citrate) as premedication for surgery procedures. They
demonstrated that administration of oral transmucosal
fentanyl citrate results in rapid increases in plasma fentanyl
concentration, with accompanying dose-related sedation,
anaesthesia, respiratory depression, and common opioid side
effects (including pruritus, nausea, vomiting, and difficulty
with micturition), but without changes in arterial blood
pressure or pulse rate. Smaller doses (0.5–1 mg) of oral
transmucosal fentanyl citrate produced analgesia for short
periods and produced some sedation, with little respira-
tory depression, thereby deserving evaluation as premedici-
ation in patients. Higher doses of oral transmucosal fentanyl
citrate (greater than 2 mg) produced respiratory depression and a higher incidence of side effects.

Stanley et al. also reported that two volunteers swal-
lowed an entire 5-mg lollipop; neither experienced seda-
tion, but both experienced 15–20 min of sleepiness ap-
proximately 60–80 min after oral ingestion. These ob-
servations suggested that, as the lollipop dissolved, some
penetrated through the mucosa of the mouth, pharynx,
and esophagus, reaching the systemic circulation via the
venous drainage and right atrium without passage through
the liver. In contrast, virtually all venous drainage from
the stomach and small intestine passes through the liver
via the portal circulation. The high hepatic clearance may
explain why oral administration of 5 mg fentanyl pro-
duced few opioid-related effects, whereas buccal ligue-
faction produced sedation, analgesia, and respiratory de-
pression. The relative lack of opioid effects in these
two volunteers was noted by our patient as well.

Little information is available about gastrointestinal
absorption of fentanyl, although The Extrapharmaco-
epia states that absorption does occur from the gastrointestinal
tract with rapid onset but short duration of action.

In summary, this case demonstrates oral ingestion of
fentanyl as well as the difficulty of detection of fentanyl
with the usual comprehensive drug screens. Detection and
quantification are now generally performed with gas
chromatography/mass spectroscopy (although radioimmu-
noassays can be used). When fentanyl abuse is sus-
pected, the laboratory should be requested to screen for
it specifically. The treating physician should be particu-
larly alert to the possibility of fentanyl abuse when working
with anesthesia and operating room personnel.

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Isolated Masseter Muscle Spasm and Increased Creatine Kinase without Malignant
Hyperthermia Susceptibility or Other Myopathies

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There is much controversy about the definition, sign-
ificance, management, and family counseling of patients

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seter muscle spasm.

who develop masseter muscle spasm (MMS) after succi-
nylcholine.1–4 The clinician must consider whether MMS
correlates with malignant hyperthermia susceptibility
(MHS) and whether to recommend further investigation
such as a muscle biopsy to evaluate the presence of my-
opathy and/or the halothane–caffeine contracture test to
diagnose MHS. Measurement of creatine kinase (CK)
within 24 h may show extreme elevations, which may be
helpful in the diagnosis of MHS or a myopathy. A previous
report5 documented that CK values of greater than
20,000 IU are highly suggestive of MHS as diagnosed by
the halothane–caffeine contracture test. The only patient
who did not have MHS had a myopathy that was dem-
CASE REPORT

A 5-yr-old child was scheduled for an inguinal hernia repair. The child had had two previous operations (one for strabismus repair) without anesthetic problems. Previous anesthetics had consisted of halothane/\text{\textsubscript{N}_2O}/\text{\textsubscript{O}_2}. Succinylcholine had not been used. The child had no signs or symptoms of muscle disease, and there was no family history of muscle diseases or abnormal reactions to anesthesia. Physical examination was normal.

The child fasted for more than 8 h and was brought to the operating room after receiving an intramuscular injection of meperidine, hydroxyzine, and atropine. Anesthesia was induced with halothane and \text{\textsubscript{N}_2O}. He then was given 1.5 mg/kg succinylcholine and 0.01 mg/kg atropine. Upon attempts at intubation, the jaw muscles were noted to be rigid, and the mouth was opened only with considerable difficulty. No other muscle rigidity was noted. The heart rate was 120 beats/min, and the temperature was 37.5°C. End-tidal CO\(_2\) was not measured. The procedure was cancelled because of the association of MMS with \text{\textsubscript{MMS}}. Arterial blood gas analysis showed a pH of 7.26, P\text{\textsubscript{O}}\(_2\) of 143 mmHg, P\text{\textsubscript{CO}}\(_2\) of 37 mmHg, and base excess of -10.0 mm. The child was treated with 1 mg/kg dantrolene and monitored in the intensive care unit. The initial postoperative CK value was 326 IU (normal 30–170 IU). CK values increased to a maximum of 40,000 IU within 12 h and 35,700 IU within 24 h and slowly decreased over 48 h. Myoglobinuria lasted less than 24 h and was treated with mannitol. The child made an uneventful recovery without manifesting other signs of malignant hyperthermia.

Nine years later, at the age of 14 yr, the child underwent extensive evaluation for neuromuscular disorders. A detailed neurologic examination performed by a neurologist did not show evidence of a myopathy. Electromyogram testing of the lower extremity and paraspinal muscles did not show abnormalities. Baseline CK values measured in both the child and the parents were normal. The halothane–caffeine contracture test to diagnose MHS as well as a routine muscle biopsy were performed at the University of South Florida.

