

## Malignant Hyperthermia (MH) Crisis, Care of the Patient with Acute or Potential in PACU

### Quick Links:

MH Triggering Agents - [Appendix A](#)

MH Signs & Symptoms – Early & Late - [Appendix B](#)

### Site Applicability

VGH & UBC: PACU

### Practice Level

RN

### Need to Know

Malignant hyperthermia (MH) is an inherited and potentially fatal disorder of skeletal muscle calcium metabolism. MH is usually triggered when a susceptible individual is exposed to volatile inhalation anesthetic agents (e.g. Desflurane, Sevoflurane) and/or depolarizing skeletal muscle relaxants, (e.g. succinylcholine). Other drugs & conditions such as physical and emotional stress are also implicated as triggering agents in MH susceptible individuals (See [Appendix A](#)).

Exposure to the trigger results in a series of hypermetabolic reactions. Generation of excess CO<sub>2</sub>, O<sub>2</sub> depletion, muscle rigidity and massive heat production occur.

Eventual cell death and rhabdomyolysis cause metabolic acidosis, hyperkalemia and myoglobinaemia/myoglobinuria. Secondary complications of an MH crisis include acute renal failure, DIC, and cardiac dysrhythmias, predominantly ventricular in origin.

The most common initial sign of MH crisis is an unexpected rise in end-tidal carbon dioxide (ETCO<sub>2</sub>) resistant to increasing the minute ventilation. Other early signs may include muscle rigidity, mixed respiratory & metabolic acidosis, tachycardia and dysrhythmias. Fever is often a late sign but may develop rapidly – rates of 1oC every 3 to 5 minutes have been documented. (See [Appendix B](#)). However, clinical presentation is not uniform.

MH may onset at any time during anaesthesia and in the early postoperative period. In addition, the patient may relapse following initial, successful treatment for an MH event – recrudescence occurs in up to 25% of patients after initial treatment & can be fatal. Onset in PACU is rare and clinical symptoms may be subtle. If unexplained tachycardia, fever and tachypnea occur in the PACU patient, MH onset or relapse as applicable should be considered.

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When an MH crisis is recognized or suspected, triggering agents are stopped immediately and changed to non-triggering agents. Dantrolene Sodium (Dantrium®, Revonto®) is the only definitive treatment for MH and IV administration begins as soon as possible. All other treatments are directed towards managing symptoms or complications of MH.

A number of other conditions such as septicemia, transfusion reaction and thyroid storm have symptoms that overlap with those of MH (hypercarbia, tachycardia, fever, dysrhythmias, muscle rigidity). However, MH treatment is generally started as soon as the patient's symptoms make an MH diagnosis probable.

## Equipment and Supplies

1. Malignant Hyperthermia Cart
  - VGH: Case Cart Room
  - UBCH: Anesthetic Supply Room
  - Page AA to bring cart as needed
2. For Dantrolene reconstitution & administration:
  - Refer to guidelines on MH Carts and PDTM
3. Cooled 0.9% NaCl for IV infusion
  - VGH: Obtain from refrigerator adjacent to OR 10 in Sterile Core
  - UBCH: Fridge outside Preop
4. Cooling blanket (as needed) (VGH only)
5. Ice (as needed)

## Practice Guideline

### Care and Management

**PACU priorities** for care & management of an acute MH crisis in PAR focus on:

- **Early recognition of onset or relapse in PACU**
- Initiating and/or maintaining emergent therapy such as Dantrolene and cooling measures
- Supportive management of MH sequelae such as
  - Ventilatory support to help blow off excess metabolic CO<sub>2</sub>
  - Volume support to facilitate renal excretion of myoglobin
- Monitoring for & treating potential complications of acute MH such as
  - Recrudescence
  - Renal compromise
  - Dysrhythmias

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- Coagulopathies
- Providing emotional support to the patient and family

