# SHORT REPORTS

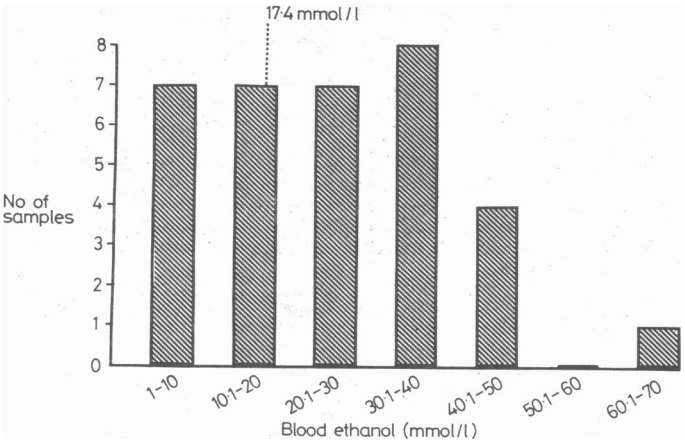
## Changing pattern of alcohol abuse in female acute medical admissions

The importance of alcoholism and alcohol related illness is well documented, and patterns of hospital admission reflect the growing problem.[1] Contemporary reports indicate that drinking problems are increasing in women and must therefore create a considerable drain on National Health Service resources. In 1979 Lennox and Tait found that 15% of female emergency admissions to a general medical unit were due or partly due to alcohol.[2] This study, performed five years later, in the same medical unit, at the same time of year, was undertaken to identify any changes in the pattern of admissions for alcohol related illness.

### Patients, methods, and results

We studied 482 consecutive female admissions (441 patients) during December 1981 to March 1982. On admission a venous blood sample was taken for blood alcohol estimation. All samples were stored at 4°C until analysed by gas-liquid chromatography.[3] Samples could not be obtained in 16 patients, who either died or took irregular discharge shortly after admission. After admission each patient was asked to complete the shortened version of the Michigan alcoholism screening test, which reportedly has the greatest predictive power of all the screening questionnaires.[4] The questionnaire was completed in 424 cases (88% of admissions), the remaining patients being unable or unwilling to cooperate. In addition, at the time of discharge each patient's consultant was invited to give his opinion on whether the patient had an alcohol problem.

Of 466 blood samples taken for alcohol estimation, 34 (7·3%) produced a positive assay result (>1 mmol/l; >4·6 mg/100 ml). In 20 patients (4·3%) the blood alcohol concentration exceeded the legal limit for driving (17·4 mmol/l; 80 mg/100 ml). The figure shows the distribution of positive results.




Distribution of blood alcohol concentrations among 34 samples found to contain alcohol. (Legal limit for driving motor vehicle 17·4 mmol/l; 80 mg/100 ml.)

*Conversion: SI to traditional units*—Alcohol: 1 mmol/l ≈ 4·6 mg/100 ml.

Alcohol was most commonly detected in women aged 31-40 years, of whom 40% had a positive assay result. The admission of 36 patients (8·2%) was considered to be due to the effects of alcohol. In 28 of these patients the screening questionnaire disclosed an alcohol problem, and the remaining eight were considered by their consultant physician to have an alcohol related problem.

On admission self poisoning was diagnosed in 55 patients (11·4%), of whom 25 had alcohol in their blood. Self poisoning was most common in the 31-40 year age group, in which 11 out of 16 patients had measurable blood alcohol values.

### Comment

The health problems caused by alcohol are being increasingly recognised, and the rising problem among women has caused particular concern. The problem has been attributed to various factors, including a reduction in the real price of alcohol coupled with an increase in disposable income of women. Advertising, particularly of fortified wines, is being increasingly aimed at women. The rapid growth of licensed premises makes the purchase of alcohol easier. The relaxation of licensing restrictions in Scotland may be particularly relevant to this study, which was conducted four years after the Licensing Scotland Act 1976 came into force.

Despite reported trends,[5] this study failed to detect a rise in alcohol related illness among female hospital admissions and, in fact, suggested a considerable fall when compared to the results of Lennox and Tait[2] ($p < 0.001$). This discrepancy may be partly explained by the fact that, in our study, 217 (45%) admissions were of women aged 70 years or over, compared with 35% five years before. If these subjects are excluded 11·6% of the women admitted had measurable blood alcohol concentrations. Similarly, the screening questionnaire plus the consultant physician's opinion showed that 14·5% of patients under 70 had an alcohol problem. Despite taking age into account, however, our results suggest at least a levelling off, and perhaps a fall, in alcohol related female hospital admissions. Reasons for this might be the present depressed economic climate, which has affected west central Scotland particularly severely, unemployment among women having almost doubled since 1977. The relaxation in licensing laws may also be relevant, as may be the higher proportion of elderly women admitted than in 1977.

