The wide disparity in the fines inflicted raises the issue of whether such cases should continue to be tried by lay magistrates. I share the view of many that the time has come when all such cases, on summary trial, should be brought before full-time, legally qualified stipendiary magistrates.

Conclusions

Six years of service has made it clear to me that there will have to be much re-thinking on the increasing problem of the drinking driver. Not only are our laws outdated and totally inadequate, but the true significance of the magnitude of the problem and of its dangers has still to be fully appreciated by the public at large. Propaganda must be increased tenfold, and here the press, radio, television, and leaders of society should, and indeed must, play their part to the full. Above all, let us hear no more the boast of so many who ought to know better: "I drive better when I have had a drink."

In the age when denigration of the police appears to be a popular pastime, it would be remiss to conclude this survey without paying tribute to the police force in Manchester. There can be no more difficult, truculent, and even violent person than the driver under the influence of alcohol. The infinite tact, patience, and forbearance of all ranks of this police force under such circumstances were an object lesson in self-control which I envied. As far as my own dealings with the police were concerned, my opinion was received at all times with politeness and courtesy and my decisions accepted without question.

I thank all those who wrote to me, from Great Britain and abroad, after the publication of my original survey in 1960, and who asked me to continue with the task. It is hoped that this final effort will prove of some interest and use to them.

Summary

A series is described of 392 persons who over the last six years had been apprehended by the police in Manchester on suspicion of being intoxicated while in charge of a motor vehicle.

Certain clinical signs appearing together were held to be diagnostically significant: slurred speech ; full bounding pulse ; impaired memory ; poor co-ordination ; widely dilated pupils with little or no reaction to strong light ; and fine lateral nystagmus.

Of the 323 drivers who were certified as intoxicated 97% of those who appeared before magistrates' courts were convicted. The percentage of convictions fell to 49% when drivers appeared before Crown courts with a jury.

Most of the drivers (71%) were between 20 and 40. Teenage offenders were rare (1%). Thirty-two per cent. of the drivers belonged to the professional classes, and all were male. None belonged to the Jewish race.

The advent of new licensing laws and the growth of gambling clubs meant that drivers were apprehended and examined at a later hour of the night than hitherto. There was a marked rise in arrests before Christmas.

It was thought that no chemical test would be of value until legislation was enacted that laid down a blood-alcohol content above which conviction would be mandatory.

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Medical Memoranda

Plasma Renin in a Case of Conn's Syndrome with Fibrinoid Lesions: Use of Spironolactone in Treatment

Brit. med. J., 1964, 2, 1636–1637

We previously reported (Brown *et al.*, 1963a, 1964a) clinical details of a patient in whom the combination of a subnormal plasma-renin concentration and increased aldosterone secretion suggested the presence of a Conn's tumour. The present paper describes the effect of prolonged treatment of this case with spironolactone, the subsequent removal of the tumour, and the demonstration of fibrinoid lesions in the renal arterioles. The case will be reported in detail elsewhere.

Case Report

In 1961 a 33-year-old man lost consciousness briefly and his doctor found the blood-pressure was raised (190/100 mm. Hg). In 1963, after an episode of weakness in the legs, he was admitted to Ashford Hospital, Middlesex (in the care of Drs. K. D. Keele and A. Polak). His blood-pressure was then 210/130 mm. Hg, the plasma potassium was 2.6 mEq/l., and the optic fundi were normal. He was subsequently transferred to St. Mary's Hospital, where these observations were confirmed (Fig. 1). Renal arteriography revealed nothing abnormal. Total exchangeable sodium (Na_E 3,773 mEq) and aldosterone secretion (1,140 µg./day) were high, while total exchangeable potassium (K_E 2,102 mEq) and plasma renin (0.6 to 4 units/l.) were below normal.

The effect of treatment with spironolactone (tab. Aldactone-A, 300 mg./day) is shown in Fig. 1. The plasma electrolytes rapidly became normal, while the blood-pressure fell more slowly. When next measured, the Na_E (3,160 mEq) and K_E (3,280 mEq) were normal. The production of aldosterone remained unchanged, although in the later months of treatment the plasma renin was consistently in the upper part of the normal range of 5 to 18 units/l. (Brown *et al.*, 1963a, 1964c).