PROBLEM	MANAGEMENT
<p><b>Temperature instability</b> related to hypermetabolic response (acute); post-treatment relapse (recrudescence)</p>	<ol style="list-style-type: none"> <li><b>Assess/monitor:</b> <ul style="list-style-type: none"> <li>Continuous temperature monitoring via nasal or esophageal probe as appropriate</li> <li>Serial CK, E+, BUN, creatinine, ABG's, lactate (prn), troponin (prn)</li> <li>Signs of onset or relapse: <ul style="list-style-type: none"> <li><b>increasing</b> PCO2 via capnography or serial ABG's</li> <li>persistent/worsening metabolic acidosis</li> <li>serum lactate &gt; 2mM/L</li> <li>jaw stiffness or abnormal muscle tone</li> <li><b>increasing</b> body temperature, tachycardia and tachypnea</li> <li><b>Side Effects of Dantrolene therapy</b> <ul style="list-style-type: none"> <li>thrombophlebitis (if given peripherally)</li> <li>muscle weakness</li> <li>dizziness</li> <li>blurred vision</li> </ul> </li> </ul> </li> </ul> </li> <li><b>Initiate or maintain Dantrolene as per medical orders:</b> <ul style="list-style-type: none"> <li>initial recommended dose is 2.5 mgm/kgm rapid IV bolus then Q5 minutes until symptoms abate – doses up to 30 mgm/kgm may be needed in some cases</li> <li>post-acute dose is 1 mgm/kgm Q4-6H or 0.25 mgm/kgm/hr for at least 24 hours</li> <li>administer Dantrolene via a central line if possible.</li> <li>Implement/maintain cooling measures for core temperature &gt; 39°C: <ul style="list-style-type: none"> <li>cooling blanket</li> <li>cooled IV infusions</li> <li>ice packs</li> <li>iced gastric or bladder lavage</li> </ul> </li> </ul> </li> </ol> <p><b>**NB: Halt all cooling measures when T 38.0°C**</b></p> <ul style="list-style-type: none"> <li>Remove/manage potential triggering agents/stimuli in patient's environment:</li> </ul>

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	<ul style="list-style-type: none"> <li>Review ordered drugs (See <a href="#">Appendix A</a>) for potential triggers</li> <li><b>Prevent/immediately stop shivering</b> <ul style="list-style-type: none"> <li><b>discontinue cooling</b> when T at 38.0°C</li> <li>administer Demerol (as ordered) for shivering</li> <li>Treat pain &amp; anxiety</li> </ul> </li> </ul>
<b>Respiratory insufficiency</b> secondary to hypermetabolism (increased O <sub>2</sub> demand & increased CO <sub>2</sub> production)	<ol style="list-style-type: none"> <li>Assess/monitor: <ul style="list-style-type: none"> <li>Oxygenation &amp; ventilation parameters as per PACU protocol and, in addition: <ul style="list-style-type: none"> <li>serial ABG's for evidence of hypoxaemia, hypercarbia, acidosis</li> <li>PetCO<sub>2</sub> (as applicable)</li> <li>excessive work of breathing/ventilatory fatigue</li> </ul> </li> <li>Supplemental O<sub>2</sub>/mechanical ventilation as per medical orders <ul style="list-style-type: none"> <li>Supranormal VE may be ordered to initially to blow off excess CO<sub>2</sub></li> </ul> </li> </ul> </li> </ol>
<b>Cardiac instability/dysrhythmias</b> secondary to hyperkalemia, acidosis, MDO <sub>2</sub> :MVO <sub>2</sub> imbalance	<ol style="list-style-type: none"> <li><b>Assess/monitor:</b> <ul style="list-style-type: none"> <li>Continuous ECG as per PACU protocol with special attention to arrhythmias of ventricular origin, ST/T changes and sudden onset chest pain</li> <li>Signs of cardiac dysfunction e.g., pulmonary crackles, hypotension, increasing CVP</li> <li>Serial K<sup>+</sup> levels, arterial pH &amp; pO<sub>2</sub>, 12 Lead ECG, troponin as indicated</li> </ul> </li> <li><b>Treat/resolve hyperkalemia</b> as per medical orders – IV NaHCO<sub>3</sub>-, IV glucose + insulin or, if life threatening, CaCl<sub>2</sub></li> <li><b>Treat/resolve acidosis</b> via hyperventilation and/or IV Na HCO<sub>3</sub>-</li> <li><b>Treat significant/persistent arrhythmias</b> <ul style="list-style-type: none"> <li>Standard anti-dysrhythmics may be used with the exception of Calcium Channel blockers</li> </ul> </li> </ol>
<b>Renal dysfunction</b> secondary to myoglobinuria	<ol style="list-style-type: none"> <li><b>Assess/monitor:</b> <ul style="list-style-type: none"> <li>Serial BUN, creatinine and urine myoglobin</li> <li>Hourly urine output <ul style="list-style-type: none"> <li>Dark/cola-coloured urine indicates presence of myoglobin</li> </ul> </li> </ul> </li> <li><b>Ensure urine output</b> of &gt; 2 ml/kg/hour via appropriate IV fluids and diuretics if myoglobinuria present</li> </ol>

