WILLIAM ELCOTT is admitted to your postanesthesia care unit (PACU) after undergoing orthopedic surgery. Almost immediately, you notice some alarming signs and symptoms: muscle rigidity, tachypnea, and tachycardia. Mr. Elcott’s blood pressure is 154/90 mm Hg and his temperature is 100.2°F (37.9°C). A quick glance at his chart tells you both vital signs have gone up since the surgery ended.

You know you need to act quickly and decisively because, based on Mr. Elcott’s signs and symptoms, you strongly suspect malignant hyperthermia. If left untreated, the condition can be rapidly fatal. You notify the anesthesia provider immediately, call for a malignant hyperthermia cart, administer 100% oxygen via a non-rebreather mask, and closely monitor his cardiac rhythm and vital signs.

What happened with Mr. Elcott to cause this problem? Let’s find out.

**The smoking gun**

Susceptibility to malignant hyperthermia is an inherited disorder. Individuals with this gene abnormality are at risk for having a violent reaction to inhaled anesthetics, like halothane (Fluothane), sevoflurane (Ultane), desflurane (Suprane), enfurane (Ethrane), and isoflurane (Forane); depolarizing muscle relaxants, like succinylcholine (Anectine); and illicit drugs, like methamphetamine and 3,4-methylenedioxymethamphetamine (Ecstasy). Fortunately, susceptibility is rare: Although accurate statistics are unavailable, it’s estimated that 1 in 15,000 children and 1 in 20,000 to 50,000 white adults have the abnormality.

The most prevalent sign of malignant hyperthermia is uncontrolled skeletal muscle hypermetabolism. Structures within the muscle cells, called the sarcoplasmic reticulum, store and release calcium ions. In malignant hyperthermia, these stores are released in a flood in reaction to a triggering agent.

**Fueling the fire**

This flood of calcium causes glycogenolysis, cell metabolism, and skeletal muscle contracture to skyrocket. The result is a fairly rapid buildup of heat and lactate. As the process continues, the body uses more oxygen and produces more carbon dioxide, leading to acidosis, hypercapnia (partial pressure of carbon dioxide \(\text{Pa}_2\)) in the blood over 45 mm Hg), and hypoxemia (partial pressure of oxygen under 70 to 80 mm Hg). Tachycardia happens early in the process; hyperthermia develops at a rate of about 1° to 2° C every 5 minutes.

Rhabdomyolysis can occur when massive amounts of myocyte (muscle cell) contents are released into the bloodstream, as happens in malignant hyperthermia. Potassium, magnesium, phosphate, acids, creatine kinase, lactate dehydrogenase, and myoglobin, which are essential to cell metabolism, are toxic when they’re released into the bloodstream. Hyperkalemia (an abnormally high level of potassium) can cause a cardiac arrhythmia or even arrest. Myoglobinuria (myoglobin in the urine) can set off acute renal failure.

All of the conditions associated with hypermetabolic reaction are ripe for causing a disaster.

**Sparking an idea**

Mr. Elcott’s signs and symptoms—muscle rigidity, tachypnea, tachycardia, rising blood pressure of 154/90 mm Hg, and rising temperature of 100.2°F (37.9°C)—led...
you to suspect malignant hyperthermia. Other clinical signs that point to this diagnosis include the following:

- \( \text{PaCO}_2 \) greater than 60 mm Hg
- unexplained sinus tachycardia, ventricular tachycardia, or ventricular fibrillation
- serum pH less than 7.25
- serum creatine kinase concentration greater than 20,000 units/L
- cola-colored urine and/or elevated level of myoglobin in the urine or blood.

When the signs all point to malignant hyperthermia, the goal of treatment is to turn down the heat and limit the damage. We’ll go over how best to achieve that next.

Chill, Will

You’ve notified the anesthesia provider, called for the malignant hyperthermia cart, begun oxygen administration, and made sure Mr. Elcott’s I.V. line is patent. If you haven’t done so yet, pull off his sheets and blankets and, if possible, lower the room temperature. You’ve got to cool down this patient! Other cooling strategies include administering chilled 0.9% sodium chloride solution and placing cold packs on the groin, armpits, and neck. A cooling blanket may be effective too. Don’t overdo it, though: To avoid hypothermia, stop all of these cooling measures when the patient’s temperature gets down to 100° F (37.8° C).

The anesthesia provider orders I.V. dantrolene (Dantrium). It’s a direct-acting skeletal muscle relaxant and the drug of choice for treating malignant hyperthermia. It’s administered by continuous rapid I.V. push, beginning with a minimum dose of 1 mg/kg (the provider orders an initial dose of 2.5 mg/kg for Mr. Elcott) and continuing until symptoms subside or the maximum cumulative dose of 10 mg/kg is reached. Monitor Mr. Elcott closely for respiratory depression.

Watch for brownish urine; it could signal myoglobinuria. Obtain blood samples for lab work, and get an arterial blood gas analysis. Keep an eye out for abnormally high levels of potassium, calcium, magnesium, and creatine kinase and for signs of respiratory or metabolic acidosis.

Also recall that I said a cardiac arrhythmia may develop. If it does, treat it according to advanced cardiac life support guidelines. One word of caution here: Don’t give a patient with malignant hyperthermia who’s been treated with dantrolene a calcium channel blocker. A serious drug interaction can occur and cause severe hyperkalemia, ventricular fibrillation, and cardiac arrest.

Smoldering embers

Let’s check back with Mr. Elcott. Your quick work has avoided a potential disaster. Although he’s making an unscheduled stop in the ICU before discharge, Mr. Elcott’s chances for a full recovery are very good. While in the ICU, he’ll continue to receive I.V. dantrolene at a rate of 1 mg/kg every 4 to 6 hours for the next 24 to 48 hours to prevent a recurrence of malignant hyperthermia. He’ll also be monitored closely for signs of complications, like renal failure and disseminated intravascular coagulation, an abnormal condition of coagulation characterized by diffuse bleeding and hypercoagulability that can arise with malignant hyperthermia.

When Mr. Elcott’s ready to be discharged home, he should be informed about his condition. The Malignant Hyperthermia Association of the United States is an excellent resource for printed and online patient-teaching materials and genetic counseling. Because malignant hyperthermia is an autosomal dominant trait, the aberrant gene need be present in only one parent for it to be passed down to offspring.

One last word of advice: Tell Mr. Elcott to alert his primary health care provider of his susceptibility to malignant hyperthermia and to wear a medical-alert bracelet. That way, if he’s unable to speak and needs to be given anesthesia, triggering agents can be avoided.

Learn more about it

