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Captopril withdrawn		180/84	72·0	156	7·2			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Captopril withdrawn		170/90	53·0	147	12·4			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Captopril withdrawn		170/90	66·5	203	16·2			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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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.

Comment

These two cases indicate that in some cases tennis elbow may have a neurological aetiology. The first major review of the condition suggested that the main predisposing factor was repeated pronation and supination movements of the elbow leading to tears of the extensor tendon from the underlying bone.[1] This view is supported in the largest survey of tennis elbow to date,[3] but other possible aetiopathogenetic mechanisms have been suggested.[4] The repetitive movements occurring in our patients support the theory of continued minor trauma resulting in small tears of the common extensor origin.

Tennis elbow is usually a self limiting condition and treatment is by conservative means including rest, splintage, non-steroidal anti-inflammatory drugs, and local steroid injection. An appreciable minority of patients have a more chronic disability and a few of these require surgical intervention. Our two cases show that reducing repetitive movements may be of therapeutic benefit and that in a few cases this may be possible by treating an underlying neurological lesion.

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Acute central cervical cord injury due to disco dancing

Acute central cervical cord injury is usually caused by sudden extension or occasionally by flexion movements in patients with cervical spondylosis. We report a case in a patient with this injury due to headbanging—a form of dancing involving rapid flexion-extension movements of the neck.

Case report

A 15 year old boy was admitted complaining of weakness of all four limbs. On the evening before admission he had been headbanging at a discotheque and after about five minutes of this activity he suddenly developed neck pain and tingling radiating first down his left arm and then down his right arm. Both arms suddenly became weak. He stopped dancing and over the next two hours developed mild weakness of his legs and a burning sensation over both shoulders.

On admission he had a flaccid quadriparesis with severe weakness of the right arm, no movement of the left arm, and mild weakness of the legs. All reflexes were absent in the left arm and only the biceps jerk was present in the right. The knee jerks were reduced and the ankle jerks were absent with bilateral extensor plantar responses. There was a loss of pain and temperature sensation from C4-L1 on the right and from C4-T1 on the left side. There was no sphincter disturbance. A diagnosis of acute central cervical cord injury was made. Cervical spine *x* ray films were normal and a myelogram showed mild enlargement of the lower cervical cord which (in width) measured 55% of the interpedicular distance at the C5 level. There was no evidence of cord compression or disc prolapse.

He was treated with dexamethasone and over the next two weeks normal power returned to his legs. Eight weeks after admission there was still severe weakness of the left arm with only slight finger flexion and extension. There was moderate wasting of the intrinsic muscles of both hands. Power was normal in the right arm except for moderate weakness of the finger extensors and intrinsic muscles of the hand. Reflexes were normal in the right arm but the left biceps jerk remained absent with an inverted supinator jerk. Both plantar responses were flexor and dissociated sensory loss persisted on the right side from C6-C8 and from T2-T9. Pain and temperature sensation had returned to normal on the left side.

Comment

After a period of rapid flexion and extension movements of his neck this patient developed an acute central cervical cord injury characterised by weakness of all four limbs—particularly the arms—a dissociated sensory loss, and hyperalgesia of his upper chest. This occurred in the absence of any bony injury, spondylosis, or disc prolapse and in a cervical spinal canal of normal dimension. These neurological signs could result only from a central lesion in the lower cervical cord. Such a lesion would affect the corticospinal fibres, the lower cervical anterior horn cells, the spinothalamic fibres crossing the cord centrally, and also the lateral spinothalamic tracts, particularly on the left side in this patient.

The syndrome of acute central cervical cord injury was first described by Schneider,[1 2] who reported development of the syndrome in 15 patients after hyperextension injuries of the neck. Many of these patients had cervical spondylosis and the remainder had compression fractures or fracture dislocation of the cervical vertebrae. The lesion was thought to be due to acute anterior compression of the cervical cord resulting in central haemorrhagic necrosis.

The pattern of predominant upper limb symptoms has been attributed to the lamination of the corticospinal tracts with the fibres to the cervical segments lying medially in the cord at this level.[3] This proposed lamination has been disputed,[4] and Hopkins and Rudge[5] suggested that corticospinal fibres were affected as they passed medially to their termination in the lower cervical cord. Injury to these fibres with damage to the anterior horn cells would explain the phenomenon of severe upper limb weakness.

In the absence of disc prolapse, spondylosis, or canal stenosis it is difficult to explain the pathogenesis of this boy's cervical cord injury. The rapid repeated flexion and extension movement of the neck with associated lengthening and shortening of the cervical cord may possibly have resulted in a shearing injury to the vascular supply and ischaemic necrosis of the central cord.

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Correction

Infant chlamydial pneumonia

Errors occurred in the table in this short report by Dr Jane Braithwaite *et al* (30 April, p 1394-5). The correct table is given below.

Serological results (reciprocal titres in microimmunofluorescence test)

Antigen	Infant		Mother	
	IgG	IgM	IgG	IgM
C trachomatis A-C	64	0	256	0
C trachomatis D-K	256	8	64 000	0
Lymphogranuloma venereum 1-3	128	8	256	0
Herpes simplex virus 1	64	0	256	0
Herpes simplex virus 2	0	0	64	0