Malignant hyperthermia in the oral and maxillofacial surgery patient: an update

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Malignant hyperthermia (MH) is a pharmacogenetic disorder of skeletal muscle that presents as a hypermetabolic response to potent volatile anesthetic gases, such as halothane, sevoflurane, desflurane, the depolarizing muscle relaxant succinylcholine, and, rarely in humans, to stresses, such as vigorous exercise and heat. The syndrome is likely to be fatal if untreated. Early recognition of the signs of MH provides the clinical diagnostic clues. Diagnostic testing relies on assessing the in vitro contracture response of biopsied muscle to halothane, caffeine, and other drugs. Dantrolene sodium is a specific antagonist of the pathophysiologic changes of MH and should be available wherever general anesthesia is administered. The prevention and treatment of acute episodes of this disorder is of paramount importance to the oral and maxillofacial surgeon. The management of such patients in the oral and maxillofacial surgery setting and the recent advances in the field of MH are presented. (Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2011;112:e1-e7)

Malignant hyperthermia (MH) is a pharmacogenetic disorder of skeletal muscle that presents as a hypermetabolic response to potent volatile anesthetic gases, such as halothane, sevoflurane, desflurane, and the depolarizing muscle relaxant succinylcholine, and, rarely in humans, to stresses, such as vigorous exercise and heat. It was first described in 1960. The apparent features of the reactions were dominated by progressive hyperthermia and hence the name MH. The “malignant” part of the name MH has been useful in emphasizing the fatal nature of the condition. Incidences of MH during routine dental and maxillofacial surgery have been reported, sometimes with fatal consequences. At the dental office, an MH-susceptible (MHS) patient exposed to excessive stresses (such as pain, fear, or other triggering agents) may exhibit signs and symptoms of MH. Oral and maxillofacial surgeons who use triggering agents like succinylcholine in their offices must be able to recognize and treat MH. As routine oral and maxillofacial surgery procedures move from the hospital to outpatient facilities where triggering agents are commonly used, the oral and maxillofacial surgeon must be aware of the signs, symptoms, and management of MH to prepare for, and hopefully prevent, an MH episode. The purpose of this article is to review the triggering agents; the incidence of occurrence; and the etiopathogenesis, diagnosis, prevention, and management of an MH crisis.

EPIDEMIOLOGY

The incidence of MH episodes during anesthesia is between 1:5000 and 1:50,000 to 100,000 anesthesias. Even though an MH crisis may develop at first exposure to anesthesia with those agents known to trigger an MH episode, on average, patients require 3 anesthesias before triggering. Reactions develop more frequently in males than females (2:1). All ethnic groups are affected, in all parts of the world. The highest incidence is in young people, with a mean age of all reactions of 18.3 years. It has been found that children younger than 15 years comprised 52.1% of all reactions. Although described in the newborn, the earliest reaction confirmed by testing is 6 months of age. The oldest is 78 years. MH crises develop not only in humans but in
other species, particularly pigs, which have been a valuable source for research. Reactions have also been described in horses, dogs, and other animals. Genetically, MH is an autosomal dominant condition; the estimated prevalence of the genetic abnormalities may be as great as 1 in 3000 individuals (range 1:3000 to 1:8500). Most patients with central core disease (CCD), an inherited myopathy characterized by muscle weakness, are susceptible to MH. Multi-minicore disease also predisposes to episodes of MH.

