CORRESPONDENCE

HYPERSENSITIVITY TO PANCURONIUM IN A PATIENT WITH VON RECKLINGHAUSEN'S DISEASE

Sir,—Hypersensitivity to neuromuscular blocking drugs has been known to occur in patients with von Recklinghausen's disease (Magbagbeola, 1970; Manster, 1970; Yamashita and Matsuki, 1975). We can find no report subsequent to that described by Yamashita and Matsuki (1975).

Recently, we experienced a hypersensitive response to pancuronium in a patient with von Recklinghausen's disease.

A 42-year-old female patient with multiple neurofibromatosis (weight 35 kg) underwent sigmoidectomy under halothane (less than 1%) and nitrous oxide in oxygen anaesthesia. Following induction of anaesthesia with thiopentone, the trachea was intubated with the aid of suxamethonium 20 mg i.v.

After the reappearance of spontaneous respiration, pancuronium 0.5 mg was administered i.v. With this dose muscle relaxation was satisfactory for surgery until the time of peritoneal closure, at which time a further dose of pancuronium 0.5 mg was administered. Thus pancuronium 1.0 mg was sufficient for a sigmoidectomy which lasted about 1 h.

Although not all patients with von Recklinghausen's disease show abnormal hypersensitivity to neuromuscular blockers, some do respond abnormally. Careful titration of the dose of these drugs is advised in such patients.

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ADENOSINE AND LEG BLOOD FLOW DURING PORCINE MALIGNANT HYPERThERMIA

Sir,—In a recent study we observed that the failure of leg blood flow to increase during porcine malignant hyperthermia (MH) limited oxygen delivery to the muscle and contributed to the rapid onset of lactic acid production (Hall et al., 1982). Although the decline in cardiac output during MH is likely to be a major factor in causing this decrease in leg blood flow, the release of vasoactive compounds from active muscle is also important in regulating local blood flow (Haddy and Scott, 1968). Adenosine has been implicated as a vasodilator in muscle, particularly in the presence of ischaemia (Rubio, Berne and Dobson, 1973; Bockman, Berne and Rubio, 1975, 1976; Tominaga et al., 1980). In an attempt to increase muscle blood flow during MH we have examined the effects of the administration of adenosine on the hyperthermic response in five Pietrain pigs (mean weight 50 kg).

The experimental preparation of the pigs and the measurements undertaken have been described previously (Hall et al., 1982). After control samples were collected adenosine 1 mmol (267.2 mg) was infused i.v. over 10 min. MH was then induced by ventilating the lungs with 1% halothane for 10 min and the administration of suxamethonium 1 mg kg⁻¹ after 5 min ventilation with halothane. Samples were collected every 10 min until the pig died.

The metabolic course of MH was unaltered by adenosine and was similar to that described in the Pietrain pig (Hall et al., 1980, 1982). Similarly, leg blood flow was not increased significantly compared with untreated pigs in spite of a two- to three-fold increase in circulating adenosine concentrations (Hall et al., 1982). We conclude that the failure of the infusion of adenosine to increase leg blood flow in MH supports our previous contention of the primary importance of cardiac output in determining peripheral blood flow. We cannot exclude the possibility, however, that increasing muscle oedema and rigidity during MH are also important factors in reducing muscle blood flow.

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