Myasthenic syndrome during treatment with practolol

SIR,—The myasthenic syndrome during treatment with large doses of practolol described by Drs R O Hughes and F J Zacharias (21 February, p 460) appears to be a clinical manifestation of the effect of β -adrenoceptor blocking agents on the neuromuscular junction observed in animals. Like other members of this group of drugs^{1,2} practolol has a curare-like action, as shown in sciatic nerve-gastrocnemius preparations of the cat³ and frog and in the frog sciatic nerve-sartorius preparation and rectus abdominis muscle.^{2,4} The dosage required to depress neuromuscular transmission was larger for practolol than for propranolol.

Though the patient's electromyogram, presumably taken in the resting state, was normal, it would be of great interest to know whether in the myasthenic state repetitive nerve stimulation evoked a gradually declining response of the muscle and whether even in the absence of the myasthenic syndrome the patient was particularly prone to fatigue. As in experimental animals the neuromuscular effects of β -adrenoceptor blockers are readily reversible the risk of a relapse of the myasthenic syndrome seems to be remote. However, the patient may be hypersensitive to D-tubocurarine and related agents and also to drugs with a local anaesthetic action, like quinidine.

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- ¹ Wislicki, L., and Rosenblum, I., *Archives Internationales de Pharmacodynamie et de Therapie*, 1967, **170**, 117.
- ² Wislicki, L., *British Journal of Pharmacology*, 1969, **36**, 174P.
- ³ Wislicki, L., *Israel Journal of Medical Sciences*, 1970, **6**, 224.
- ⁴ Werman, R., and Wislicki, L., *Comparative and General Pharmacology*, 1971, **2**, 69.

Plasma prolactin levels during a simulated dive

SIR,—Prolactin secretion in both sexes rises in response to physical and emotional stresses.^{1,2} The only natural stimulus which has been reported to reduce plasma prolactin levels is water loading³ and this effect has been disputed.⁴ We are interested in fluid and electrolyte responses to diving, and since prolactin can cause fluid and electrolyte retention⁵ we measured plasma prolactin levels in five men and one woman before and immediately after a simulated dive in the hyperbaric chamber in the department of surgery of Newcastle University. The chamber is small and we expected that as a result of the stress of being confined in it prolactin levels in the subjects would rise.

Blood samples were taken before entry to the chamber and after a two-hour simulated dive to a pressure of two atmospheres while breathing air. Prolactin levels were measured by radioimmunoassay with materials supplied by the US National Institute of Arthritis and Metabolic Diseases. The mean plasma prolactin concentration before the dive was 12.9 ± 3.3 (SEM) $\mu\text{g/l}$. The mean concentration afterwards was 3.9 ± 0.9 $\mu\text{g/l}$ ($P < 0.05$). In every subject the prolactin level fell during the dive. This completely unexpected fall in prolactin level may contribute to the diuresis which is a practical problem during dives.

The precise stimulus remains unknown.

The three obvious possibilities are the rise in pressure per se, the rise in PO_2 , and the rise in PN_2 . The same subjects were tested before and after breathing either 10% oxygen in nitrogen or 100% oxygen at normal atmospheric pressure. Prolactin levels tended to rise in both cases, suggesting that it may be the rise in pressure per se which is responsible.

We thank the members of the Newcastle University Diving Club for their enthusiastic co-operation.

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- ¹ Frantz, A G, Kleinberg, D L, and Noel, G L, *Recent Progress in Hormone Research*, 1972, **28**, 527.
- ² Noel, G L, et al, *Journal of Clinical Endocrinology and Metabolism*, 1972, **35**, 840.
- ³ Buckman, M T, and Peake, G T, *Science*, 1973, **181**, 755.
- ⁴ Adler, R A, et al, *Journal of Clinical Endocrinology and Metabolism*, 1975, **41**, 383.
- ⁵ Horrobin, D F, et al, *Lancet*, 1971, **2**, 352.

Recognising hiatus hernia

SIR,—With reference to your leading article on this subject (19 June, p 1490) I agree that there can be great difficulty in demonstrating the presence of a small hiatus hernia.