Etiopathogenesis

Numerous factors could be involved in triggering MH: age, type of anesthetic, environmental temperature, mitigating drugs administered simultaneously, and degree of stress. Symptoms are triggered by the nonhalogenated anesthetic ether, by halogenated anesthetics (halothane, isoflurane, enfurane, desflurane, sevoflurane), and by the depolarizing muscle relaxant succinylcholine. Nitrous oxide and amide local anesthetics also have been implicated in MH. The triggering agents cause an increase in the concentration of free cytoplasmic calcium, which is released from the sarcoplasmic reticulum stores via the muscle ryanodine receptor. The ryanodine receptor (RYR1) gene encodes the key channel that mediates the calcium release in skeletal muscle during excitation-contraction coupling. Mutations in the ryanodine gene account for approximately 50% of the patients who are susceptible to MH. This increase in cytoplasmic concentrations of calcium induces contracture of skeletal muscles (often first noticed in the masseter muscle), activating glycogenolysis and cell metabolism and thereby resulting in heat and excess lactate production. Activation of the oxidative cycle leads to high oxygen consumption and carbon dioxide production, followed by muscular adenosine triphosphate depletion and systemic changes, such as acidosis, hypercapnia, hypoxemia, tachycardia, and hyperthermia. During the course of the crisis, rhabdomyolysis occurs with subsequent creatinine kinase elevation, hyperkalemia potentially leading to cardiac dysrhythmias or even cardiac arrest, and myoglobinuria with the possibility of renal failure. Additional life-threatening complications include disseminated intravascular coagulation (DIC), congestive heart failure, bowel ischemia, and compartment syndrome of the limbs secondary to profound muscle swelling, and renal failure from rhabdomyolysis. Indeed, when body temperature exceeds approximately 41°C, DIC is the usual cause of death.

Differential diagnosis

A variety of unusual conditions may resemble MH during anesthesia. These include sepsis, thyroid storm, pheochromocytoma, and iatrogenic overheating. Outside the operating room, an MH-like syndrome may occur following injection of ionic contrast media into the cerebrospinal fluid, cocaine overdose, and the malignant neuroleptic syndrome.

Laboratory diagnosis

The “gold standard” for diagnosis of MH is currently the in vitro contracture test (IVCT), which is based on contracture of muscle fibers in the presence of halothane or caffeine. A normal MH diagnosis (MHN) is obtained when both tests are negative. A third diagnosis, MH equivocal (MHE), is obtained when only one of the halothane or caffeine tests is positive. Recently, the use of ryanodine (which binds selectively to the calcium release channel) or 4-chloro-m-cresol as alternatives to halothane has shown promising results. IVCT is expensive, confined to specialized testing centers, requires a surgical procedure, and can yield equivocal as well as false positive and negative results. DNA analysis, however, offers an alternative to the IVCT, requiring only a blood specimen, which can be sent to an accredited diagnostic laboratory. DNA testing for MH was first suggested in 1990, when a mutation within the ryanodine receptor gene (RYR1) encoding the skeletal muscle calcium release channel was identified. Since then, about 50% of MHs have been linked to RYR1, with more than 100 mutations associated with MH identified within this gene. However, because of the heterogeneity of the disorder, as well as discordance within families, a negative DNA result cannot be used to rule out MH susceptibility. A variety of minimally invasive diagnostic tests are in development at present. One uses nuclear magnetic resonance spectroscopy to evaluate ATP depletion during graded exercise in vivo. MH patients have a greater breakdown of ATP and creatine phosphate, as well as an increase in acid content compared with controls.

Management

Acute MH crisis

The following MH protocol sequence has been modified from the recommendations of the Malignant Hyperthermia Association of the United States [MHAUS] and would be appropriate should an acute MH crisis occur in the ambulatory oral and maxillofacial surgery center (Table 1).

