special reference to some published histopathologic studies in chronic uraemia. The prevailing morphological basis of chronic uraemic neuropathy is known to be axonal degeneration with secondary segmental demyelination and myelin remodelling.^{8,9} A few studies have also shown that in late and severe cases of chronic uraemia, marked pathological changes can be mostly observed in the axon accompanied by some definite changes in the myelin, compared with the early and mild cases.^{10,11} Furthermore, even when some cases had no clinical neuropathy, mild histological evidence of neuropathy, (mostly myelin changes) can be found.¹²

From these findings, we have formulated some important observations. In acute uraemia, especially in the very early stage, morphological changes in the sural nerves may not occur, while mild myelin abnormalities may be noted in the later stages. This proves to parallel the situation in chronic uraemia where duration and severity are found to be independent factors. It is believed, and with this we agree, that in acute uraemia, there may not be enough time for a specific "uraemic toxin" or the presence of a uraemic environment to cause measurable alterations in the clinical, pathological and nerve conduction studies.¹³

While research is extensively pursued regarding the mechanisms involved in uraemic neuropathy, we think our data provide further proof of the need for recognition of duration and severity in the evolution of peripheral neuropathy.¹⁴

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Anterior tibial compartment syndrome secondary to systemic capillary leak syndrome

Sir: Systemic capillary leak syndrome is an uncommon disease characterised by recur-

rent bouts of generalised oedema and hypovolaemic shock without eosinophilia. The cause is unknown and the pathophysiology involves the rapid transfer of plasma to the extra-vascular space due to an increase in capillary permeability.¹ We report here a case of this syndrome who developed a bilateral anterior tibial compartment syndrome and skeletal muscle necrosis.

A 37 year old man was admitted to the hospital because of hypovolaemic shock. Two days previously he began to complain of malaise, thirst and myalgia involving both legs, without previous strenuous exercise. On admission physical examination revealed generalised oedema and signs of shock. The haematocrit was 78% and the white blood cell count was 38 000/mm³ with 1% eosinophils. There was metabolic acidosis and hyponatraemia. Central venous pressure was -5 cm of water. Large volumes of parenteral fluid were administered and the shock improved within 24 hours. Thirty-six hours after admission, he experienced excruciating pain in both legs. On examination, both shins were swollen and tender with motor and sensory loss in the area of the peroneal nerves. The pulses were present. Creatine kinase was 4 859 IU/l (normal up to 85 IU/l), serum glutamic oxaloacetic transaminase was 166 IU/l (normal 7-20 IU/l) and lactic dehydrogenase 772 IU/l (normal 80-240 IU/l). Concentric needle electromyography of the tibialis anterior, peroneus longus and extensor digitorum brevis muscles of both legs showed abundant fibrillation and positive wave potentials. The voluntary pattern on maximal effort revealed a severe loss of motor units in all these muscles. Sensory action potentials of the peroneal nerves were unelicitable. Further examination some months later showed bilateral drop foot with atrophy of the tibialis anterior muscles, sensory loss in both peroneal nerves and the electromyogram detected signs of chronic denervation. The following studies were normal or negative: chest and abdominal radiography, abdominal ultrasonography, blood and urine cultures, echinococcus serology, Waaler-Rose, antinuclear antibodies, immunoglobulin and complement factors levels, T-3, T-4, TSH, ACTH, cortisol, catecholamines, urinary vanil-mandelic acid and 5-hydroxy-indol acetic acid. After this episode, the patient suffered two more similar bouts but without skeletal muscle necrosis. A monoclonal IgG-lambda gammopathy was discovered during the recovery period of the third episode.

Our patient had the typical clinical picture of systemic capillary leak syndrome. Other

possible causes of angioedema and hypovolaemic shock were ruled out.² The compartment syndrome was due to increased tissue pressure, secondary to plasma leakage, compromising the circulation to the muscles and nerves.³ To our knowledge this is the first reported case of bilateral anterior tibial compartment syndrome due to this syndrome.

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Matters arising

Changes of inherent muscle stiffness in Parkinson's disease

Sir: In a recent paper, Berardelli *et al*¹ suggest that changes in the intrinsic muscles stiffness could contribute to the slowness of wrist movements in patients with Parkinson's disease. This is a very interesting observation which is in agreement with the recent paper of Watts *et al*² and supports an assumption made by our research group in an earlier paper.³ In our paper electrophysiological studies of gait in Parkinsonian patients gave evidence that altered mechanical properties of muscle contribute to rigidity in this disease.

In the paper of Berardelli *et al*¹ I miss, however, any comment on a paper⁴ which came from the same laboratory a few years earlier. In this latter paper the authors stated that "... unlike the studies of Dietz *et al*² on the muscles of the leg, we could find no evidence for any fundamental changes in mechanical properties of arm muscles which would contribute to the stiffness of patients with Parkinson's disease."

It would be of interest to know in how far the discrepancies between these observations could be attributed to the different task studied, or methodical approach used in these two papers.

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Rothwell and Marsden reply:

Two quite different methods were employed

in the papers of Rothwell *et al*¹ and Berardelli *et al*.² In the paper by Rothwell *et al*¹ subjects were instructed to hold a constant joint position against an isotonic load. When the load was increased passively, the joint angle changed by the same amount and at the same speed in patients with Parkinson's disease as it did in normals. This suggests that the stiffness of the active limb was the same in both groups of subjects. This result does not conflict with that of Watts *et al*³ since these authors measured stiffness in a totally relaxed limb.

In the paper by Berardelli *et al*,² we examined rapid self-initiated wrist flexion movements and found that those of patients with Parkinson's disease were slower than normal, even though the absolute size of the first burst of agonist EMG activity was the same in both groups. One possible explanation that we put forward was a change in the active stiffness of the joint. But if this was unaffected, then there might have been a change in the EMG-force relationship of the flexor muscles, so that for a given size of EMG burst, the flexors generated less force in the patients than in normals. However, as was pointed out in the text, other explanations are possible (such as differences in skin resistance or electrode placement in the two groups), but were not investigated since this finding was a minor point in the paper.

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