

## CORRESPONDENCE

more accurately reflect *in vivo* responsiveness; *i.e.*, the individual muscle fiber basis for masseter spasm is not yet explained.

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*In Reply:*—We greatly appreciate the interest of Drs. Melton, Gronert, and Antognini in our study. We do not believe that their findings of no response to halothane in cut masseter muscle bundles is necessarily inconsistent with our own observation of an increased sensitivity to caffeine and halothane in skinned muscle fibers.<sup>1-3</sup> Skinning muscle fibers may permit greater access of caffeine and halothane to the exposed contractile apparatus, which would account for the fact that the ranges of caffeine and halothane threshold concentrations in our study were lower than those found using different skinning and storage methods and different muscles.<sup>1,2</sup> However, the results from skinned masseter muscle were obtained using the same standardized testing methodology, laboratory, technicians, and equipment used to study the vastus muscle. In addition, the loss of low-molecular-weight intracellular proteins from skinned fibers such as ions, nucleotides, lipid derivatives, or ryanodine receptor-protein interactions may lead to different results from those seen with cut bundles.<sup>4</sup>

If we compare the results of Dr. Melton's results with our own, it appears that masseter and vastus sensitivities to halothane and caffeine may depend on modulators of sarcoplasmic reticulum function that are destroyed in skinned fiber (*e.g.*, dihydropyridine receptors from the T tubules) but not in cut muscle bundle preparations. It would have been surprising that all cut masseter muscle bundles contract in response to halothane *in vitro* because volatile anesthetics *in vivo* do not systematically exert sustained increased in masseter muscle tone. Different experimental conditions may reflect different levels of control of electrocontraction coupling, and may account for variations in observed response of masseter muscle *in vitro*. Clearly, additional studies are needed to clarify the role of endogenous modulators and environmental factors in the triggering of a masseter spasm episode.

## Reference

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