium is restricted the water turnover decreases and the polyuria disappears. In this patient the polyuria was surprisingly absent as a symptom, although the urine volume was about 2 litres. Obviously the presence or absence of polyuria depends upon the patient's level of salt intake.

The high-sodium intake caused considerable loss of potassium, and this was probably an important factor in producing the low serum potassium found on the day after operation. The cardiac arrest during the operation might have been related to this, and obviously potassium repletion should have been more energetic in the pre-operative period.

After operation, aldosterone excretion fell to very low levels and remained so when he was reinvestigated four months later. It appears that these patients may be in a position similar to those with Cushing's syndrome due to an adenoma. It is well known that after removal of such an adenoma it may be many months before endogenous A.C.T.H. production returns. It seems that the hormone controlling aldosterone secretion is also slow to return after removal of an adenoma. This apparently causes the patients surprisingly little disturbance. This patient was in sodium balance on a normal salt intake when aldosterone excretion was just over 1 μ g./day. However, at this time, in spite of having a creatinine clearance of 130 ml./min., his blood urea was 50 mg./100 ml. It seems reasonable to assume that he attained sodium balance by a mechanism the reverse of the "escape mechanism." This probably involves excessive reabsorption of fluid in the proximal tubule, as appears to happen also in cirrhotics with ascites (Schedl and Bartter, 1958), and this may cause excessive reabsorption of urea.

Some of the apparent decrease in renal function which occurs after removal of aldosterone-secreting tumours might be explained by this mechanism.

Summary

A case of hyperaldosteronism with hypertension but without polyuria or polydipsia is reported. In spite of a continued high level of aldosterone excretion the

patient was able to attain sodium balance on a varying intake. This was accompanied by alteration in the urinary volume. Removal of the tumour resulted in a cure of the hypertension.

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The new British Standard for Peters' aural specula (B.S. 3259 : 1960) provides requirements for the size of the conical joint for fitting the specula to the mount, the size range of specula, and essential features relating to the lens. The purpose of this standard is primarily to ensure interchangeability of specula fittings on the mount of the body of the instrument, and to provide a uniform size designation. The power of the lens has been specified; otherwise materials and general details of design have been left to individual choice. Price 3s. (postage extra to non-subscribers) from the Sales Branch, British Standards Institution, 2, Park Street, London, W.1.