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.

B. Drenger
Baltimore

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 undergone general anaesthesia; the usual clinical doses of suxamethonium and pancuronium had been given, with no unusual effect detected. Anaesthesia was induced with fentanyl 0.15 mg, thiopentone 250 mg and suxamethonium 80 mg. Nasotracheal intubation and recovery from muscle paralysis were totally uneventful.

The blood-gas and serum potassium data were compatible with previously published data. Cooperman [4], in an unspecified number of Parkinsonian patients, observed insignificant changes in serum potassium concentrations after suxamethonium and the results of the regional curare test on eight Parkinsonian patients (aetiology not specified) were found to be within normal limits [5]. Nevertheless, until more conclusive evidence attesting to its safety is available, I am in agreement with Dr Severn in urging caution in the use of suxamethonium in patients with Parkinson’s disease. Moreover, as with any neuromuscular disorder, neuromuscular function should be monitored when neuromuscular blocking drugs are administered to patients with Parkinsonism.

A. M.-H. Ho
St Catharines, Ontario

REFERENCES

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, Gravelle [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 neurone 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

### Table I. Serum potassium concentrations and arterial blood pH before and after suxamethonium

<table>
<thead>
<tr>
<th></th>
<th>Before induction of anaesthesia</th>
<th>After intubation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2 min</td>
<td>8 min</td>
</tr>
<tr>
<td>Serum potassium</td>
<td>4.0</td>
<td>4.1</td>
</tr>
<tr>
<td>(mmol litre⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arterial blood pH</td>
<td>7.37</td>
<td>7.40</td>
</tr>
</tbody>
</table>
CORRESPONDENCE

digitorum brevis and calculated that there is a reduction in the number of functioning motor units of the deep peroneal nerve. They suggested that undamaged motor neurones take over the function of adjacent damaged nerves, and that this damage occurs because of the loss of some trophic influence from higher centres.

There would seem, therefore, to be adequate cause to justify Gravlee’s concern. The question of reproducibility of results is a different matter. Sica and colleagues claimed that the reduction of functioning motor units occurs most rapidly in the first 9 months of the disease (although they acknowledged the difficulty of defining the onset of Parkinsonism). Gravlee’s patient had long standing and well controlled Parkinsonism, the symptoms of which worsened rapidly on withdrawal of L-Dopa 5 days before surgery. Is it possible that clinical deterioration was associated with an acute loss of functioning lower motor neurones, with a predictable response to suxamethonium? It would be worth adding this complication to the list of dangers of abrupt preoperative withdrawal of L-Dopa.

A. M. SEVERN
Manchester

REFERENCES


PROSTAGLANDINS AND THE LOWER OESOPHAGEAL SPHINCTER

Sir,—The paper by Jones and colleagues [1] considered lower oesophageal sphincter (LOS) tone in a group of patients undergoing suction termination of pregnancy. All those studied had been given gemeprost, and although the possible effect of prostaglandins on the LOS was mentioned, it was not explored fully in the paper.

Prostaglandins of the E group are known to relax circular smooth muscle in the gastrointestinal tract of humans and some animals, with the consequent effect of lowering the LOS tone [2]. Prostaglandins of the F group, however, are excitatory to this group of muscles, and therefore cause LOS pressure to increase [3].

Furthermore, indomethacin has been shown to increase the tone of the LOS in human subjects [3], possibly because of its inhibitory effect on prostaglandin synthetase, leading to a reduction in endogenous prostaglandin E within the LOS muscle.

Gemeprost is a prostaglandin E analogue, and although its effect on the cervix is mainly by a local action, some systemic absorption must occur also. To rule out any effect this may have on the LOS tone, it would be interesting to know the results of a similar study in a comparable group of patients not given gemeprost.

A. MAHONEY
London

REFERENCES


Sir,—Thank you for giving us the opportunity to reply to Dr Mahoney’s letter. We welcome his comments regarding the effects of prostaglandins on the LOS. Gemeprost, a prostaglandin E analogue is now used almost universally in the first trimester before termination of pregnancy because of its beneficial local action on the cervix. The effects of this agent on the LOS are unknown and we would agree that a study comparing barrier pressures in a group of patients not given gemeprost would be of interest.

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