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 paper[4] 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* 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. The 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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