A VOICE on the EMS radio interrupts your thoughts: “Med 2 en route with 21-year-old male involved in motorcycle crash. Patient was wearing helmet and had no loss of consciousness. At present he is conscious, alert, and oriented, but complaining of abdominal pain. Heart rate 110 bpm with BP 120/90. ETA 5 minutes.”

You rush to assist with setting up the trauma room. Moments later, your patient arrives, appearing anxious and complaining of severe abdominal pain, which he rates at 8 on a 0-to-10 pain intensity scale. You note his skin is pale and diaphoretic. He’s tachypneic, and a quick radial pulse check shows that he’s tachycardic. As you call for the ED physician, you realize that you’re seeing signs of impending shock. This man could die from unresolved hypovolemic shock unless the team responds quickly.

Hypovolemia is the most common cause of shock. Hypovolemia is the most common cause of shock.1,2 (For other shock types, see What’s shock?) Although it’s often due to injury and massive blood loss, it can also have nontraumatic causes, such as a gastrointestinal bleed or ruptured ectopic pregnancy. Hypovolemia can also be caused by fluid losses and third-spacing from burn injuries, or dehydration from frequent vomiting or diarrhea.

This article discusses the causes, treatments, and nursing care for patients with traumatic hypovolemic shock. For a refresher on pathophysiology, see Pathophys particulars.

Compensated or not?

One classification system categorizes hypovolemic shock into three stages: compensated, uncompensated, and irreversible.3 The value of this method is that the name of each phase describes what’s seen clinically.

Early compensated shock occurs when the body’s compensatory mechanisms are adequate to maintain cardiac output. Signs and symptoms of the compensatory response include tachycardia, tachypnea, oliguria, and anxiety due to sympathetic nervous system (SNS) stimulation; and skin changes (pallor, decreased capillary refill time, cool temperature, and diaphoresis) secondary to peripheral vasoconstriction, also

By Jeff Strickler, MA, RN, CEN, CFRN, EMT-P
due to SNS stimulation. Decreased cardiac output leads to decreased renal perfusion, which activates the renin-angiotensin-aldosterone system, leading to oliguria and further SNS activation. BP is often normal in early stages of shock, but progression of shock leads to orthostatic or postural hypotension.

Uncompensated shock occurs when compensatory mechanisms start to fail and can no longer maintain adequate cardiac output, causing hypotension. Because a decrease in systolic BP doesn’t occur until at least 20% of blood volume is lost, it’s important to note that hypotension is a late sign.⁴

Irreversible shock is often associated with losses of more than 25% of total blood volume. Unchecked, shock leads to cellular ischemia and subsequent acidosis, cellular necrosis, and organ failure that can’t be resolved, even if cardiac output increases.² Once cellular breakdown and acidosis reach critical levels, reperfusion may lead to reperfusion injury, during which oxygen-free radicals overwhelm remaining cellular activity and cause neutrophil infiltration, microvascular damage, and impairment of the microcirculation.

Case study progression
Back to our patient… As the team begins assessment, you note that he’s becoming increasingly confused.

What’s shock?¹,²,¹³
A clinical syndrome of inadequate tissue perfusion, shock results in a decreased supply of oxygen and nutrients to cells. The body responds initially by activating numerous compensatory mechanisms to improve cellular perfusion. If these fail, shock leads to widespread cellular necrosis, multiple organ dysfunction and failure, and death.

Although there are various types of shock, including hypovolemic, cardiogenic, neurogenic, anaphylactic, and septic, the final common pathway in all types of shock is impaired cellular metabolism.

His abdomen is markedly distended and tender to light palpation. Despite receiving 100% oxygen via a nonrebreather mask, he remains tachypneic. After placing him on a cardiac monitor, you see sinus tachycardia at 120 bpm. An indwelling urinary catheter is inserted with minimal initial output. His BP is now 90/70. You realize that your patient is now decompensating.

You recognize that an alteration in level of consciousness (LOC), tachypnea, tachycardia, peripheral vascular changes, and oliguria are signs of shock. The drop in BP is an ominous sign that shock is progressing.