Preventive measures

Preventive measures include a thorough anesthetic history to determine the possibility of the patient or a family member having experienced an MH episode. When suspicion of MH exists, family members should not be given trigger anesthetic agents, i.e., potent volatile anesthetic agents, such as halothane, sevoflurane, desflurane, enfurane, isoflurane, or succinylcholine, and testing is recommended. Patients with any form of myotonia should not
Table I. Management of an acute MH crisis in the ambulatory oral and maxillofacial center

1. First, recognize the signs of an MH episode (Table II).
2. Stop all triggering agents, call for assistance, and obtain dantrolene.
3. Hyperventilate with 100% oxygen at a flow of more than 10 L/min.
4. Halt the procedure as soon as possible; if emergent, continue with nontriggering anesthetic technique. If needed, deepen anesthesia with opioids, benzodiazepines, barbiturates, or propofol. Do not waste time changing the circle system and CO2 absorbent.
5. Place an endotracheal tube (if one is not in place already) as soon as possible.
6. Give an initial dose of dantrolene 2.5 mg/kg intravenously through large bore cannula. Dantrolene 2.5 mg/kg is repeated every 3-5 minutes until there is a fall in heart rate, normal cardiac rhythm, reduction in muscle tone, and decline in body temperature. Sometimes, more than 10 mg/kg (up to 30 mg/kg may be required).
7. Acidosis is treated with 1-2 mEq/kg of sodium bicarbonate in the absence of blood gas analysis.
8. Monitor temperature and cool the patient with core body temperature <39°C with blankets, ice packs on the axilla, chest, back, and groin. Lavage open body cavities like stomach, bladder, or rectum, if possible. Infuse cold saline intravenously and stop cooling if temperature <38°C.
9. Dysrhythmias usually respond to treatment of acidosis and hyperkalemia. Use standard drug therapy (beta blocker, esmolol 0.25 mg/kg IV, or lidocaine 1 mg/kg IV) except calcium channel blockers, which may cause hyperkalemia or cardiac arrest in the presence of dantrolene.
10. Hyperkalemia—Treat with hyperventilation, bicarbonate 1-2 mEq/kg IV. For pediatric, 0.1 U insulin/kg and 1 mL/kg 50% glucose; for adult, 10 units regulator insulin IV and 50 mL 50% glucose and calcium chloride 10 mg/kg or calcium gluconate 10-50 mg/kg for life-threatening hyperkalemia. Check glucose levels hourly.
11. Transport to the emergency department for admission to intensive care unit. Follow ETCO2, electrolytes, blood gases, CK, core temperature, urine output and color, coagulation studies. If CK and/or K+ rise more than transiently or urine output falls to less than 0.5 mL/kg/h, induce diuresis to >1 mL/kg/h and give bicarbonate to alkalize urine to prevent myoglobinuria-induced renal failure. Venous blood gas (e.g., femoral vein) values may document hypermetabolism better than arterial values. Central venous or PA monitoring as needed and record minute ventilation. Place Foley catheter and monitor urine output.

The postacute phase treatment of an MH crisis should include the following:
1. Observation in an intensive care unit for at least 24 h because of the risk of recrudescence.
2. Dantrolene 1 mg/kg every 4-6 h or 0.25 mg/kg/h infusion for 24 h. Further doses if needed.
3. Frequent checks on vitals and laboratory values, especially arterial blood gases. CK every 8-12 h, less often as values trend downward.
4. Follow urinary myoglobin and institute therapy to prevent myoglobin precipitation in renal tubules and the subsequent development of acute renal failure. CK levels above 10,000 IU/L are a presumptive sign of rhabdomyolysis and myoglobinuria. Follow standard intensive care therapy for acute rhabdomyolysis and myoglobinuria (urine output >2 mL/kg/h by hydration and diuretics along with alkalinization of urine with Na-bicarbonate infusion with careful attention to both urine and serum pH values).
5. Counsel the patient and family regarding MH and further precautions; refer them to MHAUS. Fill out and send in the AMRA form (http://www.mhreg.org) and send a letter to the patient and her/his physician. Refer patient to the nearest biopsy center for follow-up.

AMRA, Adverse Metabolic or Muscular Reaction to Anesthesia; CK, creatine kinase; IV, intravenous; MH, malignant hyperthermia; MHAUS, Malignant Hyperthermia Association of the United States.