In pointing out the difficulty for the endoscopist when determining the axial relationship between the squamocolumnar epithelial junction and the diaphragmatic hiatus your comments are justified if you are referring to simple forward viewing (with the viewing tip of the endoscope situated in the lower end of the oesophagus). I would, however, commend to your readers a simple endoscopic manoeuvre which I have frequently found to be most helpful in detecting the small hernia when, after forward viewing, the issue remains in doubt. I have found that by using reverse flexion (or J-flexion) of the endoscope within the stomach cavity one obtains a far better appreciation of the presence of a small hiatus hernia when looking at it from below. When viewed in this way the hernia is seen as a distinct recess with both a definite upper extremity through which the endoscope emerges from the oesophagus and a clearly defined lower extremity where the diaphragmatic impression on the stomach is very readily recognisable. The air insufflation necessary to distend the stomach sufficiently to permit reverse viewing may in fact help to unmask the small or latent hiatus hernia, but it is unlikely that it ever causes any artefactual distortion of the anatomy at the oesophagogastric junction.

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Diabetic ketoalkalosis

SIR,—We read with interest the report of patients with diabetic ketoalkalosis by Drs K C Lim and C H Walsh (3 July, p 19). Recently we have treated a patient who presented with similar clinical features.

A 68-year-old man with previously undiagnosed diabetes was admitted with a history of malaise and nausea for three months with prolonged vomiting for three days. He was drowsy, dehydrated, and tachypnoeic with acetone on his

breath. Urine analysis showed 2% glucose and 3+ ketones. Diabetic ketoacidosis was initially diagnosed and he was treated with intravenous saline, potassium supplements, and insulin by low-dose infusion. Subsequently the results of the initial investigations showed a metabolic alkalosis. The blood standard bicarbonate was 29.0 mmol (mEq)/l, hydrogen ion concentration 29.5 mmol/l (pH 7.53), and Pco_2 3.6 kPa (27 mm Hg); the blood glucose level was 35.3 mmol/l (636 mg/100 ml), serum sodium 139.0 mmol (mEq)/l, serum potassium 3.9 mmol (mEq)/l, and blood urea 21.5 mmol/l (130 mg/100 ml). Apart from a slightly elevated blood urea all biochemical values were normal four hours later except for the blood glucose, which fell steadily to within the normal range over the next eight hours. On discharge from hospital the patient's diabetes was well controlled with tolbutamide 1 g daily.

As in the cases described by Drs Lim and Walsh, severe prolonged vomiting was a major presenting feature in our patient, who differed, however, in that he was not previously known to be diabetic. We would accordingly agree with their conclusion as to the importance of a hydrogen ion measurement in the initial investigation of patients suspected to be in diabetic ketoacidosis, and these cases further underline the potential hazard of routine bicarbonate therapy in their initial management.

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Short-term recovery of mental efficiency after anaesthesia

SIR,—I should like to congratulate Mr J E S Simpson and his colleagues (26 June, p 1560) on drawing attention to the problems of recovery after anaesthesia for day case surgery.

In the study reported the fentanyl group received pancuronium, a long-acting muscle relaxant comparable in duration of action to curare, of which Lee's *Synopsis of Anaesthesia*¹ says, "Occasionally some residual paresis of the muscles of accommodation persists for 24 hours after operation, making reading difficult." This was neatly demonstrated by Hannington-Kiff² in the case of gallamine reversed by neostigmine by using a Maddox wing to measure the balance of the extraocular muscles. In the performance efficiency tests used a difficulty in focusing will reduce the score, and it is significant that two of the 18 patients in the fentanyl group were unable to do the cancellation of letter pairs test because of diplopia 7-10 hours after operation. It is thus possible that a misleadingly low result for the mental efficiency of the fentanyl group was obtained owing to the residual effect of the long-acting muscle relaxant on the eye muscles rather than that they were less alert than the halothane group.

If the time taken for the premedication to work is one hour, the operation time is 35 minutes, the time for the patient to be fit for discharge is 7-9 hours, and the day unit is open from 8-30 to 5 pm, the number of patients that can be operated on is rather limited.

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¹ Lee, J A, and Atkinson, R S, *Synopsis of Anaesthesia*, 7th edn. Baltimore, Williams and Wilkins, 1973.

² Hannington-Kiff, J G, *Proceedings of the Royal Society of Medicine*, 1970, **65**, 73.