As always, your nursing assessment starts with the ABCs (assess and support airway, breathing, and circulation) followed by determining the patient’s pertinent medical history and the mechanism of traumatic injury. When a patient is still adequately compensating for the fluid loss, this history may provide the first clue to the injury’s real extent and nature. Also perform a complete head-to-toe assessment to determine locations of pain, ecchymoses, or distention that could point to occult bleeding.

An adequate assessment includes evaluation of the chest, abdomen, and pelvis. Evaluation of the abdomen is conducted initially through a focused assessment sonography for trauma (FAST) scan, which can identify pericardial fluid in the chest as well as intraperitoneal fluid in the abdomen. If the patient is hemodynamically stable, then a CT scan is indicated for more definitive evaluation of any injuries.

Long-bone fractures such as femur fractures can also cause significant hypovolemia, so prepare to obtain extremity films as appropriate.

A central venous pressure reading of less than 4 mm Hg (<5 cm H₂O) indicates hypovolemia.³ Lab analysis shows decreased levels of hemoglobin and hematocrit, elevated lactate levels, and the presence of an arterial base deficit greater than −2 mmol/L.

Serum lactate and arterial base deficits are considered proxies for oxygen debt. Serum lactate levels that remain high signal that the body is attempting to produce energy through anaerobic metabolism.

Base deficit is considered a surrogate marker of metabolic acidosis. Patients with a significant arterial base deficit are more likely to die from the oxygen debt and poor metabolic state.⁷

Normalization of both lactate levels and base deficit are considered resuscitation endpoints for determining the degree of oxygen debt and the patient’s response to resuscitation. These are more significant indicators than normotension.⁶

Many current efforts are leading to more direct measurements of cellular perfusion, such as measuring gastric pH or oxygenation in the tissues (StO₂). Via infrared spectrometry, StO₂ noninvasively measures hemoglobin oxygen saturation in subcutaneous tissue or skeletal muscles.⁷

Be aware of special considerations for certain age groups and patient populations. For example, pediatric patients may not exhibit classic signs and symptoms of hypovolemic shock before loss of more than 25% blood volume.³ Because compensatory mechanisms in a child are particularly robust, BP may be maintained until about 30% of blood volume has been lost.

These other patient groups may also have atypical signs and symptoms:

- Older adults may not exhibit classic signs due to inadequate physiologic reserves and an inability to initiate compensatory mechanisms—for example, because of reduced alpha-1 adrenergic receptor responsiveness in older adults.
- Patients on beta-adrenergic antagonists may not be able to initiate the expected tachycardic response.
- Pregnant patients may be able to lose up to 1500 mL of blood without a change in BP, secondary to alterations in blood volume and
hemodynamics related to pregnancy. During the third trimester of pregnancy, compression of the vena cava can reduce venous return to the heart. Placing these patients on their left side or manual displacement of the uterus to the left can dramatically increase cardiac output.

**Treatment priorities**
Management of patients with hypovolemic shock has three primary goals:
- Maximize oxygen delivery by ensuring an adequate airway (which may have been impacted as the LOC decreases) and by improving oxygenation through administration of high-flow oxygen via a nonrebreather mask or mechanical ventilation.
- Control hemorrhage through basic means, such as direct manual pressure, interventional angiography, or surgical intervention.
- Restore and maintain adequate cardiac output. To meet this goal, I.V. fluid replacement is a top priority. (In early shock from dehydration, oral fluid replacement may be adequate.) The I.V. fluid of choice is an isotonic crystalloid, such as 0.9% sodium chloride solution or Ringer's lactate solution.

The type of fluids used to treat hypovolemia is still controversial. Crystalloids are generally recommended, but colloids are often used to expand the intravascular space. However, most recent studies fail to demonstrate improved survival with colloids to offset the expense of their use.

Additionally, hypertonic crystalloid solutions are sometimes administered to shift fluids from the intracellular and interstitial spaces into the intravascular space. But research has produced conflicting evidence about the effectiveness of this strategy except as a means to manage increased intracranial pressure.