Table II. Signs of malignant hyperthermia
- Increasing ETCO2
- Trunk or total body rigidity
- Masseter spasm or trismus
- Tachycardia/tachypnea
- Mixed respiratory and metabolic acidosis
- Increased temperature (may be late sign)
- Myoglobinuria

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receive succinylcholine. Patients with hypokalemic periodic paralysis, CCD, Duchenne or Becker muscular dystrophy, paramyotonia, or myotonia fluctuans should not receive trigger agents. All patients receiving more than a brief general anesthetic should have their core temperature monitored. Young patients (younger than 12 approximately) should not receive succinylcholine for elective procedures, so as to avoid the possibility of hyperkalemia response in a patient with undiagnosed muscular dystrophy. Patients who are MH susceptible should be cautioned regarding the remote, but conceivable possibility of heat stroke in environments in which exposure to high heat and humidity is possible.

Management of the MH-susceptible patient for anesthesia

The MHAUS recommendation states that intravenous conscious sedation with local anesthesia may be safely administered to the MHS patient in the office setting for such procedures as the surgical removal of impacted third molars. Deep sedation or general anesthesia may also be used (obviously so long as no triggering agents are administered) (Table III). Because laryngospasm may occur in the unconscious patient, and succinylcholine is contraindicated in MHS patients, a nondepolarizing muscle relaxant must be available. More rigorous standards are recommended if intubation anesthesia or a prolonged procedure under general anesthesia is anticipated (Table IV). Once the patient has undergone such an anesthetic without incident, he...
or she may be treated similar to any other patients. Pretreatment with dantrolene is not necessary.

**DISCUSSION**

Even with proper management of MH, mortality is reported at 5% to 10%.10 If MH triggers could be removed from the office, patients would not be at risk of developing MH. Barbiturates, benzodiazepines, narcotics, nitrous oxide, propofol, and local anesthetics can be used instead of the inhalation anesthetics that trigger MH. Triggering anesthetics can be eliminated from the oral and maxillofacial surgeon’s office. However, succinylcholine remains the best drug for emergency management of laryngospasm and rapid-sequence endotracheal intubation.21 Because succinylcholine can trigger an MH crisis, dantrolene should be available for acute management of MH. Dantrolene is a hydantoin derivative that inhibits calcium release from the sarcoplasmic reticulum without influencing its reuptake. Reconstituting dantrolene is time-consuming and cumbersome, and calling for help immediately is crucial. Dantrolene is stored in 20-mg lyophilized aliquots with mannitol and must be reconstituted with 60 mL of sterile water (without a bacteriostatic agent) per vial. Warming the diluent speeds dissolving the dantrolene. Dantrolene cannot be mixed with normal saline or lactated Ringer’s, as it precipitates in these solutions. The recommended dose is 2 to 3 mg/kg every 5 minutes, up to a total dose of 10 mg/kg if needed. Therefore, every operatory where general anesthesia is being used must stock at least 36 vials of dantrolene sodium, which is roughly equivalent to 10 mg/kg in an adult patient.20 The reconstituted drug must be used in 6 hours. Although dantrolene has been life saving, it is not a perfect drug. Drawbacks include phlebitis, respiratory muscle weakness with difficulty weaning from a ventilator, difficulty with solvation, and others. Given the time-consuming process of mixing the initial dose of 9 vials of dantrolene, each with 60 mL of sterile water (total of 540 mL), for a 70-kg patient, it may be a good idea to stock the initial intravenous bolus dose (9-12 vials) in the office and identifying a hospital with an emergency department that has dantrolene available. It is unlikely that the oral and maxillofacial surgeon managing an MH crisis will have sufficient time to administer more than the first dose of dantrolene while managing the acidosis, hypercarbia, hyperkalemia, dysrhythmias, and so on. After calling for medical assistance and initiating the treatment of MH, it would be best to transport the patient to an emergency department that has dantrolene available and can obtain blood gases, electrolytes, and laboratory studies needed to guide management of the crisis. The patient with a severe MH crisis will need to be admitted to the intensive care unit for monitoring and continued administration of dantrolene. If the outpatient office is located in a remote area or an emergency department with dantrolene is not near the office, it will be necessary to stock the entire 36 vials.

