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Malignant Hyperthermia
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**Definition**

Malignant hyperthermia (MH) is an autosomal-dominant disorder that is inherited. The disturbance of calcium homeostasis associated with MH affects skeletal muscle (Schneiderbarger, Johannsen, Roewer, & Schuster, 2014). This hypertensive muscular disorder is triggered by exposure to depolarizing muscle relaxants such as succinylcholine or halogenated volatile anesthetics (Vamvakas, D’Alonzo, & Vavault, 2009). When the dysfunctional receptor is exposed to triggering agents, such as halogenated volatile anesthetics and succinylcholine, a depolarizing neuromuscular blocking agent, a prolonged opening of this dysfunctional receptor occurs and results in the uncontrolled release of calcium within the sarcoplasmic reticulum, causing a hypermetabolic state in the susceptible individual (Kim, 2012). As a result, MH occurs more often in children and young adults than in the adult population. MH occurs more often in adult males than adult females. Distribution across racial groups is variable (Stratman et al., 2009). While a link between MH and numerous myopathies is suspected, a predisposition has only been identified in X-linked myopathy, central-core disease, and King-Dubosh syndrome (Stratman et al., 2009).

Although MH is a rare inherited disorder, this potentially life-threatening inherited disorder may occur anywhere from once in every 3,000 procedures involving general anesthesia (Boggs, McCaig, & Powers, 2012). While obtaining thorough patient and family histories related to anesthesia events will reduce occurrences of MH, thorough patient and family histories related to anesthesia events will reduce occurrences of MH and relevant family histories. More efficient treatment and improved understanding of the pathophysiology related to this disorder have resulted in an 11.7% decline in the mortality rate from MH from 2000 to 2005 (Johlie, Stintz, Schott, & Gaul, 2012).

However, to further improve outcomes for patients with malignant hyperthermia, it is imperative for multidisciplinary medical team members to promptly recognize early symptoms of MH and accurately diagnose this disorder (Dirksen et al., 2013). Early recognition has the appropriate treatment (Dirksen, Van Winkle, & Schuster, 2014, p. 189).

**Relevance**

MH treatment
See Figure 2 for recommended roles and treatment (Dirksen, Van Wicklin, & Schuster, 2011, Thomas, J., & Crowhurst, T. (2013). The ultimate goal is to prevent the occurrence of MH. As discussed by Beggs, McCaig, and Powers (2012), prior to the administration of anesthesia, obtaining thorough patient and family histories related to anesthesia reactions may alert the anesthesiologist to the patient’s possible predisposition to MH and dictate the selection of non-triggering MH anesthetics agents. However, when prevention is not realized, continued vigilance and crisis preparedness on the part of multidisciplinary medical team members will further reduce the MH mortality rate and improve patient outcomes (Schneiderbarger et al., 2014).

**Underlying Pathophysiology**

The following physiologic manifestations are linked to MH:

1. **Muscular rigidity** most often involves the extensor muscles of the back, neck, and extremities. This muscle rigidity is often accompanied by a hypermetabolic state in the susceptible individual (Kim, 2012).

2. **Hyperkalemia** results from damage to the cell membranes allowing intracellular contents, such as magnesium, potassium, creatine, and myoglobin to seep into the bloodstream (Dirksen et al., 2013).

3. **Rhabdomyolysis** occurs when skeletal muscle sarcoplasmic reticulum causing a hypermetabolic state in the susceptible individual (Kim, 2012).

4. **Hypoxia** and **acidosis** occur due to hyperkalemia and the kidneys stimulating the sympathetic nervous system resulting in increased blood pressure, decreased cardiac output, and increased myocardial oxygen demand (Dirksen et al., 2013).

5. **Hypercapnia** is a common and early sign of MH in mechanically ventilated and intubated patients (Kim, 2012).

6. **Failure of the kidneys** results from damage to the cell membranes allowing intracellular contents, such as magnesium, potassium, creatine, and myoglobin to seep into the bloodstream (Dirksen et al., 2013).

7. **Failure of the cardiovascular system** results in rapid heart rate and/or rapid breathing (Dirksen et al., 2013).

8. **Failure of the kidneys** results from damage to the cell membranes allowing intracellular contents, such as magnesium, potassium, creatine, and myoglobin to seep into the bloodstream (Dirksen et al., 2013).

9. **Cardiac arrhythmias/failure**: Potentially fatal dysrhythmias occur due to hypokalemic and the kidneys inability to excrete excess K (Dirksen et al., 2013).

10. **Increased levels of CO2** is a common and early sign of MH (Dirksen et al., 2013).

11. **Excessive production of CO2** results from damage to the cell membranes allowing intracellular contents, such as magnesium, potassium, creatine, and myoglobin to seep into the bloodstream (Dirksen et al., 2013).

**Significance of Pathophysiology**

**References**