The halothane–caffeine contracture test was performed and interpreted in accordance to standards set by the North American Malignant Hyperthermia Group. A portion of the vastus lateralis muscle was used for testing. Less than 4 h elapsed from excision of the muscle to completion of the test. Anesthesia consisted of sedation with midazolam and fentanyl and a lateral femoral cutaneous and femoral nerve block performed with 1% mepivacaine. Six strips were analyzed for their response to incremental doses of caffeine. Five strips were analyzed for their response to 5% halothane. The contracture response to 5% halothane varied from 0.0 g to 0.35 g; a normal response is ≤0.7 g contracture. All 6 strips tested with caffeine showed normal responses (i.e., caffeine-specific concentration < 4 mM; response to 2 mM caffeine ≤ 0.2 g; 7% of maximum contracture occurred at greater than 2 mM caffeine concentration). The results of the halothane–caffeine contracture test were therefore interpreted as normal. The muscle was sent to the Armed Forces Institute of Pathology for detailed examination. Histologic (hematoxylin and eosin, domon's trichrome, Mason's trichrome, periodic acid Schiff) examination did not show signs of a myopathy (i.e., there was no fiber atrophy or inflammation). Detailed histochemical examination (myofibrillar adenosine triphosphatase, reduced nicotinamide adenine dinucleotide dehydrogenase, non-specific esterase, myoadenylate deaminase) showed a normal distribution of Type I and Type II fibers, a lack of ragged red fibers, a lack of central cores, and no evidence of a myopathy. Biochemical assay of enzyme activity for carnitine palmitoyl transferase deficiency was performed because patients with this myopathy frequently have normal histology. Enzyme activity was normal. The child and family members have continued to do well without signs or symptoms of a myopathy. The child has not received other anesthetics.

DISCUSSION

There have been few children with masster muscle rigidity and extremely elevated CK values who have undergone muscle biopsy testing for MHS. This is the first case documented of a child who had MMS followed by CK values greater than 20,000 IU but who did not show evidence of a myopathy by physical examination, electromyography, or detailed histologic examination or evidence of MHS by the halothane–caffeine contracture test. Rosenberg and Fletcher examined 77 patients who developed MMS after receiving succinylcholine. In 7 patients, CK values were greater than 20,000 IU. Six of these patients had an abnormal halothane–caffeine contracture test, and MHS was therefore diagnosed. The remaining patient, who had a normal contracture test, had Becker's muscular dystrophy demonstrated on histologic examination. The correlation of MHS as diagnosed by muscle biopsy to CK values >20,000 IU was statistically significant at P < 0.05 by the test of significance between two proportions. Indeed, a CK value >20,000 IU was the only predictor of MHS in the study. Dysrhythmias, postoperative fevers, arterial blood gases, myoglobinuria, and histologic examination of the muscle did not correlate with a positive contracture test. The authors concluded that "a CPK of >20,000 IU in the perioperative period is indicative of either MH susceptibility or an underlying myopathy."

There are many possible reasons for the discrepancy between the results in our case and those reported previously. One reason might be that our patient did not meet criteria for inclusion in the previous study. We doubt this, however, because our patient was referred to our muscle biopsy center by a practicing anesthesiologist who clearly documented "jaw muscle rigidity" after succinylcholine administration. This is the same inclusion criteria used in the previous study. Another possible reason is that the muscle biopsy itself was performed differently in our laboratory and could have led to a false negative result. However, the halothane–caffeine contracture test was performed using standardized techniques and criteria for normal versus abnormal results in our laboratory and in Rosenberg and Fletcher's laboratory are similar. More importantly, there has never been a documented false negative halothane contracture muscle test result in any
biopsy center in North America using recommended standards.\textsuperscript{6} It is therefore unlikely that in our case the MHS negative result was incorrect. The incidence of false positive results (specificity) has recently been shown to vary greatly among different biopsy centers.\textsuperscript{7} It is therefore possible that one of the six previously reported\textsuperscript{8} patients with a positive contracture result may not be malignant hyperthermia–susceptible and therefore may be similar to our patient. It is also possible that our patient has an underlying myopathy that was not diagnosed by our investigation. This is unlikely, however, because family history, physical examination, baseline CK value, electromyography, and exhaustive histologic examination all were normal.

The most probable cause of the apparent discrepancy may be statistical. A $P$ value of $< 0.05$ shows only that the two groups are different $95\%$ of the time. Therefore, one might expect, as more patients are tested, to find a normal patient like ours. Indeed, Rosenberg has recently examined a child with MMS and with CK values $> 50,000$ IU within $24$ h who had a normal contracture test and normal histologic examination.\textsuperscript{‡}

We are left to assume that a child for whom MHS is not diagnosed by biopsy and for whom no myopathy is found by histologic examination can have greatly increased CK values after halothane and succinylcholine. In Littleford et al.’s study\textsuperscript{4} on continuing the triggering anesthetic after MMS, some patients’ CK values increased to as high as $138,240$ IU. Although Littleford et al.’s study is controversial, none of those patients developed fulminant malignant hyperthermia. The results of muscle histology or the halothane–caffeine contracture test were not reported.\textsuperscript{4} Increases of CK to as great as $180,000$ IU have been reported in healthy patients after $20$ min of leg exercises.\textsuperscript{8}

In conclusion, we describe a patient who developed isolated MMS and CK values of $> 20,000$ IU with normal muscle by extensive histologic examination and a normal halothane–caffeine contracture test response. The likelihood of MHS or a myopathy in patients who develop MHS after succinylcholine and who have CK values of $> 20,000$ IU is great but not absolute. Patients should be counseled appropriately. Although the contracture test may have false positive results, it should be recommended to such patients, as should neurologic evaluation and muscle biopsy for myopathies.

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Tight Mask Fit Could Have Prevented "Airway" Obstruction

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Recent investigations by March and Crowley\textsuperscript{1} demonstrate the potential inadequacies of anesthesia apparatus checkout procedures despite the use of a Food and Drug Administration (FDA)–recommended checklist.\textsuperscript{‡} Our institution uses an adaptation of this checklist, which is printed on the back of each anesthetic record, and requires a signature by the anesthesiologist (fig. 1). Our

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