CORRESPONDENCE

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Duchenne's Muscular Dystrophy and Malignant Hyperthermia: Another Warning

To the Editor:—Duchenne's muscular dystrophy (DMD) is an x-linked disorder associated with skeletal muscle destruction and cardiac muscle dysfunction. After a progressive period of increasing disability, death usually occurs in the second decade of life. Sudden cardiac arrest in the recovery room and acute exacerbation of the muscle destruction have been reported following anesthesia and surgery.[1,2] A few cases of malignant hyperthermia (MH) have been diagnosed in patients with DMD based on clinical criteria.[3,4] Recently Kelfer *et al.*[5] described a patient with DMD who, after a benign halothane anesthetic, suffered a cardiac arrest in the recovery room. Muscle biopsy investigation, using the technique of calcium uptake into isolated sarcoplasmic reticulum showed that the patient was also susceptible to developing MH.

Brownell *et al.* also recently have reported masseter spasm and ventricular fibrillation in a patient with DMD following succinylcholine.[6] The caffeine contracture test on the biopsied vastus lateralis muscle showed a response indicating MH susceptibility.

We recently have performed biopsies the vastus lateralis muscle of a 4-year-old boy with muscle weakness and histologic evidence consistent with DMD. There was no family history of MH. Femoral nerve block with 1% carbocaine and sedation with diazepam was used to provide anesthesia. We found that the muscle, when tested for the response to halothane responded with 0.75 g contracture to 2% halothane; consistent with susceptibility to MH.[7,8] The response to caffeine was normal, however, we have observed that in approximately 50% of biopsy specimens from MH positives, the response to caffeine is normal, although a response diagnostic of MH is observed with halothane alone.[8]

Malignant hyperthermia susceptibility has been associated with several specific muscle disorders such as central core disease and King Denborough syndrome, as well as nonspecific myopathies.[9] It now seems appropriate to state that patients with DMD are at greater risk for MH compared with the general population. Although not all DMD patients are at risk for MH and certainly not all MH patients are likely to have DMD, nevertheless, precautions appropriate for MH should be considered for DMD patients about to undergo anesthesia.

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Preoperative Nifedipin Treatment and Anesthesia in Patients with Coronary Heart Disease

To the Editor:—In their outstanding review, Reves *et al.*[1] stressed the increasing importance of calcium channel blockers and called our attention to the problems that may occur when patients maintained on these drugs require general anesthesia.

In order to obtain more insight into the interaction

TABLE 1. Hemodynamic Response to Anesthesia and Surgery

Preoperative treatment	N	Before Induction HR	MAP	PCWP	CI	Intubation HR	MAP	PCWP	CI
Nifedipin	13	67 ± 11	87 ± 15	8 ± 3	2.83 ± 0.54	77 ± 16	96 ± 20	9 ± 4	2.61 ± 0.75
Beta-blocker	14	59 ± 10	87 ± 14	10 ± 4	2.82 ± 0.72	82 ± 16	98 ± 22	11 ± 4	2.87 ± 1.0
Nifedipin + beta-blocker	35	61 ± 11	81 ± 12	9 ± 4	2.65 ± 0.43	76 ± 12	95 ± 18	11 ± 5	2.72 ± 0.6
	N	Anesthesia HR	MAP	PCWP	CI	Sternotomy HR	MAP	PCWP	CI
Nifedipin	13	70 ± 15	79† ± 13	11 ± 4	2.38 ± 0.47	62 ± 12	92 ± 14	13 ± 4	2.10 ± 0.42
Beta-blocker	16	65 ± 12	87 ± 10	11 ± 4	2.45 ± 0.53	55 ± 11	99 ± 10	12 ± 4	2.14 ± 0.48
Nifedipin + beta-blocker	35	63 ± 10	80† ± 9	10 ± 4	2.16 ± 0.43	60 ± 10	90 ± 15	11 ± 3	2.12 ± 0.6

HR = heart rate (beats/min); MAP = mean arterial pressure (mmHg); PCWP = pulmonary capillary wedged pressure (mmHg); CI = cardiac index ($l \cdot min^{-1} \cdot m^{-2}$). Values are means ± standard deviation, † = significantly different from the beta-blocker group ($p < 0.05$).

between preoperative long-term calcium channel blockade and anesthesia and intraoperative stress, we analysed retrospectively, 70 consecutive patients (age 56 ± 8) with coronary heart disease and angina pectoris, scheduled for aortocoronary bypass surgery. Sixty-four per cent of the patients had three-vessel disease, 66% were in functional class 3–4, and the preoperative LVEDP was 20 ± 6 mmHg. Preoperatively 13 patients were taking nifedipin, 16 were taking beta-blockers, and 35 were taking both nifedipin and beta-blockers. Two patients were being maintained on verapamil, and four were on long-acting nitrates only. The average nifedipin dose was 44 ± 20 mg daily, and the last dose was given orally 1 h before anesthesia. All other medication was continued up to the night before surgery. For anesthesia, flunitrazepam, fentanyl, pancuronium, and nitrous oxide were used and nitroglycerin added iv when required.

Table 1 shows the hemodynamic data prior to, and 10 min after, induction and the peak response to intubation and sternotomy in the patients on nifedipin and beta-blockers.

Except for lower arterial pressure under anesthesia in the nifedipin groups, the analysis of variance did not reveal any significant differences between the groups. Up to now we have not seen any adverse effects that could be attributed to preoperative nifedipin treatment.

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Methadone Titration to Avoid Excessive Respiratory Depression

To the Editor:—It is unfortunate that for the past several decades the excellent clinical properties of methadone largely have been ignored by American anesthesiologists. In this regard, Gourlay *et al.*[1] are to be commended for their excellent article in which they comprehensively ascertained the pharmacokinetics and pharmacodynamics of this narcotic in the perioperative period. They were able to produce satisfactory analgesia with 20 mg of methadone given upon induction with the analgesic effect lasting well into the postoperative period.

It is the potential for prolonged respiratory depression, however, that deters most anesthesiologists from using this narcotic. While this problem does exist, we believe that the risk has been overstated: in particular, we have found that titrating the drug before induction