resulted in minimal (4.7%) reduction in systolic arterial pressure. Similar results (5.5% reduction) were shown when methadone was added to the local anaesthetic drug.

These changes in arterial pressure were observed before immersion in the water bath, including the period of placing the patient in the hydraulic cradle. The patients could move themselves to the cradle almost unassisted, as motor function was only slightly affected. Ephedrine 5–10 mg i.v. was required in 7 of 144 (4.8%) patients to correct hypotensive episodes. The article cited by Dr Lim [2] showed that, using 0.5% bupivacaine 15–25 ml, a much higher rate of hypotensive episodes occurred and i.v. ephedrine had to be used in 23% of the patients. The stage of body immersion in warm water was accompanied by a small and statistically insignificant increase in systolic arterial pressure in most of the patients in our study.

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REFERENCES

PARKINSONISM AND THE ANAESTHETIST

Sir,—The effects of neuromuscular blocking drugs on patients with neuromuscular disorders have always been interesting to anaesthetists. In an excellent review on Parkinsonism and the anaesthetist [1], Severn cited the only reported case of hyperkalaemia after suxamethonium in a patient with Parkinson’s disease [2], presumably of the idiopathic type. However, because of the absence of other similar reports, doubts have been expressed on whether or not the patient had hyperkalaemia for other reasons. Azar [3] suggested that, as the patient had undergone two lumbar laminectomies for low back pain with the possibility of muscular denervation, the hyperkalaemia could have resulted from potassium release from denervated muscles rather than Parkinsonian muscles.

I measured serum potassium and arterial blood-gases before and after administration of suxamethonium in a 54-yr-old, 58-kg female with long-standing Parkinsonism secondary to use of neuroleptic drugs (table I). She was otherwise healthy and required a mandibular alveoplasty. In the previous 4 months she had twice undergone general anaesthesia; the and required a mandibular alveoplasty. In the previous 4 months she had undergone general anaesthesia; the

<table>
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<th>Table I. Serum potassium concentrations and arterial blood pH before and after suxamethonium</th>
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<td>Before induction of anaesthesia</td>
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<td>Serum potassium (mmol litre⁻¹)</td>
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<td>4.0</td>
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<td>Arterial blood pH</td>
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Sir,—Two questions should be asked of this report by Dr Ho of suxamethonium-induced hyperkalaemia in a patient with Parkinsonism. First, could the hyperkalaemia have resulted from any other cause? Second, is there any independent evidence of a functional change in the neuromuscular junction that might lead one to expect an abnormal response to suxamethonium?

In his case report, Gravlee [1] discussed the possible reasons for an increase in serum potassium from 4.2 to 7.6 mmol litre⁻¹ following the infusion of suxamethonium 800 mg. He concluded that the most likely explanation lay in the effect of Parkinsonism on the modulation of lower motor neuron activity.

Sica and colleagues [2] provided evidence of changes in the neuromuscular junction in Parkinsonism. They demonstrated abnormally large muscle action potentials in the extensor

BRITISH JOURNAL OF ANAESTHESIA

REFERENCES