

SUCCINYLCHOLINE NEUROMUSCULAR BLOCK IN THE MYASTHENIA GRAVIS PATIENT

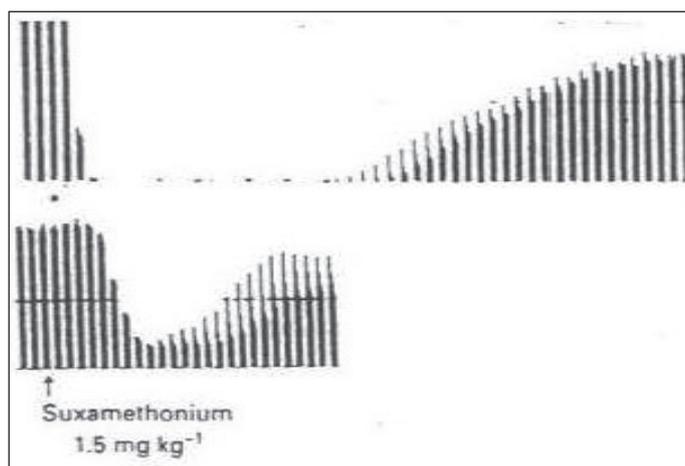
Myasthenia gravis (MG) is an autoimmune disease resulting from the production of antibodies against the acetylcholine receptors of the endplate. These antibodies reduce the number of active receptors, brought about either by functional block of the receptors, by increased rate of receptor degradation, or by complement-mediated lysis^{1,2}.

Under normal conditions, only 25-30% of the endplate receptors (AChR) are required to maintain neuromuscular transmission; the remaining 70-75% of the receptor pool constitutes a "safety margin". In myasthenia gravis, there is a decrease of the functional AChR, with a subsequent decrease of the "safety margin"³.

The decrease of "functional" endplate receptors in MG can decrease the response to the chemical transmitter acetylcholine, as well as to the depolarizing muscle relaxant succinylcholine. In contrast, the decreased "safety margin" results in a marked sensitivity to nondepolarising relaxants. In normal patients, the wide "safety margin" may explain the slow onset of non-depolarizing block, as well as the rapid onset of succinylcholine depolarizing block. In myasthenic patients (Fig. 1), the decreased "safety margin" not only potentiates the neuromuscular block of non-depolarizing neuromuscular blockers, but also speeds its onset of action. In contrast, this can decrease the depolarizing action of succinylcholine, with a subsequent resistance and delayed onset of action⁴.

In normal patients, repeated doses of succinylcholine manifest a progressively diminishing neuromuscular block, secondary to progressive endplate receptor desensitization. In contrast, the administration of successive doses of succinylcholine to the myasthenic patient (Fig. 2) results in a progressive potentiation of its neuromuscular block secondary to gradual desensitization of the endplate receptors and the development of phase two block⁵.

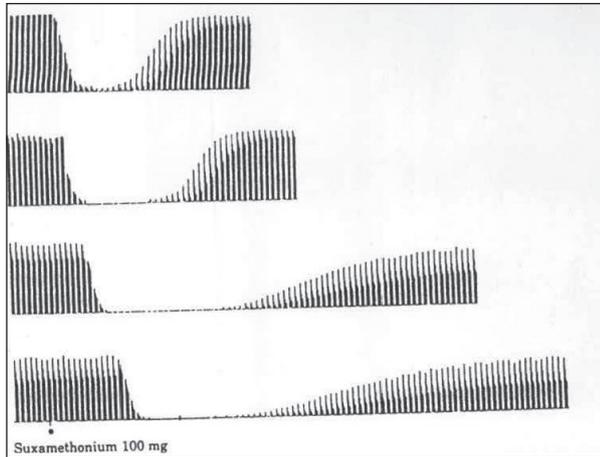
Fig. 1
Electromyographic response to ulnar nerve stimulation. Succinylcholine 1.5 mg.kg⁻¹ resulted in a rapid and complete depolarizing neuromuscular block ($T_4:T_1 > 0.8$) in the normal patient (upper tracing), and resulted in a slower onset of incomplete block ($T_4:T_1 < 0.5$) in the myasthenic patient (lower trace).