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	3. <b>Administer NaHCO<sub>3</sub></b> - as ordered to alkalinize the urine
<b>Coagulation dysfunction</b> secondary to DIC	<ol style="list-style-type: none"> <li><b>Monitor/assess:</b> <ul style="list-style-type: none"> <li>Signs of coagulopathy: <ul style="list-style-type: none"> <li>unexpected bleeding/oozing from mucous membranes, surgical site(s), arterial &amp; venous cannulation sites</li> <li>petichiae &amp; haematomae</li> <li>serial coagulogram, CBC &amp; platelets</li> </ul> </li> </ul> </li> <li><b>Maintain adequate tissue perfusion &amp; oxygenation</b> via IV fluids, supplemental O<sub>2</sub>/ventilatory support, and hemodynamic &amp; cardiac management</li> <li>Administer coagulation factors and/or anticoagulants as ordered</li> </ol>
<b>Compartment syndrome</b> secondary to muscle edema	<ol style="list-style-type: none"> <li><b>Assess/monitor:</b> <ul style="list-style-type: none"> <li>CSMW to extremities as per PACU protocol</li> </ul> </li> </ol>
<b>Neurological dysfunction</b> – seizures or coma - secondary to hyperthermia, hypo-perfusion, acidosis, E+ imbalance	<ol style="list-style-type: none"> <li><b>Assess/monitor</b> <ul style="list-style-type: none"> <li>NVS as per PACU protocol</li> <li>Serial ABG's, E+</li> </ul> </li> <li><b>Maintain adequate tissue perfusion &amp; oxygenation</b></li> <li><b>Treat/resolve hyperkalemia and/or acidosis</b></li> <li><b>Implement seizure precautions prn</b></li> </ol>
<b>Pain</b> secondary to sustained muscle contraction and surgical procedure	<ol style="list-style-type: none"> <li><b>Assess/monitor:</b> <ul style="list-style-type: none"> <li>Pain level/sedation score as per PACU protocols</li> <li>Differentiate expected surgical pain from residual muscle soreness</li> </ul> </li> <li><b>Administer IV analgesics as per medical orders</b></li> </ol>

## Expected Client / Family Outcomes

The patient will demonstrate resolution of the actual/potential MH event as evidenced by:

- Vital signs within normal limits for the patient
- Oxygenation and ventilation parameters within normal limits
- Stable, perfusing cardiac rhythm with no evidence of myocardial ischaemia
- Normothermia
- Metabolic parameters – pH, HCO<sub>3</sub><sup>-</sup>, CK, serum/urine myoglobin, electrolytes – within normal limits or with evidence of substantial resolution of previous abnormalities
- Renal function within normal limits
- Absence of unexpected/abnormal bleeding

## Related Documents

- Muscle weakness/easy tiring may persist for several months.
- Testing of self & family members for MH susceptibility
- Ensure health care providers (surgeons, anesthesiologists) are notified re MH event prior to future surgeries/anesthetics
- Medical Alert bracelet and wallet card
- Resources for MH information and support, i.e., MH Association of the US at [www.mhaus.org/](http://www.mhaus.org/) / Tel: 1-209-417-3722

## Documentation

Document initial and ongoing assessments & interventions including VS, LOC, sensory/motor level, surgical parameters, medications given, complications/problems experienced and patient outcomes in accordance with PACU standards.

## Related Documents

- [Care of the Post Anaesthetic Patient in Phase I \[D-00-07-30260\]](#)