Azumolene, a 30-fold more water-soluble analogue of dantrolene, also works to decrease the release of intracellular calcium by its action on the ryanodine receptor.21 In MHS swine, azumolene was found to be as potent as dantrolene.22 It has yet to be studied in vivo.

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**Table III.** The safety of selected drugs in patients susceptible to malignant hyperthermia

<table>
<thead>
<tr>
<th>Do not use (trigger agents)</th>
<th>Cyclopropane</th>
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<tbody>
<tr>
<td>Depolarizing muscle relaxants (e.g., succinylcholine)</td>
<td>d-Tubocurarine</td>
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<tr>
<td>Ether</td>
<td>Methoxyflurane</td>
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<tr>
<td>Rapid intravenous potassium</td>
<td>Volatile inhalational anesthetics (desflurane, enfurane, halothane, isoflurane, sevoflurane)</td>
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<tr>
<td>Use cautiously</td>
<td>Catecholamines</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>Phenothiazines (chlorpromazine, prochlorperazine)</td>
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<tr>
<td>Safe to use</td>
<td>Anticholinergics</td>
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<td>Anticholinesterases</td>
<td>Anticholinesterases</td>
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<tr>
<td>Barbiturates</td>
<td>Benzodiazepines</td>
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<td>Calcium</td>
<td>Droperidol</td>
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<td>Droperidol</td>
<td>Etomidate</td>
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<td>Ketamine</td>
<td>Local anesthetics</td>
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<td>Narcotics</td>
<td>Nitrous oxide</td>
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<tr>
<td>Nitrous oxide</td>
<td>Nondepolarizing muscle relaxants</td>
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<tr>
<td>Nonsteroidal anti-inflammatory drugs</td>
<td>Propofol</td>
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*Adapted from Wappler F. Malignant hyperthermia. Eur J Anaesthesiol 2001;18:632.*

**Table IV.** Management of an MH-susceptible patient for surgery under intubation/general anesthesia

1. Avoid the use of MH-triggering drugs. The anesthesia machine should be prepared by flowing 100% oxygen through the machine at 10 L/min for at least 20 minutes. The ventilator should also be included in purging the machine by cycling the ventilator at the time of the oxygen flow. Vaporizers should be disabled, drained, or removed if possible.
2. Continuously monitor the patient’s exhaled CO2 concentration.
3. Use a continuous temperature monitor (e.g., in the nasopharynx, axilla, esophagus, or rectum).
4. Have an MH cart in the operating room, stocked with adequate supplies of dantrolene sodium.

MH, malignant hyperthermia.
in humans, but may present a suitable alternative to dantrolene in the treatment of MH.

Until a nondepolarizing muscle relaxant with properties similar to succinylcholine is available, it is difficult to justify the use of nondepolarizing agents for emergency situations. Further studies are needed to determine if low nonparalyzing doses of nondepolarizing muscle relaxants like rocuronium can be used to reverse laryngospasm. Until all triggering agents can be removed from the outpatient surgical facility, it is recommended that the oral and maxillofacial surgeon identify an emergency room that has dantrolene available and have a minimum of 10 to 12 vials of dantrolene at the outpatient facility.\textsuperscript{21}

Increase in masticatory muscle tension following succinylcholine administration may provide the first sign that an individual may be susceptible to MH in the absence of a positive family history.\textsuperscript{23} Doubts were raised as to the validity of masseter muscle spasm as a reliable indicator of MH susceptibility\textsuperscript{24}; however, this doubt was based on a false premise.\textsuperscript{25} Further illustration of the need to consider a patient developing masseter muscle spasm following succinylcholine as susceptible to MH was presented by Ramirez et al.,\textsuperscript{26} where they reported masticatory muscle rigidity in a trauma patient leading to difficulty in tracheal intubation following succinylcholine use. Subsequently, the patient developed metabolic features of MH when isoflurane was used for fixation of a humerus fracture.\textsuperscript{26}

