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### Malignant Hyperthermia

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# Malignant Hyperthermia

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## Introduction

Complications related to the anesthetic process are multifaceted and abundant. As a future nurse anesthesia student, the underlying pathophysiology, diagnosis, and treatment of anesthesia-related complications are of particular interest. One such anesthesia-induced life-threatening metabolic process involves the hypermetabolism of skeletal muscle. This pharmacogenetic process, known as malignant hyperthermia (MH), has a variable incidence rate ranging from 1:10,000 to 1: 250,000 anesthetic cases. However, the prevalence of the genetic abnormalities may be as great as one in 400 individuals (Rosenberg, Pollock, Schiemann, Bulger, & Stowell, 2015, p. 1). “Malignant hyperthermia (MH) is a rare, but life-threatening, autosomal-dominant inherited disorder that may lead to metabolic crisis of skeletal muscle in susceptible individuals following exposure to triggering agents, such as volatile anesthetics or depolarizing muscle relaxants” (Schneiderbanger, Johannsen, Roewer, & Schuster, 2014, p. 355).

## Significance of Pathophysiology

According to Dirksen et al. (2013), the MH response spurs a cascade of reactions, including increased sympathetic activity, increased production of carbon dioxide, heat from rapid use of adenosine triphosphate (ATP), increased oxygen consumption, excess lactate production, and cellular damage and destruction. The cellular membrane disruption that occurs with MH has multiple consequences, including potassium, phosphate, magnesium, and myoglobin leakage into the extracellular fluid, causing a rise in serum levels (Klingler et al., 2014). Complications related to the progression of these cellular alterations include: skeletal muscle damage, hyperthermia, renal failure, cardiac arrest, and possible death (Dirksen et al., 2013).

## Underlying Pathophysiology

The pathophysiologic process initiated by these pharmacologic insults is characterized by a disruption in the regulation of calcium within muscle cells. This buildup of intracellular calcium results in the initiation of an immense hypermetabolic reaction stimulating widespread muscle contraction (McCance & Heuther, 2014, p. 501). This buildup of intracellular calcium is a result of “uncontrolled sarcoplasmic calcium release caused by mutations in the ryanodine receptor subtype 1 (RYR1)” (Schuster, Johannsen, Schneiderbanger, & Roewer, 2013). This gene encodes an ion channel in the skeletal muscle cell through which calcium flows. “In the presence of an abnormal RYR1 gene in MH-susceptible individuals, a triggering agent such as halothane, isoflurane, sevoflurane, desflurane, or enflurane, either alone or in combination with the depolarizing muscle relaxant succinylcholine, initiates uncontrolled calcium release. This sets off the classic actin-myosin troponin interaction, shortening of muscle fibers, and consequent muscle contraction. The uncontrolled rise in intracellular calcium causes a sustained state of muscle contraction, leading to the hypermetabolic MH response” (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013, p. 331).

## Signs & Symptoms

“Almost all patients who are MH susceptible have no phenotypic changes without anesthesia, it is impossible to diagnose susceptibility without either exposure to the “trigger” anesthetics or by specific diagnostic testing” (Rosenberg et al., 2015, p. 2). This reinforces the importance of educating anesthesia providers, and nurses alike on the signs and symptoms of this pharmacogenetic process. “Characteristic clinical signs of MH during general anesthesia include hypoxemia, hypercapnia, tachycardia, muscular rigidity, acidosis, hyperkalemia and hyperthermia” (Schuster et al., 2013, p. 1). The “malignant hyperthermia clinical grading scale” bases the level of severity of MH crisis on six indicators: rigidity, muscle breakdown, respiratory acidosis, temperature increase, cardiac involvement, and other indicators of metabolic derangement not part of a single process (Heytens, Forget, Scholtes, & Veyckemans, 2015, p. 506). “An increase in end-tidal carbon dioxide despite increased minute ventilation provides an early diagnostic clue” (Rosenberg et al., 2015, p. 1).

