Self-taming of Succinylcholine-induced Fasciculations

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Induction of anesthesia with a thiopental–succinylcholine (SCh) sequence, followed by tracheal intubation, is a commonly used technique. Pretreatment with a subparalytic dose of a nondepolarizing relaxant has been used to eliminate the initial SCh-induced muscle fasciculations. However, the antagonistic effect not only prevents the fasciculations but can also delay the onset of and diminish the SCh-induced block. The present report shows that "self-taming," of SCh-induced muscle fasciculations can be achieved by a small dose of succinylcholine used as a pretreatment before the subsequent full dose needed for laryngoscopy and tracheal intubation.

METHOD

Fifty adult patients undergoing different types of surgical procedures were investigated. Patients were premedicated with meperidine, 50–100 mg, and atropine, 0.6 mg, im. Anesthesia was induced by a slow injection, over 30 seconds, of thiopental, 5 mg/kg, immediately followed by 10 mg succinylcholine. After 45–60 seconds, the full blocking dose of succinylcholine, 1 mg/kg, was injected over a period of 5 seconds. The incidences and intensities of muscle fasciculations following both doses of succinylcholine were carefully evaluated. In all patients, complete muscle relaxation was observed during direct laryngoscopy and tracheal intubation. In ten of the patients, the neuromuscular responses to both the pretreatment dose and full dose of succinylcholine were monitored by stimulating the ulnar nerve with a Block-Aid Monitor and recording the resulting thumb adduction by a Grass force-displacement transducer connected to a Grass polygraph. After recovery of neuromuscular transmission, d-tubocurarine, 3 mg, was injected, followed after 3 minutes by the same total dose of succinylcholine. Each patient acted as his own control.

RESULTS

Fasciculations

The pretreatment dose of succinylcholine, 10 mg, resulted in no visible muscle fasciculation in 40 patients, while minimal localized fasciculations were observed in eight patients, and moderate fasciculations in two patients. The subsequent full dose of succinylcholine (1 mg/kg) was followed by no visible fasciculation in 40 patients, minimal localized fasciculations in seven patients, and moderate fasciculations in three patients (table 1).

Recordings of Neuromuscular Block

In the ten patients whose neuromuscular blocks were evaluated by use of a force-displacement transducer, it was observed (fig. 1) that the duration of succinylcholine-induced block following pretreatment with 3 mg d-tubocurarine was always less than the response following succinylcholine pretreatment.

DISCUSSION

Succinylcholine is a depolarizing agent that acts on the neuromuscular junction to produce initial muscle fasciculations before the onset of block. Ricker and Okamoto have attributed these fasciculations to a prejunctional depolarizing action of succinylcholine. This results in repetitive firing of the motor nerve terminals and antidromic discharges that manifest as uncoordinated muscle fasciculations. This is followed by failure of invasion of the depolarized part of the motor terminals by the nerve action potential, resulting in neuromuscular block.

Pretreatment with a subparalytic dose of nondepolarizing relaxant has been recommended to prevent SCh-induced fasciculations. However, the competitive and antagonistic interaction can delay the onset and decrease the degree of SCh-induced block. Using labelled depolarizing agents, it has been shown that uptake by skeletal muscles can be markedly inhibited by paralytic doses of d-tubocurarine. Adequate block in patients pretreated with nondepolarizing neuromuscular blockers may be restored by increasing the dose of succinyl-

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Accepted for publication November 8, 1976.

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choline by 50–70 per cent. However, the responses of patients to nondepolarizing relaxants show wide variation. This can upset the balance of doses in occasional patients, and predisposes to incomplete SCh-induced block.

Self-taming of SCh-induced muscle fasciculations by pretreatment using a small dose of the same drug (about 0.15 mg/kg) is not associated with paradoxical antagonism, and might be more adequate than pretreatment with nondepolarizers, which antagonize both the fasciculations and the block induced by succinylcholine.

The pretreatment dose of succinylcholine does not produce significant fasciculation, probably because of its small amount. The taming effect of the pretreatment dose may be attributed to induction of neuromuscular desensitization and/or accommodation. The depolarization achieved by the subsequent full dose of succinylcholine may be, therefore, enough to produce neuromuscular block without reaching the threshold necessary for electrical excitation of the nerve terminals and muscle membrane.

Self-taming (despite its complex explanation) is a simple technique that can be applied clinically to minimize the initial SCh-induced muscle fasciculations, while providing optimal neuromuscular block.

REFERENCES