Severe or untreated cases of acute rhabdomyolysis may result in life-threatening hyperkalemia, myoglobinuria, renal failure, and multiorgan system failure.\textsuperscript{27} Schenk et al.\textsuperscript{28} consider early continuous venovenous hemofiltration (CVVH) as a valuable therapeutic option in the management of severe rhabdomyolysis. However, the potential benefits of early CVVH on outcome of acute myoglobinuric renal failure remain to be established.

A review of the literature reveals several cases of MH related to dental treatment.\textsuperscript{4,5,29,30} Nitrous oxide was implicated as the triggering agent in a 1985 report.\textsuperscript{12} Although some believe nitrous oxide to be a trigger of MH, its frequent use as an anesthetic agent with susceptible patients would seem to discount this theory.\textsuperscript{13} The MHAUS considers its use safe in MHS patients (Table III). Contraindication for the use of amide local anesthetics is based on in vitro muscle contracture studies.\textsuperscript{11} Amide anesthetics have been demonstrated to cause muscle contraction, whereas ester anesthetics caused relaxation of muscle tissue, which inhibits contracture.\textsuperscript{11} Reviews of the literature have not demonstrated a clear link between amide anesthetics and MH and it generally is accepted that amide anesthetics are safe for MHS patients.\textsuperscript{11,31,32}

Choung\textsuperscript{33} described a postoperative variant of MH, termed human stress syndrome, in a 17-year-old boy with no significant history who underwent orthognathic surgery. Characterized by more subtle manifestations of MH without any extreme elevation in body temperature, human stress syndrome can result from exposure to physical or emotional stress outside a medical setting.\textsuperscript{33}

Molecular genetic diagnostics hold great promise for a noninvasive diagnostic test that is highly reproducible and requires minimal biological material. As mutations in the gene encoding the calcium-release gene in skeletal muscle have been described as causal for MH, guidelines have been developed for clinical molecular testing.\textsuperscript{1}

In vitro contracture testing to diagnose MH susceptibility requires a muscle biopsy, which may be associated with severe side effects for the patient. The caffeine-halothane contracture test requires a fresh muscle sample that must be obtained immediately before testing and therefore in close proximity to the biopsy center. The North American protocol\textsuperscript{34} has sensitivity and a specificity of 97% to 98% and 78% to 80%, respectively (although specific values will vary from laboratory to laboratory). Thus, there is a 2% chance that contracture testing will incorrectly mislabel an MHS individual as normal, whereas there is a 20% chance that a person without the disease will be labeled as susceptible.

There have been several attempts at developing a less invasive test. Schuster et al.\textsuperscript{35} investigated a metabolic test that involves intramuscular injection of caffeine and halothane, and subsequent measurement of interstitial lactate by microdialysis to differentiate between MHS and MH non-susceptible (MHN) individuals. In contrast to genetic analysis, because of its functional characteristics, the test is independent of the locus of an MH-causative mutation. With genetic analysis, more than 150 MH-associated mutations were described and 28 MH-causative mutations cover 30% to 50% of MH families.\textsuperscript{36} For epidemiologic and economic reasons, primary genetic screening in any MH suspect seems likely to be impossible. McKinney et al.\textsuperscript{37} indicated the feasibility of using calcium release as an assay for RyR1 function in human and porcine B-lymphocytes. Bina et al.\textsuperscript{38} successfully investigated a blood test approach for malignant hyperthermia testing in a swine model by demonstrating that 4-chloro-m-cresol stimulation of porcine lymphocytes induced increased adenosine formation in MHS cells relative to those from normal swine. However, evaluation of adenosine formation in response to RyR1 agonists in human lymphocytes is needed. Wehner et al.\textsuperscript{39} observed that in myotubes derived from MH patients with identified mutations in RyR1, calcium release induced by 4-CmC or caffeine was increased compared with controls. The increased calcium response in myoblasts segregated well with the MH phenotype. However, this assay, although promising, still
requires surgical biopsy and myotube culture and thus is not a significant improvement over the current Caffeine Halothane Contracture Test.

