that cover the entire range up to 100 pounds are available, by changing load cell accessories it can be used to study a variety of muscles having different forces of contraction.

We have used this transducer and holder for as long as four hours with no baseline drift. No problem has been encountered using the transducer. Stability of the baseline is good, showing no drift from control to complete paralysis and then recovery (fig. 4). The sensitivity of the recording system for detecting impaired transmission is beyond that which can be noted by watching the hand responses. Even minute amounts of fatigue with tetanic stimulus are evident from examination of a written record.

REFERENCE


Burns of the Skin Caused by a Peripheral-nerve Stimulator

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A review of the anesthesia literature has revealed no report of burns resulting from the use of the Wellcome peripheral nerve stimulator to monitor the effects of neuromuscular blockers. This report documents that surface burns may result from the use of this instrument.

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REPORT OF A CASE

An obese 17-year-old girl was anesthetized with nitrous oxide, meperidine, and d-tubocurarine for an elective cholecystectomy. The patient's prior medical history was unremarkable.

Muscle relaxation for the operative procedure was obtained with d-tubocurarine. The degree of neuromuscular block was evaluated by intermittent stimulation of the median nerve with a Wellcome peripheral nerve stimulator, using the spherical-ended probe electrodes. The probes were applied firmly to the skin over the area of the median nerve at the wrist; excessive pressure was avoided. A tetanic stimulus was applied for a period of approximately 5 seconds. The result was a fade of tetanus (Wedensky inhibition). The unit was set to deliver maximum stimulation.
During the course of the procedure, the ventral surface of the arm developed erythema, followed by the appearance of punctate lesions, which subsequently became vesicular (fig. 1). The punctate lesions were 1 mm in diameter, and the entire circle was approximately 3/4 cm in area. Dermatologic consultation confirmed that the lesions were burns.

We found that burns were produced on five successive patients whenever the spherical-ended probes were used, even with different units of the Wellcome peripheral nerve stimulator. A single application of tetanic stimulation of 5 seconds' duration was sufficient to produce the lesions. The voltage was varied from the scale-indicated maximum to three fourths of maximum and half of maximum. Burns resulted at each setting. Half-power was the minimal level of stimulation that would elicit a noticeable muscular contraction. Further studies indicated that burns also resulted from a single application of full-strength tetanic stimulation of 5 seconds' duration with and without the use of electrode paste. The patients did not complain of pain from the burns, and the lesions healed within three days.

The Wellcome peripheral nerve stimulator is a battery-powered transistorized instrument designed to monitor, either continuously or intermittently, the effects of neuromuscular blockers on the myoneural junction. The instrument has a no-load (open-circuit) output of 375 volts and a single-pulse duration of approximately 1.3 msec. A single twitch stimulus is automatically produced at either 5- or 10-second intervals. Stimuli at frequencies of 1/sec or 50/sec may be selected as desired. The output of 375 volts is more than adequate to provide supramaximal stimulation of peripheral nerves using surface electrodes. The tetanic stimulus rate of 50/sec ensures a full tetanic response of stimulated nerve groups.

Two types of surface electrodes are supplied for use with the instrument. One is the plate electrode, designed to be securely affixed to the patient's arm or face. The second type is the spherical-ended probe, which is molded into a compact twin-plug unit. Without the use of needles, this electrode allows intermittent monitoring; it is suitable for quick ward or recovery room use whenever muscle paralysis from neuromuscular blockers is suspected following surgery.

The manufacturer advises that the surface electrodes should be moistened with a conductive jelly or saline solution, but that this is not essential except during use for obese patients.

Literature supplied by Burroughs, Wellcome and Company (Australia) Ltd. with their peripheral nerve stimulator suggests using the device (Wellcome peripheral nerve stimulator) for routine surface stimulation of the facial nerve, either where a branch crosses the zygomatic arch or where it issues from the parotid gland.

It should be noted that while the Wellcome peripheral nerve stimulator is a valuable tool for monitoring neuromuscular blockade, there is a distinct possibility that it will produce small punctate burns even when the manufacturer's recommendations are followed.

In view of the foregoing observations, the authors recommend that routine use of the spherical-ended probes in the area of the facial nerve or in other exposed areas be avoided. Burns have not been observed with the use of needle electrodes.
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Prolongation of a Pancuronium-induced Neuromuscular Blockade by Polymyxin B

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Several antibiotics prolong the neuromuscular blockades induced by nondepolarizing neuromuscular blockers.1 We report an instance of pancuronium-induced neuromuscular blockade which was apparently prolonged as a result of intraoperative wound irrigation with Polymyxin B and, possibly, bacitracin. Pyridostigmine, neostigmine, edrophonium, and calcium chloride were either ineffective or incomplete antagonists of this blockade.

REPORT OF A CASE

A 76-year-old man was admitted in Nov. 1972 for open reduction and internal fixation of a right femoral neck fracture. His most recent operation had been a resection of a thoracic aortic aneurysm in 1968 under general anesthesia without known problems. No medication was taken prior to hospitalization. In the hospital, before operation, he received thiamine, multivitamins, stool softeners, codeine, and chloral hydrate. With the exception of congenital ocular palsies and the hip fracture, physical examination disclosed no abnormality. Serum sodium, potassium, chloride, and bicarbonate were 135, 3.5, 97, and 27 mEq/l, respectively. Preoperative blood urea nitrogen and glucose levels were 18 and 105 mg/100 ml, respectively. Urine, chest and skull roentgenograms, and EKG were normal.

No preanesthetic medication was given. Anesthesia was induced and maintained with nitrous oxide, 60 per cent in oxygen, and an end-tidal halothane concentration of 0.75 per cent, the latter determined by gas chromatography. The trachea was intubated without the use of any other drug. Neuromuscular function was evaluated by supramaximal stimulation of the ulnar nerve at the wrist, measuring force of adduction of the thumb with a Grass FT-10 force-displacement transducer; results were recorded on a polygraph. A Grass S-H stimulator delivered the stimuli at 0.3 pulse/sec with a 0.1 msec duration. Ventilation was controlled. Thirty-five minutes after induction of anesthesia, pancuronium bromide, 2.4 mg/m² (4.8 mg), was administered intravenously, resulting in 100 per cent depression of twitch height (fig. 1). Approximately 50 minutes later, arterial PO₂, P CO₂, and pH were 123 torr, 28 torr, and 7.55, respectively, with a base excess of +4. Esophageal temperature was 36.2 C. At this time the surgical wound was irrigated with 1 liter of 0.9 per cent sodium chloride containing Polymyxin B, 250,000 units, and bacitracin, 50,000 units.

Because 100 per cent depression of twitch height still existed 103 minutes after administration of pancuronium, and the end of the surgical procedure was anticipated, antagonism of the neuromuscular blockade was attempted by a bolus intravenous injection of pyridostigmine, 14.5 mg, and atropine, 0.6 mg (fig. 1). Because the twitch had returned to only 10 per cent of the con-