EDITORIAL

SUCCINYLCHOLINE-TRIGGERRED "MASSETER SPASM"

- MAY BE A VARIANT NORMAL RESPONSE -

Succinylcholine-triggered "masseter spasm"[1] is a marked increase in tension of the jaw muscles after administration of succinylcholine (Sch). Muscle testing reveals 50% incidence of malignant hyperthermia (MH) in patients who have an episode of succinylcholine-triggered "masseter spasm"[2]. However, succinylcholine-triggered "masseter spasm" may be the normal pharmacologic response of the masseter muscle to succinylcholine [3-5].

A tremendous breakthrough in the understanding of "masseter spasm" is the recent finding that the increased tone in the masseter muscle seen after succinylcholine may be a normal response[3-5]. Correlative study of the physiologic and morphologic characteristics of the masseter muscle of the rat has shown that the masseter muscle does not easily fatigue by tetanic stimulation, and is rich with oxidoreductive enzymatic activity[6]. Such characteristics may explain, in part, the continued contracture of the masseter muscle without fatigue in response to succinylcholine. A study of structure, pattern of innervations, and mechanical properties of vertebrate muscles has shown that succinylcholine-induced contracture may occur in "tonic" muscle fibers of mammals[7]. Thus, succinylcholine-induced contracture may be the normal pharmacologic response of the masseter muscle, similar to the succinylcholine-induced contracture of the extraocular muscles[8].

The masseter muscles contain slow "tonic" fibers that can respond to depolarizing neuromuscular blockers with a contracture. There is a spectrum of responses: a tight jaw that becomes a rigid jaw and then a very rigid (locked) jaw. In more than 80% of patients with isolated succinylcholine-induced trismus but not associated with rigidity of other muscles, or signs of hypermetabolism, it is a variant normal response, similar to the succinylcholine-induced contracture of the extraocular muscles.

The extraocular muscles are "tonic" muscles, and unlike other mammalian striated muscles, they are multiply innervated, and have several neuromuscular junctions along the surface of each muscle cell[9]. Also, in contrast with other muscles, the extraocular muscles contain both mature and immature fetal receptors. That is why succinylcholine, instead of causing a brief contraction followed by paralysis, the drug causes a long-lasting contracture response, associated with an increase of the intra-ocular pressure. The incidence of succinylcholine-induced "masseter spasm" may be as high as 2.6% in children with strabismus[10].

Muscle testing (in-vitro contracture test) in patients who had an episode of "masseter spasm" revealed a 50% incidence of MH susceptibility, and yet very few patients who have MH susceptibility even had an episode of MH.

Succinylcholine-triggered "masseter spasm" should always be presumptive of MH susceptibility until proven otherwise. However, before condemning a patient as MH susceptible, we must exclude other causes of "masseter spasm" such as a variant normal response, a contracture response of a denervated masseter muscle, or the presence of myotonia[11].

Previous reports have shown that succinylcholine-induced muscle contracture can occur in

denervated limb muscles[12]. This has been attributed to denervation supersensitivity to succinylcholine secondary to extrajunctional spread of the endplate receptors over the entire muscle membrane, and to change of the mature receptors into the immature fetal type(up-regulation). A similar response can occur at the denervated masseter and temporalis muscles which are innervated by the mandibular division of the trigeminal nerve. Similar to the denervated limb muscles, Sch-induced contracture of the denervated jaw muscles can be relaxed by a high dose of nondepolarising neuromuscular blocking drug[12-14].

Succinylcholine can also trigger generalized tonic contracture of skeletal muscles including "masseter spasm" in myotonic patients. Myotonia is characterized by hperexitability of skeletal muscles, which respond by repetitive firing of action potentials to either direct or indirect muscle stimulation. The disease is observed in patients with three hereditary muscle disorders that compromise the myotonic syndrome: myoyonia congenita, myotonia dystrophica, and paramyotonia; the three disorders are probably manifestations of a single disease. Also, hypothyroidism can result in a myotonia-like syndrome11. Abnormal response to succinylcholine has been observed in myotonic animals and in man. Sch depolarizes the endplate, producing a long-lasting endplate potential which is capable of firing repetitive action potentials associated with tonic contracture of the myotonic skeletal muscles allover the body. Sch-induced myotonic contractures can be prevented and/or controlled by nondepolarising muscle relaxants. Thus, nondepolarising relaxants have been successfully used to control Sch-induced myotonic contractures[11].

Muscle testing (in-vitro contracture test) in patients who had an episode of "masseter spasm" revealed a 50% incidence of MH susceptibility, and yet very few patients who have MH susceptibility have ever had an episode of MH[15-17].

The pathology of skeletal muscles in MH is restricted to the excitation-contraction coupling and the sarcoplasmic reticulum, while the neuromuscular junction and the contraction elements are normal[18]. Thus, MH contracture is not inhibited by neuromuscular blockers, but by dantrolene which inhibits the action potential-contraction coupling. It may be reasonable to suggest that the "masseter spasm" does not mark MH susceptibility, if it is isolated, not associated with hypermetabolism, as evidenced by increased body temperature, and elevated end-tidal CO_2 associated with decreased SVO_2. Also, if the "masseter spasm" is readily relieved by nondepolarising muscle relaxant. In contrast, succinylcholine-induced "masseter spasm" secondary to other causes such as a normal variant response, myotonia 11 or denervation[12-14] is usually isolated except when associated with myotonia, is not associated with hypermetabolism, and is readily relieved by nondepolarising muscle relaxant.

In conclusion, it can be suggested that an isolated succinylcholine-induced 'masseter spasm", which is not associated with hypermetabolic signs, and is readily relieved by nondepolarising muscle relaxant may not indicate malignant hyperthermia susceptibility.

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