Generally, every 1 mL of blood loss requires 3 mL of fluid to restore adequate cardiac output. Patients
Lessons learned from recent military actions

Many advances in the management of traumatic hypovolemic shock have been developed in the combat zones of Iraq and Afghanistan. Two changes are leading to the better control of bleeding following traumatic injury:
- hemostatic granules/powders used as a foundation to initiate rapid clotting.
- renewed use of tourniquets for control of external hemorrhage. Studies have shown that despite earlier fears, patients can tolerate prolonged tourniquet application without serious complications.

Recent experiences in combat medicine have also shown the value of whole blood over packed red cells. Administering whole blood or the supplement of packed red cells with plasma, platelets, or coagulation factors has reduced the incidence of the complications associated with severe hypovolemic shock. Although a conclusive recommendation for the ratio of red blood cells to plasma hasn’t been established, it’s much closer to 1:1 than originally believed.

Blood substitutes have been studied extensively, but despite some promising research, no clinically viable product is currently available.

Case closed
As you continue to assess and support your patient, the ED team moves at

Diagnostic imaging and ongoing care

After fluid resuscitation, the priority becomes identifying the underlying cause of shock. Many patients with traumatic hypovolemic shock require diagnostic imaging studies, such as ultrasound or computed tomography scanning. Severe internal injury and ongoing blood loss may require surgery to locate and stop bleeding.

Ongoing nursing assessment should target adequate response to treatments. Monitor for improving LOC, increasing urine output (greater than 0.5 mL/kg/hour or at least 30 mL/hour in an average-sized adult), and hemodynamic stability. Continual monitoring of endpoints of resuscitation is critical. (As previously mentioned, these endpoints are indicators of tissue perfusion, such as serum lactate levels and restoration of normal arterial base deficits.)

Assess for and manage the trauma triad of hypothermia, coagulopathy, and acidosis, which predisposes the patient to a poor outcome. Coagulopathy can occur in patients who receive large amounts of volume resuscitation due to the dilution and consumption of platelets and clotting factors. This coagulopathy should be corrected by the administration of fresh frozen plasma, platelets, and cryoprecipitate.

In the past, pneumatic antishock devices hadn’t demonstrated improved patient outcomes, so it’s no longer recommended to treat shock. Its only application is immobilization of pelvic or lower extremity fractures.

A recent large, randomized, placebo-controlled study demonstrated that IV tranexamic acid significantly reduced the risk of death from hemorrhage in bleeding trauma patients. For more on promising treatment advances for hypovolemic shock, see Lessons learned from recent military actions.
shock is at high risk for significant morbidity and mortality. Your knowledge, preparation, and rapid interventions support the patient’s survival and optimal recovery from this life-threatening disorder.

REFERENCES
9. Tyagi R, Donaldson K, Loftus CM, Jallo J. Hypothermia. Specimens are taken while a coworker establishes a second large-bore venous access. Using the fluid warmer helps prevent hypothermia. A bedside FAST scan shows a significant amount of free intraperitoneal fluid. The on-call surgeon decides to operate immediately due to the ongoing internal bleeding and hemodynamic instability.

After surgical repair of a lacerated mesenteric artery and normalization of his fluid status, the patient recovers uneventfully.

Being prepared saves lives
Despite aggressive resuscitative efforts, a patient in traumatic hypovolemic shock is at high risk for significant

an accelerated pace. As prescribed, you administer a bolus infusion of Ringer’s lactate through the peripheral venous access established by the EMTs while a coworker establishes a second large-bore venous access. Using the fluid warmer helps prevent hypothermia. Specimens are taken for initial lab tests and a call is placed to the blood bank requesting type-specific units of RBCs. A stat portable chest X-ray reveals no pneumothoraces, traumatic hemomediatinum, or hemothorax.

A bedside FAST scan shows a significant amount of free intraperitoneal fluid. The on-call surgeon decides to operate immediately due to the ongoing internal bleeding and hemodynamic instability.

After surgical repair of a lacerated mesenteric artery and normalization of his fluid status, the patient recovers uneventfully.

Traumatic hypovolemic shock: Halt the downward spiral