We thank the consultant physicians of the Victoria Infirmary for their cooperation throughout this study.

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(*Accepted 11 March 1983*)

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## Captopril induced reversible renal failure: a marker of renal artery stenosis affecting a solitary kidney

Captopril may be effective in resistant hypertension as well as in renal artery stenosis.[1] Patients with resistant hypertension, however, often have widespread vascular disease and some degree of renal impairment. During treatment with captopril in 22 consecutive patients with "difficult" hypertension attending this clinic we found three cases of reversible renal failure. Subsequent investigation suggested that abrupt reversible renal failure during treatment with low dose captopril may be diagnostic of renal artery stenosis affecting a solitary functioning kidney.

### Case reports

Rapid deterioration in renal function occurred in three patients with hypertension taking low dose captopril (table). This was not associated with dehydration, a fall in blood pressure, proteinuria, or urinary deposit. Each patient recovered fully within a month of stopping captopril despite improved control of blood pressure with other antihypertensive drugs. In each case aortography showed a solitary functioning kidney with tight stenosis of the renal artery.

*Case 1*—This patient had had a myocardial infarction in 1967, a left iliac

*Clinical details*

| | Captopril daily dose (mg) | Blood pressure (mm Hg) | Weight (kg) | Serum creatinine (μmol/l) | Serum urea (mmol/l) | Other drugs taken during treatment with captopril | Intravenous urography | Aortography |
|---|---|---|---|---|---|---|---|---|
| Case 1 (man aged 53): | | | | | | | | |
| Before captopril | | 210/84 | 75·5 | 143 | 6·6 | Atenolol 50 mg, hydrochlorothiazide 50 mg, triamterene 100 mg | Right: contracted kidney Left: normal size; persistent nephrogram | Right: renal artery occluded at origin Left: tight stenosis of renal artery |
| Captopril (1) | 75 | 182/88 | 73·5 | 306 | 17·1 | | | |
| Captopril (2) | 150 | 180/90 | 72·7 | 372 | 19·4 | | | |
| Captopril withdrawn | | 180/84 | 72·0 | 156 | 7·2 | | | |
| Case 2 (woman aged 68): | | | | | | | | |
| Before captopril | | 195/100 | 51·0 | 188 | 6·7 | Atenolol 50 mg, frusemide 80 mg, hydralazine 200 mg | Right: end stage kidney Left: normal kidney | Massive aortic aneurysm causing left renal artery stenosis; right renal artery not seen |
| Captopril | 25 | 195/100 | 52·0 | 456 | 31·6 | | | |
| Captopril withdrawn | | 170/90 | 53·0 | 147 | 12·4 | | | |
| Case 3 (man aged 61): | | | | | | | | |
| Before captopril | | 200/100 | 66·0 | 219 | 14·9 | Methyldopa 750 mg, frusemide 250 mg, minoxidil 25 mg, prazosin 10 mg | Right: normal Left: resected | Right renal artery stenosis 5 mm from origin |
| Captopril | 25 | 170/90 | 67·0 | 411 | 30·5 | | | |
| Captopril withdrawn | | 170/90 | 66·5 | 203 | 16·2 | | | |

*Conversion: SI to traditional units*—Creatinine: 1 μmol/l ≈ 11·3 μg/100 ml. Urea: 1 mmol/l ≈ 6 mg/100 ml.

bypass graft in 1981, and left ventricular failure in 1982. He smoked 20 cigarettes a day. The most recent blood pressure measurement was 170/82 mm Hg, when he was taking atenolol 50 mg, frusemide 120 mg, and minoxidil 40 mg daily with no deterioration in renal function.

*Case 2*—This woman had a non-functioning right kidney and an abdominal aortic aneurysm affecting the renal vessels (shown by ultrasonography). The most recent measurement of blood pressure, recorded in the outpatient department, was 160/68 mm Hg, with no deterioration in renal function. She was taking atenolol 100 mg, frusemide 120 mg, hydralazine 200 mg, and prazosin 6 mg daily.

