RELATIONSHIP BETWEEN EXERCISE-INDUCED MYOLYSIS AND MALIGNANT HYPERThERMIA

Sir,—We read with interest the article of Hackl and colleagues [1] on the possible relationship between exercise-induced myolysis and malignant hyperthermia. Indeed, “human stress syndrome” should be considered and contracture tests performed when there are important symptoms during exercise.

Our personal experience concerns 11 patients biopsied because of “exercise-induced myolysis”, and tested according to the European Malignant Hyperthermia Group procedure [2]. Only one of them, a 2-yr-old boy presenting with hyperthermia and faintness when exposed to exercise, stress and heat, had a positive contracture test to halothane and caffeine. All the others, aged 8—43 yr had clear-cut negative results. Histopathological and histochemical investigation of the muscle did not reveal any precise mechanism to account for the rhabdomyolytic episode.

We are anxious to know if the authors believe that their patient had a familial genetic defect producing the abnormal contracture tests. If so, have they biopsied other members of the family? Furthermore, have they looked for a metabolic myopathy in the two patients?

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REFERENCES


In short, we agree with Krivosic-Horber and colleagues that MH trait is just one possible trigger for exercise-induced symptoms, myolysis, or both. Considering the limited number of cases, the fact that the MH trait was more common among our patients is probably a coincidence.

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MYOTONIAS AND SUSCEPTIBILITY TO MALIGNANT HYPERThERMIA

Sir,—While the recent paper by Lehmann-Horn and Iaizzo [1] was intriguing, their final sentence is strange: “...non-triggering anaesthesia is indicated.” Their results and discussion strongly suggest that, with the exception of a depolarizing drug such as suxamethonium, the volatile agents are safe in myotonia patients. Suxamethonium is, of course, contraindicated because of the benign contracture it is known to produce, but the likelihood of clinical MH is probably not different from that of the general population.

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REFERENCES


Sir,—Thank you for the opportunity to respond to Dr Gronert’s comment on our paper. In our work we showed that myotonic conditions are not accompanied by a genetic susceptibility to malignant hyperthermia (MH). On the other hand, we found that not only suxamethonium, but also caffeine and halothane, could cause a temporary increase in the baseline force in the in vitro contracture test. This is more likely to be in the form of a tetanic contraction (i.e. associated with electrical activity) than in the form of a contracture. Therefore, the myotonic reaction apparently can be intensified by several substances, in addition to depolarizing substances. That is the reason for our qualifying statement.