CASE REPORTS

Control of Succinylcholine-induced Myotonia by *d*-Tubocurarine

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Succinylcholine can precipitate generalized tonic contraction of skeletal muscles in myotonic patients.[1, 2] Persistent generalized rigidity, if uncontrolled, can result in potentially lethal complications, such as difficulty in ventilating the patient[3] and malignant hyperpyrexia.[4, 5]

Inhalation of halothane has failed to control myotonia following the injection of succinylcholine.[6] Halothane does not modify the neuromuscular effects of succinylcholine.[7] It may even increase muscle contractility by a direct positive inotropic effect.[7, 8] It has also been considered unwise to give the patient an antidepolarizing muscle relaxant.[1] The following report, however, indicates that *d*-tubocurarine can be used safely to control succinylcholine-induced myotonia, thus helping to terminate this life-threatening complication.

REPORT OF A CASE

The patient was a healthy-looking 43-year-old woman. The chest and heart were clinically normal. Arterial blood pressure was 120/80 mm Hg. The patient had a history of hypothyroidism, for which she was receiving thyroid medication. She was scheduled for anteroposterior colporrhaphy.

Premedication consisted of meperidine, 100 mg, and atropine, 0.6 mg, injected intramuscularly 60 minutes before surgery. Infusion of lactated Ringer's solution via a venous cannula was started. Anesthesia was induced with 350 mg thiopental. When the patient was asleep, succinylcholine, 75 mg, was injected intravenously. This was not followed by the usual fasciculations, but the patient developed, within 30 seconds, generalized tonic muscular contractions. Her fists were tightly clenched, her jaw was locked, and her whole body became extremely rigid. Intubation was impossible. However, ventilation with 100 per cent oxygen was possible, using a tightly-fitting face mask. High pressure was necessary to inflate the patient's lungs and to keep her color normal.

After four minutes, there was no change in the degree of rigidity. *d*-Tubocurarine, 15 mg, was injected intravenously, and this was followed immediately by muscular relaxation. Within three minutes, it was possible to intubate the trachea with a cuffed orotracheal tube (No. 9). Anesthesia was maintained with nitrous oxide–oxygen, supplemented with 1 per cent halothane using a Boyle Mark III circuit. Halothane was discontinued after 20 minutes. The patient breathed spontaneously and respiration was assisted throughout the surgical operation, which lasted 55 minutes. The temperature, monitored by an esophageal thermometer, remained 37 C. Recovery was uneventful. No reversal of *d*-tubocurarine block was necessary.

Postoperative neurologic examination disclosed no abnormalities except that myotonia could be demonstrated by tapping the thenar eminences bilaterally.

Electromyography was done. Insertion of the needle evoked a prolonged train of spikes and positive waves, which recurred with the slightest needle movement. The same pattern was elicited by maximum effort on the part of the patient, with waxing and waning of the response in both amplitude and frequency. This EMG pattern was accompanied by an audible dive-bomber effect.

DISCUSSION

Myotonia is characterized by hyperexcitability of skeletal muscles, which respond by repetitive firing of action potentials to either direct or indirect stimulation.[9] The disease is usually observed in patients with the three hereditary muscle disorders that comprise the myotonic syndrome: dystrophia myotonica, myotonia congenita, and paramyotonia[10]; the three are probably manifestations of a single disease. The present patient did not belong in any of the three groups, although a definite myotonic response could be elicited by electromyography. However, she had a history of

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hypothyroidism, which can contribute to a myotonia-like syndrome.[10-14]

Abnormal responses to succinylcholine in myotonia have been observed in animals[15, 16] and in man.[1-3, 6] Succinylcholine depolarizes the endplate, producing an endplate potential which is capable of firing repetitive action potentials associated with tonic contraction of skeletal muscles.[17, 18] It is now accepted that the defect in myotonia is muscular, which may explain why mechanical myotonia elicited by direct stimulation of muscle is completely unaffected by *d*-tubocurarine.[19] On the other hand, myotonia indirectly elicited by nerve impulses, acetylcholine,[19, 20] or succinylcholine[17] can be prevented by prior injection of small doses of *d*-tubocurarine. *d*-Tubocurarine can both prevent and control succinylcholine-induced myotonia, as was shown in the reported case. This could be expected in view of the finding that *d*-tubocurarine not only diminishes endplate depolarization induced by depolarizing agents[21] but also can rapidly repolarize the endplate which has been depolarized by such agents.[22]

d-Tubocurarine does not produce abnormal neuromuscular responses in myotonic patients.[17] It can be used safely to control succinylcholine-induced myotonia, particularly if rigidity is persistent or complicated by difficulty in ventilating the patient or hyperpyrexia, or both.

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