*Case 3*—This exsmoker had had an aortoiliac graft and left nephrectomy in 1979, amaurosis fugax in 1981, and left ventricular failure in 1982. His blood pressure has been controlled, on an outpatient basis, at 166/72 mm Hg. He was taking methyldopa 750 mg, frusemide 500 mg, spironolactone 100 mg, minoxidil 25 mg, nifedipine 60 mg, and prazosin 20 mg daily and had a slightly increased serum creatinine concentration (320 μmol/l (3·6 mg/100 ml)) in association with a 2 kg weight loss reflecting some dehydration.

## Comment

The most common causes of reversible renal failure after treatment with captopril are dehydration and hypotension, although the drug may induce immune complex glomerulopathy with proteinuria. Our three cases show that renal failure may occur with doses as low as 25 mg/day in the absence of any of the above factors. The risk of inducing renal failure in transplant recipients with renal artery stenosis has been known for some time.[2] A series similar to our own showed that reversible renal insufficiency may occur with bilateral renal artery stenosis or with stenosis affecting a solitary kidney, although the dose of captopril used was higher (75-200 mg).[3]

Our observations are compatible with the clinical pharmacology of captopril. In man only a small dose (5 mg) is required to prevent the vasoconstriction that results from the formation of angiotensin II.[4] Unique to the renal vascular bed, this hormone exerts a pressor effect mainly on the efferent arteriole when the pressure in the afferent arteriole falls as in renal artery stenosis.[5] Thus the glomerular filtration rate is maintained. In most cases of renovascular hypertension inhibition of angiotensin II formation by captopril is not associated with appreciable deterioration in renal function,[1] presumably because of appropriate compensation by the normal kidney. When renal artery stenosis affects the renal artery of a solitary functioning kidney, however, no such compensation is possible and the fall in the filtration fraction leads to uraemia. In the absence of other causes, therefore, deterioration in renal function during treatment with low dose captopril may be a marker of renal artery stenosis in a solitary functioning kidney.

All three patients had severe vascular disease before treatment with captopril began. Two had undergone operations for claudication and the third had an aortic aneurysm extending across the renal vessels. Because of the high risk of renovascular disease we suggest that captopril should be used with caution in such patients.


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(*Accepted 15 March 1983*)


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# Reversible neurological causes of tennis elbow

Tennis elbow or lateral epicondylitis is a common and easily recognisable condition. Despite this, its pathology is ill understood and accordingly management is largely empirical. Since it is recognised as being associated with repeated pronation/supination of the forearm[1] occupational factors are often implicated in the pathogenesis[2] and may lead to a protracted course. We report two cases in which the symptoms and signs of tennis elbow seemed to arise as a result of differing neurological disorders.

## Case reports

*Case 1*—A 55 year old telephone engineer was first seen in June 1976. Eight months previously he had noted a tendency for his right arm to shake when he tried to write. When we saw him we noted that he gripped the pen with excessive force and hyperflexion of the thumb. The right common extensor origin was tender and was injected with hydrocortisone. He received a course of electrical deconditioning for his writer's cramp, and as this improved, the symptoms of his tennis elbow became less troublesome. He was referred back again in January 1982 as his writer's cramp had recurred and was causing difficulty with his employment. Again there was associated pain over the common extensor origin which was not helped by local steroid injection. Some improvement in the writer's cramp resulted from treatment with propranolol 40 mg four times a day with some commensurate improvement in the tennis elbow.

*Case 2*—A 37 year old woman was first seen in July 1981. After a series of convulsions at the age of 3 months she had a left hemiparesis. Over the previous eight years she had been subject to complex partial seizures. The deep tendon reflexes were clearly brisker on the left and the left plantar response was extensor. The left arm was held in a dystonic posture and persistent athetotic movements of the forearm were apparent. An electroencephalogram showed no abnormalities and the seizures were controlled with phenytoin. In June 1982 she complained of pain at the left common extensor origin. On examination there was a full range of movement at the elbow but the extensor origin was tender. The pain was made worse by forced extension of the wrist. A diagnosis of tennis elbow was made and it was considered that the persistent involuntary movements were likely to be aetiologically important. Local injection of methyl prednisolone produced no improvement. Tetrabenazine in a dose of 25 mg three times a day was therefore introduced in an effort to reduce the involuntary movements. As they became much less noticeable a concomitant improvement in the elbow pain was reported. The improvement was unfortunately not maintained and in February 1983 the dose of the tetrabenazine had to be reduced because of depression.