Although research has focused on the ryanodine receptor, other defects have been described. For example, the CACNL1A3 gene that encodes the α-1 subunit of the skeletal muscle dihydropyridine receptor has been implicated to play a causative role. In addition, calsequestrin-1 is a calcium-binding protein in skeletal muscle that, when absent, leads to MH-like episodes in mice. Whether this defect is present in humans remains to be determined, but such a defect could explain susceptibility to MH in patients who do not have abnormal ryanodine receptors. Interestingly, Capacchione et al. reported a patient with exertional rhabdomyolysis and malignant hyperthermia in a rare patient with ryanodine receptor type 1 gene, L-type calcium channel α-1 subunit gene, and calsequestrin-1 gene polymorphisms.

Whenever possible, nontriggers agents should be considered for outpatient procedures to help avoid or minimize MH cases. For instance, one study used only the nontriggering agents propofol and vecuronium (nondepolarizing muscle relaxant), and reported no malignant hyperthermia in 23,000 office-based plastic surgery procedures performed under general anesthesia. In addition, multiple studies have demonstrated the safety of intravenous conscious sedation. A balanced anesthetic technique using thiopental, nitrous oxide, and an opioid supplemented by a nondepolarizing muscle relaxant is typically suggested for the MH-susceptible patient. However, malignant hyperthermia can also be triggered by nontriggering agents in fewer than 1% of susceptible patients. Therefore, continued and reasonable use of general anesthesia, especially with triggering agents, in office settings and ambulatory surgical centers mandates active malignant hyperthermia protocols.

In wake of the possibility of an MH attack, temperature monitoring is currently considered a standard for patients undergoing general anesthesia, although very brief procedures (e.g., 15 minutes) may be an exception. Disposable thermocouple and thermistor probes are available for monitoring core temperature. Preferred sites include tympanic membrane, esophagus, nasopharynx, and rectum. These sites constitute anatomical areas of highly perfused tissues, the temperature of which is uniform and high in comparison with the rest of the body. Skin surface monitoring is not considered accurate or reliable because greater fluctuations occur and it is less reflective of core temperature.

The MHAUS and the Ambulatory Surgery Foundation (ASF) of the United States have jointly issued guidelines for the transfer of care with regard to the development of MH in suspected MH patients from ambulatory centers to a hospital with all the necessary equipment and expertise to treat such an emergency. The patient must be shifted with a report data indicating the cardiovascular signs, temperature and site, minute ventilation with end-tidal CO₂, electrolytes (if available), intravenous site, amount of dantrolene administered and response, presence of muscular rigidity, presence of urinary catheter, and color of urine. The recommendations suggest that the patient be shifted when thought to be stable, which can be recognized by decreasing ETCO₂, decreasing or stable heart rate, declining temperature, dantrolene administration has begun, and generalized muscle rigidity if present is declining. The anesthetist or surgeon transferring care of the patient must have direct telephonic conversation with the physician/anaesthesit accepting care of the patient at the receiving health care facility.

CONCLUSIONS

Unless procedures involve the use of topical or local anesthetics, MHS individuals are not candidates for office-based surgery at the average oral and maxillofacial clinic. Anyone identified for susceptibility to triggering agents should be referred to an accredited ambulatory surgical center or hospital for surgery. Even without a positive history of MH, any surgical facility using potent inhalation anesthetics or succinylcholine must be prepared to treat MH.

REFERENCES


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