## Implications for Nursing Care

“Many clinicians are unprepared to manage an MH crisis in the perioperative setting because it requires the use of low-frequency, high-risk skills and procedures” (Cain, Riess, Gettrust, & Novalija, 2014, p. 301). Nurses should familiarize themselves with the signs and symptoms of MH, and be ready to implement their institutions treatment plan in the event of this complication. The widely accepted treatment for MH is to first remove the triggering anesthetic agent when an impeding MH crisis is expected (Cain et al., 2014, p. 302). Dantrolene is a commonly used medication in the treatment of MH, which directly interferes with skeletal muscle contractions by decreasing the level of calcium in the muscle cells. Dantrolene does not block neuromuscular transmission, but rather potentiates nondepolarizing neuromuscular blockade (Brandom, & Riaz, 2015, p. 17). Active cooling in patients suffering from a MH crisis is paramount, with ice being the most widely used surface coolant. The patient’s core temperature should be lower than 39 degrees Celsius (Cain et al., 2014, p. 302).

<b>1</b> Recognition	<ul style="list-style-type: none"><li>• Unexplained increase in ETCO<sub>2</sub> <b>AND</b></li><li>• Unexplained tachycardia <b>AND</b></li><li>• Unexplained increase in oxygen requirement (Previous uneventful anaesthesia does <b>not</b> rule out MH)</li><li>• Temperature changes are a late sign</li></ul>	
<b>2</b> Immediate management	<ul style="list-style-type: none"><li>• <b>STOP</b> all trigger agents</li><li>• <b>CALL FOR HELP.</b> Allocate specific tasks (action plan in MH kit)</li><li>• Install clean breathing system and <b>HYPERVENTILATE</b> with <b>100% O<sub>2</sub> high flow</b></li><li>• Maintain anaesthesia with intravenous agent</li><li>• <b>ABANDON/FINISH</b> surgery as soon as possible</li><li>• Muscle relaxation with non-depolarising neuromuscular blocking drug</li></ul>	
<b>3</b> Monitoring & treatment	<ul style="list-style-type: none"><li>• Give <b>dantrolene</b></li><li>• Initiate active <b>cooling</b> avoiding vasoconstriction</li><li>• <b>TREAT:</b><ul style="list-style-type: none"><li>• <b>Hyperkalaemia:</b> calcium chloride, glucose/insulin, NaHCO<sub>3</sub><sup>-</sup></li><li>• <b>Arrhythmias:</b> magnesium/amiodarone/metoprolol <b>AVOID</b> calcium channel blockers - interaction with dantrolene</li><li>• <b>Metabolic acidosis:</b> hyperventilate, NaHCO<sub>3</sub><sup>-</sup></li><li>• <b>Myoglobinaemia:</b> forced alkaline diuresis (mannitol/furosemide + NaHCO<sub>3</sub><sup>-</sup>); may require renal replacement therapy later</li><li>• <b>DIC:</b> FFP, cryoprecipitate, platelets</li></ul></li><li>• Check plasma CK as soon as able</li></ul>	<p><b>DANTROLENE</b> 2.5mg/kg immediate iv bolus. Repeat 1mg/kg boluses as required to max 10mg/kg</p> <p><b>For a 70kg adult</b></p> <ul style="list-style-type: none"><li>• <b>Initial bolus: 9 vials dantrolene</b> 20mg (each vial mixed with 60ml sterile water)</li><li>• Further boluses of 4 vials dantrolene 20mg repeated up to 7 times.</li></ul> <p><b>Continuous monitoring</b> Core &amp; peripheral temperature ETCO<sub>2</sub> SpO<sub>2</sub> ECG Invasive blood pressure CVP</p> <p><b>Repeated bloods</b> ABG U&amp;Es (potassium) FBC (haematocrit/platelets) Coagulation</p>
<b>4</b> Follow-up	<ul style="list-style-type: none"><li>• Continue monitoring on ICU, repeat dantrolene as necessary</li><li>• Monitor for acute kidney injury and compartment syndrome</li><li>• Repeat CK</li><li>• Consider alternative diagnoses (sepsis, phaeochromocytoma, thyroid storm, myopathy)</li><li>• Counsel patient &amp; family members</li><li>• Refer to MH unit (see contact details below)</li></ul>	

## Conclusion

MH is a rare, but life-threatening complication. Many clinicians may find themselves unprepared to manage a patient in a MH crisis. Hospitals should encourage their anesthesia providers to take part in routine clinical simulations to be better prepared to react promptly and efficiently. The research surrounding MH is ongoing, and complicated due to the rarity of these events. The cessation of previously known triggering agents, such as halothane, has substantially decreased the incidence of MH in the last 20 years (Heytens, Forget, Scholtes, & Veyckemans, 2015, p. 508). However, the need for research and development of treatment plans for this complication is vital to the continued decline in MH crisis events.

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