CORRESPONDENCE

MASSETERIC MUSCLE SPASM

Sir,—We read with interest the recent paper by Leary and Ellis investigating the phenomenon which is often described as masseteric muscle spasm, following administration of suxamethonium [1]. Research in this area is very important because suxamethonium, with its unique properties combining, in most cases, a very fast onset of profound paralysis with a rapid termination of its action, continues to be used widely in clinical practice. It is disappointing that the authors have failed to define some terms, leading to confusion as to what they were measuring.

The accepted definitions of the relevant terms are:

Muscle tone—the resistance offered by a muscle during passive joint movements which act to stretch the muscle. It therefore comprises two elements: a passive element resulting from the natural elasticity of the muscle being stretched, and an active resistance to the stretch generated in the monosynaptic stretch reflex arc. This implies that the measurement of muscle tone must be dynamic.

Myotonia—the inability of a muscle to relax after contraction. It is a result of the repetitive firing of muscle fibre action potentials in disease states arising from instability of the plasma membrane of the muscle. This implies that electromyographic evidence of this phenomenon is necessary to be sure that it is occurring.

Spasm—an involuntary tetanic contraction of skeletal muscle generated by neural activity, often as a result of severe pain. This suggests that a paralysed muscle cannot be in spasm.

Contracture—an involuntary contraction or shortening of skeletal muscle induced usually by chemical/pharmacological agents. This is mostly an effect directly on muscle fibres, with no neural involvement.

The authors used a variety of terms, sometimes interchangeably. For example, the “myotonometer” described in the paper is not measuring muscle tone, because this can only be measured as a response to movement; instead, it is measuring some aspect of the contractile force generated during initial depolarization of the muscle fibres. An increase in muscle tone per se would not lead to shortening of the muscle, although the effects of myotonia would. If electromyographic measurements had been combined with this measurement and had shown repetitive firing of the muscle fibres with the increased force, then suxamethonium may have been shown to be producing myotonia in the masseters.

The patient dismissed in the paper as having “no increase in tone” but whose mouth could not be opened, is a most interesting case. In the absence of electromyographic data, the phenomenon is difficult to interpret, but could have been caused by a very large increase in muscle tone. This might be the problem encountered in clinical practice and called masseter spasm.

There have been many explanations for the abnormal responses to suxamethonium. Theories include hydrolysis of the drug before administration because of incorrect storage, and that attempts at intubation have been made prematurely, before the drug has had time to work. It could be argued that the investigation of such phenomena should begin by studying

the response of the masseters to clinical doses of suxamethonium injected over a period of about 1–2 s, as in clinical practice. Ellis and Leary may be observing responses which are not relevant to clinical practice by diluting the drug and injecting it over 5–10 s, especially in the lower dose of 0.25 mg kg\(^{-1}\). Communication of the results of these studies would be facilitated also by adherence of the investigators to conventional definitions and use of the appropriate measurement.

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REFERENCE

INTRATHECAL MORPHINE-6-GLUCURONIDE

Sir,—The paper by Hanna and colleagues [1] raises important issues which need to be addressed.

The dose of intrathecal morphine used was very small. Regular clinical use of intrathecal morphine in cancer patients shows a ratio of 1:25 for intrathecal to oral dose. Appropriate doses for the patients in this study would have been 2.4, 4 and 20 mg, respectively, and not 0.5 mg as used. The dose chosen was confirmed to be non-analgesic by the significant consumption of pethidine.

The results from three patients cannot be used to obtain valid means and standard deviations or used for pharmacokinetic analysis, therefore these interpretations are meaningless. Additionally, the conclusions drawn by the authors are