Baraka A. Onset of neuromuscular block in myasthenic patients. *Br J Anaesth* 69(2): 227-228, 1992 (with permission).

Fig. 2

Electromyographic response to ulnar nerve stimulation by a train-of-four stimulus every 20s, showing the neuromuscular block of successive doses of succinylcholine 100mg in the myasthenic patient.

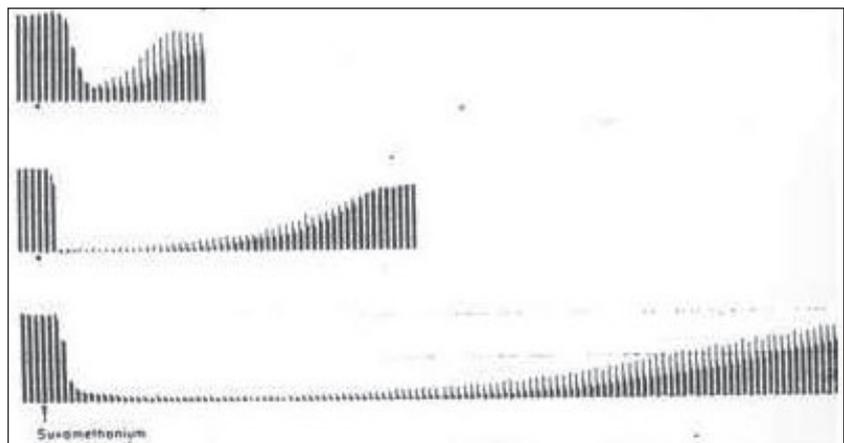


Baraka A, Baroody M, Yazbeck V. Repetitive doses of suxamethonium in the myasthenic patient. *Anaesthesia* 48 (9): 782-83, 1993 (with permission).

The response to succinylcholine in the myasthenic patient is also complicated by the interaction with the preoperative pyridostigmine therapy (Fig. 3). The anticholinesterase action of pyridostigmine does not only inhibit the acetylcholine esterase, but also the plasma cholinesterase with a subsequent delayed hydrolysis of succinylcholine and potentiation of its action. Our previous report⁶ suggests that the response to succinylcholine in the myasthenic patients can

Fig. 3

Electromyographic response to ulnar nerve stimulation by a train-of-four every 20s, showing the effect of succinylcholine 1.5 mg.kg⁻¹. In the upper tracing (no preoperative anticholinesterase therapy), only partial block was achieved. In the middle tracing, (pyridostigmine discontinued the evening of surgery) complete block was achieved and 75% recovery was observed after 16 min. In the lower tracing (pyridostigmine continued till the morning of surgery), 75% recovery was achieved after 35 minutes.



Baraka A. Suxamethonium block in the myasthenic patient – correlation with plasma cholinesterase. *Anesthesia* 47: 217-219, 1992 (with permission).

show marked variations according to the level of their plasma cholinesterase activity. The degree and duration of succinylcholine block in the myasthenic patient receiving pyridostigmine is inversely related to their plasma cholinesterase activity; in patients managed till the morning of surgery by pyridostigmine, succinylcholine administration results in a complete and prolonged neuromuscular block.

In conclusion, the neuromuscular block of succinylcholine in the myasthenic patients can show wide variations. Myasthenic patients untreated with anticholinesterases show resistance to succinylcholine. However, repeated doses of succinylcholine can result in gradual desensitization of the endplate receptors to the chemical transmitter acetylcholine and to the depolarizing action of succinylcholine resulting in a progressive development of prolonged phase II block. In myasthenic patients treated by pyridostigmine which inhibits both acetylcholine esterase and plasma cholinesterase, succinylcholine block is potentiated; the degree of potentiation is inversely related to the plasma cholinesterase activity.

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6. BARAKA A: Suxamethonium block in the myasthenic patient – correlation with plasma cholinesterase. *Anesthesia*; 1992, 47: 217-219.