The spironolactone treatment was then stopped and during the next two months the plasma electrolytes, Na_E, K_E, plasma renin, and blood-pressure reverted to levels near the initial values (Fig. 1).

The patient's personal arrangements prevented operation at this stage, and he was treated with oral potassium supplements (156 mEq/day) for the next three months. During this period Na_E and K_E again became normal and the plasma renin rose, although the blood-pressure remained high (Fig. 1).

At operation (Mr. Kenneth Owen) a tumour (20 by 15 mm., weighing 5 g.) was removed from the left adrenal gland, and a biopsy of the left kidney was carried out. During the next two weeks the patient retained 911 mEq of potassium, while the net loss of sodium was 745 mEq. On his discharge three weeks later the blood-pressure was 140/90 mm. Hg, and the plasma electrolytes were normal (Na^+ 137 ; K^+ 4.8 mEq/l., Tco_2 28 mMol/l., and blood urea 26 mg./100 ml.

Histological examination (Dr. R. A. Parker) showed that the tumour was a benign cortical adenoma. The renal arterioles showed severe hyaline changes and fibrinoid lesions (Fig. 2).

DISCUSSION

The histological lesions of malignant hypertension have apparently not been reported previously in this condition (Conn *et al.*, 1964b ; Relman, 1963), although Kaplan (1963) described a case with severe retinopathy.

The effect of prolonged treatment with a spironolactone in this case is similar to that previously reported (Brown *et al.*, 1963b) in a patient in whom hypertension and overproduction of aldosterone continued after reconstruction of a stenosed renal artery. As discussed previously (Brown *et al.*, 1963b), this earlier patient, in whom the plasma renin was also subnormal, might have harboured a small tumour which was not found at operation, although, theoretically, prolonged renal disease might cause autonomous aldosterone production without an adrenal tumour. In addition to these cases, we found a subnormal plasma-renin concentration pre-operatively in a third patient (studied by permission of Professor M. D. Milne), from whom a Conn's tumour was subsequently removed. These findings confirm earlier predictions (Brown *et al.*, 1963a, 1963b, 1964a ; Laragh *et al.*, 1963 ; Kirkendall *et al.*, 1964) concerning the value of plasma-renin estimations in the diagnosis of Conn's syndrome.

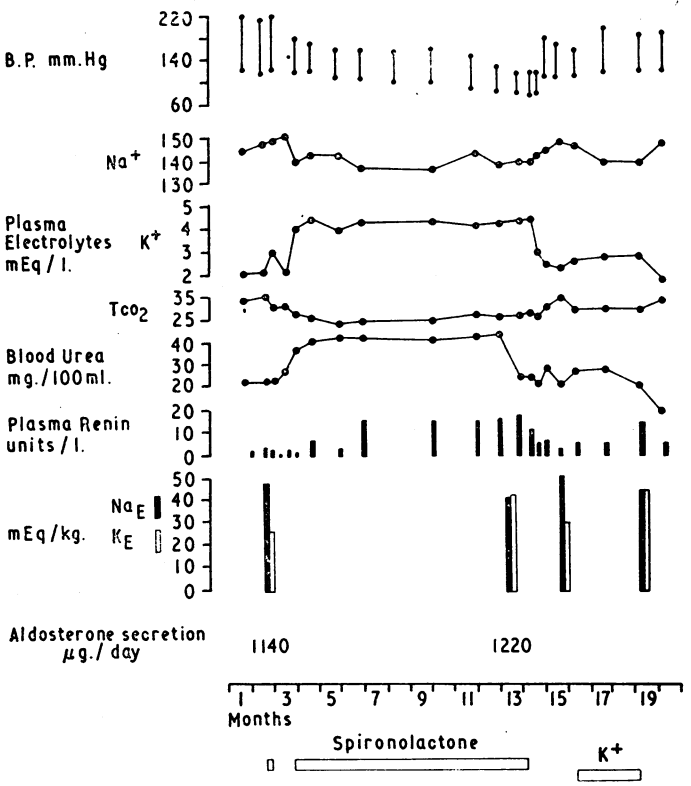

FIG. 1.—Effect of prolonged therapy with spironolactone followed by a shorter period of treatment with potassium supplements.

