reports about *all* cases available so far for analysis. Pending further evidence, the association of fentanyl with MAOIs should not be condemned on the basis of a sole, inconclusive case.

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Paramyotonia and MH

To the Editor:

It is incorrect for Drs. Howell and Douglas to include paramyotonia congenita in "a group of disorders which are called the myotonic dystrophies."[1] While both paramyotonia congenita and myotonic dystrophy cause episodes of myotonia and muscle weakness, only myotonic dystrophy is characterized by progressive muscle wasting and atrophy. Signs and symptoms of paramyotonia congenita are limited to skeletal muscle, but myotonic dystrophy affects all three types of muscle, as well as other organ systems. Finally, paramyotonia congenita and hyperkalaemic periodic paralysis appear to be the same disorder, with a gene locus on chromosome 17 (sodium channel α-subunit).[2] The gene locus for myotonic dystrophy is located on chromosome 19.[3]

Dr. Crone states that malignant hyperthermia (MH) is associated with the myotonias, without citing references. The only myopathy definitely associated with MH is central core disease.[4] No cause-effect relationship has been made between MH and the myotonias. Contracture test results in myotonic patients have been negative or equivocal.[5] Malignant hyperthermia is a disorder of skeletal muscle calcium homeostasis; myotonia congenita is a chloride channel disorder (chromosome 7),[6] and paramyotonia is due to a sodium channel defect;[2] MH and myotonic dystrophy are both characterized by raised intramyoplasmic calcium concentrations, but by two different mechanisms.[5] In MH, a defect in the sarcoplasmic reticulum causes uncontrolled calcium release in the presence of anaesthetic triggers. In myotonic dystrophy, there is an abnormal influx of *extracellular* calcium, leading to muscle damage. In a report by Aldridge of 49 anaesthetics given to 17 myotonic dystrophy patients, there were no cases of MH, despite the frequent use of volatile agents.[7] This is the largest series reported in such patients, as far as I am aware. Newberg *et al.* were unable to trigger MH episodes in myotonic goats, the classic animal model of myotonia congenita.[8]

Hopefully, as we gain more knowledge about the pathophysiology of muscle disorders, a number of misconceptions regarding the association of MH to other myopathies can be put to rest.

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REPLY

We wish to thank Dr. Allen for updating our information with respect to the genetics of the myotonias and hyperkalemic periodic paralysis. At the time that the article was written much of the information that he quotes was unavailable. In fact, the 1992 ASA Refresher Course on MH still listed myotonia congenita as a disease that appears possibly related to MH.