## References

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## Appendix A

### APPENDIX A: TRIGGERING & SAFE AGENTS FOR MALIGNANT HYPERTHERMIA

TYPE	AGENT
Triggering (Unsafe) Agents	<b>Depolarising Muscle Relaxants</b> <ul style="list-style-type: none"> <li>Succinylcholine</li> </ul> <b>ALL Volatile General Anaesthetics, e.g.</b> <ul style="list-style-type: none"> <li>Desflurane</li> <li>Sevoflurane</li> <li>Isoflurane</li> <li>Enflurane</li> <li>Ether</li> </ul> <b>Other</b> <ul style="list-style-type: none"> <li>K<sup>+</sup> salts</li> <li>Ca<sup>++</sup> Channel Blockers (not a trigger but can precipitate hyperkalemia and/or cardiac arrest when used with Dantrolene sodium)</li> </ul>
Non-Triggering (Safe) Agents	<b>ALL Non-Depolarising Muscle Relaxants, e.g.</b> <ul style="list-style-type: none"> <li>Rocuronium</li> <li>Cisatracurium</li> <li>Mivacurium, etc</li> </ul> <b>Nitrous Oxide</b> <b>Intravenous Anaesthetics, e.g.</b> <ul style="list-style-type: none"> <li>Ketamine (does not trigger but not recommended due to sympathomimetic effects)</li> <li>Propofol</li> <li>Sodium thiopental</li> <li>Opioids</li> <li>Benzodiazepines</li> </ul> <b>ALL Ester &amp; Amide Local Anaesthetics, e.g.</b> <b>Reversal Agents</b> <ul style="list-style-type: none"> <li>Neostigmine with Atropine or Glycopyrrolate</li> </ul> <b>Other</b> <ul style="list-style-type: none"> <li>All standard ACLS drugs <i>with the exception</i> of Ca<sup>++</sup> Channel blockers</li> </ul>
Other (Possible) Triggers	<b>Shivering</b> <b>Significant emotional/physical stress</b> <ul style="list-style-type: none"> <li>Pain</li> <li>Anxiety/agitation</li> </ul>

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## Appendix B

### APPENDIX B: CLINICAL SIGNS IN MALIGNANT HYPERTHERMIA

<p><b>EARLY</b></p> <ul style="list-style-type: none"> <li>Signs &amp; symptoms reflect systemic, uncontrolled hypermetabolism</li> <li>Clinical presentation is not uniform &amp; time of onset is variable</li> </ul>	<p><b>CLINICAL SIGNS:</b></p> <ul style="list-style-type: none"> <li><b>Hypercarbia</b> <ul style="list-style-type: none"> <li>Usually noted as significant <math>\uparrow</math> in <math>P_{ET} CO_2</math> unresponsive to <math>\uparrow V_E</math></li> </ul> </li> <li><b>Tachypnea</b> (if patient breathing spontaneously)</li> <li><b>Tachycardia</b> <ul style="list-style-type: none"> <li>Often in association with ventricular dysrhythmias</li> </ul> </li> <li><b>Hypertension</b></li> <li><b>Muscle Rigidity</b> <ul style="list-style-type: none"> <li>Masseter muscle rigidity post-succinylcholine administration</li> <li>Generalized rigidity unresponsive to NMB agents</li> </ul> </li> </ul> <p><b>LAB WORK:</b></p> <ul style="list-style-type: none"> <li><math>\uparrow P_{ET} CO_2</math> and <math>PaCO_2</math></li> <li><math>\downarrow pH</math> (respiratory and metabolic)</li> <li><math>\downarrow PaO_2</math></li> <li><math>\uparrow K^+</math></li> </ul>
<p><b>LATER</b></p> <ul style="list-style-type: none"> <li>signs &amp; symptoms reflect effects of continuous skeletal muscle contraction (heat generation &gt; heat dissipation, marked <math>\uparrow</math> in <math>O_2</math> consumption, rhabdomyolysis)</li> </ul>	<p><b>CLINICAL SIGNS:</b></p> <ul style="list-style-type: none"> <li><b>Hyperthermia</b> <ul style="list-style-type: none"> <li>once initiated, body temperature can <math>\uparrow</math> 1-2°C Q5minutes</li> </ul> </li> <li><b>Skin mottling/cyanosis</b></li> <li><b>Dark/cola-coloured urine</b> <ul style="list-style-type: none"> <li>reflects myoglobinuria secondary to rhabdomyolysis</li> </ul> </li> <li><b>Abnormal/unexpected bleeding</b> <ul style="list-style-type: none"> <li>possible DIC</li> </ul> </li> </ul> <p><b>LAB WORK</b></p> <p><b>Above plus:</b></p> <ul style="list-style-type: none"> <li><math>\uparrow CK</math></li> <li>myoglobin in blood and urine</li> <li><math>\uparrow BUN/creatinine</math></li> <li><math>\uparrow INR/PTT/FSP</math>, <math>\downarrow</math> platelets</li> </ul>

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