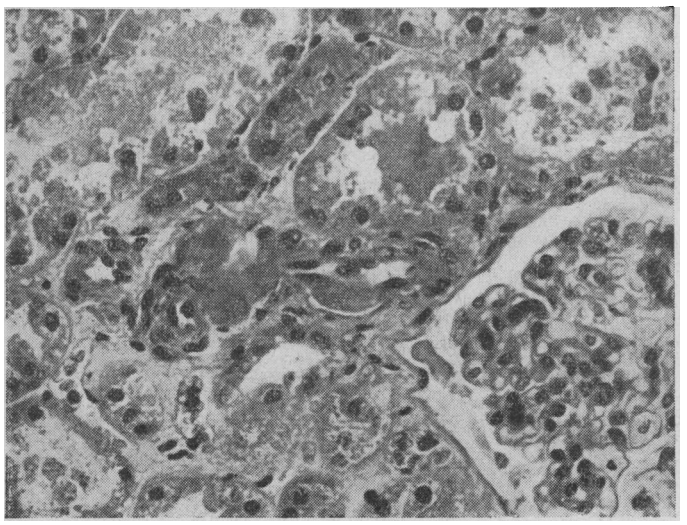

FIG. 2.—Renal biopsy showing fibrinoid arteriolar lesions. (H. and E. ×300.)

Using the technique developed by Boucher *et al.* (1964), Conn *et al.* (1964a) failed to detect plasma "renin activity" in three patients with Conn's syndrome. However, renin activity as measured in this way depends upon factors other than plasma-renin concentration (Brown *et al.*, 1964b, 1964c). Since the originators of the method failed to detect renin activity in 3 out of 16 normal subjects (Veyrat *et al.*, 1964), the test is likely to give misleading results.

Some patients with severe hypertension produce excessive quantities of aldosterone and may simulate cases of Conn's syndrome, although adrenal cortical tumours are not present (see Gowenlock and Wrong, 1962). The cause of the overproduction of aldosterone in these cases is not yet finally established, although when due to overactivity of the renin-angiotensin system the circulating renin may be high, as in a patient recently studied by us in conjunction with Dr. M. A. Barraclough (Barraclough *et al.*, 1965). The estimation of plasma renin may then help to differentiate these cases from patients with Conn's tumour. However, so far as we are aware, circulating renin and aldosterone have not been estimated concurrently in a series of hypertensive patients. It cannot, therefore, be concluded that the renin-angiotensin system is always responsible for the adrenal overactivity in these circumstances. Theoretically, excessive aldosterone production might occur in conjunction with a low plasma renin and hypertension in the absence of a Conn's tumour if aldosterone-stimulating factors other than the renin-angiotensin system were involved. The value of plasma-renin estimations in the differential diagnosis of patients with hypertension and overproduction of aldosterone must therefore await further experience, but on present evidence a high circulating renin is incompatible with the diagnosis of a Conn's tumour.

We thank numerous workers who assisted us during these studies, particularly Dr. C. L. Cope for aldosterone estimations, Mr. Kenneth Owen for the operative findings, and Dr. R. A. Parker for the photomicrograph.

J. J. BROWN, M.B., B.SC., M.R.C.P.
Lecturer in Medicine.
D. L. DAVIES, M.B., B.S.
Pfizer Research Fellow.
A. F. LEVER, M.B., B.SC., M.R.C.P.
Lecturer in Medicine.
W. S. PEART, M.D., F.R.C.P.
Professor of Medicine.
J. I. S. ROBERTSON, M.B., B.SC., M.R.C.P.
Senior Lecturer in Therapeutics.

Medical Unit,
St. Mary's Hospital,
London.

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