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Pancuronium and the Patient with Myasthenia Gravis

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Pancuronium, a nondepolarizing neuromuscular blocking agent,[1] has recently been made available for general clinical usage in the United States. Patients with myasthenia gravis should be "sensitive" to pancuronium. To test this hypothesis we evaluated the neuromuscular blocking effects of pancuronium in two myasthenic patients undergoing thymectomy.

METHODS

An 8-year-old boy and a 21-year-old man, with myasthenia gravis of 4 and 6 months' duration, respectively, were studied. Written consent was obtained for each study. All anticholinesterase therapy was discontinued 8 hours prior to operation. Premedication consisted of atropine sulfate, 0.3 and 0.4 mg, im, respectively, one hour prior to induction of anesthesia. Following topical nasopharyngeal anesthesia with 2 per cent tetracaine and transtracheal block with 4 per cent lidocaine, a nasotracheal tube was passed into the trachea with the patient awake. Anesthesia was then induced with sodium thiopental and maintained with 70 per cent nitrous oxide and meperidine, iv. Esophageal temperature was maintained between 35 and 36.5 C. Controlled ventilation kept Pa_{CO_2} between 36 and 40 torr for the duration of the study. Neuromuscular transmission was studied by supramaximal stimulation of the ulnar nerve at the wrist with a Wellcome peripheral nerve stimulator, and thumb adduction was measured with a force-displacement transducer and recorded on a Brush recorder.[2]

A dilute solution of pancuronium bromide containing 0.01 mg/ml was infused at a rate of 1 ml/min until twitch height was reduced to 50 per cent of control. When twitch height had recovered to 60 per cent of control, additional pancuronium was administered to reduce twitch height to 5 per cent of control (95 per cent twitch depression) and the infusion of pancuronium then stopped. When twitch height had recovered to 10 per cent of control, 2.5 mg neostigmine and 1 mg atropine were administered iv.

RESULTS

Infusion of pancuronium, 0.1 mg/m² BSA, or 0.0033 mg/kg in Patient 1 and 0.0025 mg/kg in Patient 2, over a 10-minute period, reduced twitch height to 50 per cent of

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control (table 1). After recovery to 60 per cent of control twitch height, an additional 0.05 mg pancuronium in one patient and an additional 0.7 mg in the other were needed to obtain 95 per cent twitch depression. This represents total doses of 0.15 and 0.45 mg/m² BSA, or 0.005 and 0.011 mg/kg, respectively, to obtain 95 per cent twitch depression. The 95 per cent twitch depression lasted approximately 35 minutes. Following administration of neostigmine when twitch height had returned to 10 per cent of control value, 90 per cent recovery of control twitch height was obtained in 15 minutes, with minimal fade on tetanic (50 Hz) stimulation. Each patient was awake, responsive, and breathing spontaneously with the nasotracheal tube in place at the conclusion of the operation.

TABLE 1. Twitch Depression and Pancuronium Dosage in Two Myasthenic Patients

	Per Cent Twitch Depression	Pancuronium Dose (mg/m² BSA)	Pancuronium Dose (mg/kg)
Patient 1 8 years old, 1.02 m² BSA, weight 30 kg	50	0.1	0.0033
Patient 2 21 years old, 2.02 m² BSA, weight 80 kg	50	0.1	0.0025
Patient 1	95	0.15	0.005
Patient 2	95	0.45	0.011

Discussion

It is well known that the patient with myasthenia gravis is "sensitive" to nondepolarizing muscle relaxants.[3,4] Our data indicate that the myasthenic patient is "sensitive" to the new nondepolarizing drug, pancuronium. Miller and colleagues[5] have shown that 0.82 mg/m² BSA pancuronium was necessary to achieve 50 per cent twitch depression during anesthesia with 70 per cent nitrous oxide and 0.4 per cent halothane. Although we cannot compare halothane anesthesia with nitrous oxide/oxygen/narcotic anesthesia, we may surmise, matching our data with those of Miller, that the myasthenic patient is at least eight times as sensitive to pancuronium as the nonmyasthenic patient. Because Miller *et al.* used halothane anesthesia, which is known to potentiate the action of nondepolarizers,[5] myasthenic patients are perhaps even more sensitive than the present report indicates, as we used nitrous oxide/oxygen/narcotic anesthesia.

Lund and Stover[6] showed that in conscious volunteers using grip strength as an indicator, 50 per cent depression was obtained by 0.014 mg/kg of pancuronium. Although, again, it is difficult to compare the responses of conscious volunteers with those of anesthetized patients, our data indicate sensitivity at least five times as great for the myasthenic patient compared with the nonmyasthenic patient, when compared with Lund's data.

Data of Katz[7] indicate that 0.04 to 0.08 mg/kg pancuronium is necessary to produce 95 per cent twitch depression. Our data indicate that the myasthenic patient is at least four times as sensitive as the normal patient with regard to this extent of depression of twitch when compared with Katz's data.

The pancuronium was easily antagonized by neostigmine at 10 per cent of control twitch height. This concurs with findings of Katz[7] and Miller *et al.*[8] with regard to both the dosage and the time course of reversal of neuromuscular blockade. We realize that the variability of the studies, *i.e.*, Grass stimulator versus Wellcome nerve stimulator, makes the comparisons somewhat empiric. We feel they are valid, nevertheless.

The current popularity of "balanced anesthesia" combined with the potential viscero toxicity of some halogenated anesthetic agents may cause the anesthesiologist to select a nitrous oxide/oxygen/narcotic/muscle relaxant technique for the patient with myasthenia gravis undergoing operation. Our data indicate that this is a satisfactory technique provided the patient is not given an excessive dosage of a nondepolarizing muscle relaxant. In this regard pancuronium appears safe for the patient with myasthenia gravis, but we recommend unequivocally the use of a nerve stimulator to monitor neuromuscular blockade, to avoid overdosage with muscle relaxant.

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A Simple Method to Determine Patency of the Ulnar Artery Intraoperatively Prior to Radial-artery Cannulation

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Percutaneous radial-artery cannulation for continuous pressure monitoring and arterial blood–gas sampling is now routine in operating rooms and intensive care units. The incidences of thrombosis of the radial artery after cannulation have been as high as 20–60 per cent in recent series[1–3]. Even with complete occlusion of the radial artery, ulnar-artery collateral circulation usually prevents serious sequelae to the area of the hand whose normal vascular supply is from the occluded artery. Assessment of the patency and distribution of ulnar-artery flow prior to radial-artery cannulation is, therefore, necessary.

The most commonly employed method is the modified Allen's test.[4] This technique is simple and requires no special equipment. However, it does require patient cooperation, adequate lighting for observation of the blush, and freedom to move and manipulate the patient's hand. Allen's test cannot be performed intraoperatively while the patient is anesthetized.

In those cases in which unanticipated intraoperative radial-artery cannulation is indicated, a simple method using plethysmography to determine ulnar-artery patency and distribution can be performed. A finger-pulse transducer (Model T-301, Lexington Instru-

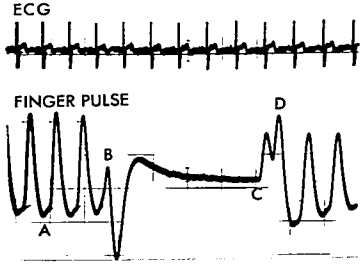

FIG. 1. The finger-pulse transducer is placed over the patient's thumb and a pulse contour is noted (*A*). The examiner compresses both the radial and ulnar arteries (*B*), resulting in loss of the pulse on the monitor screen. Release of the pressure over the ulnar artery (*C*) results in immediate return of pulsations in the thumb, due to flow through collaterals (*D*).

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