Anesthesiologists routinely care for patients during life-threatening emergencies. Anesthesia providers often belong to code teams and frequently are included in rapid response teams.\textsuperscript{1,2} They perform these services outside the operating room (OR), on a consulting basis, and under substantial time pressure. The patient may be unknown to the anesthesiologist prior to the event. The cause can be trauma happening outside the hospital, rapid progression of medical or surgical disease, or iatrogenic complications of other treatments. Whatever the cause, the ability to rescue patients who are rapidly deteriorating is critical to hospital outcomes, and important enough to justify in-house anesthesiologist staffing at most medium or large facilities. Surprisingly, residency training devotes little instructional time to this topic; instead, the anesthesiologist is expected to assemble the necessary skills from experience in OR airway management, time spent in intensive care units (ICUs), and on-the-job training after graduation.
This article reviews sudden patient decompensation, the treatment principles for common events, and the basic facts and techniques that the anesthesiologist must master in order to cope with in-hospital emergencies.

**Pathophysiology of Sudden Instability**

Patients “crash” from a surprisingly small number of proximate causes: hypoxia, hypovolemia, cardiac dysfunction, or a neurologic event. Loss of ability to move oxygen through the lungs and into the bloodstream can occur suddenly, as can loss of ability to deliver oxygen to tissues. Other abrupt changes in patient condition can arise from failure of local oxygen delivery to the brain, resulting from stroke or increased intracranial pressure. Rare causes of sudden deterioration include seizures, sepsis, adverse effects of medication, and poisoning.

When called to assist with a suddenly deteriorating patient, the anesthesiologist should begin with the ABCs: airway, breathing, and circulation. While applying the basic facts and techniques that the anesthesiologist must master in order to cope with in-hospital emergencies.

<table>
<thead>
<tr>
<th>Table 1. Challenges in Emergency Intubation Outside the OR</th>
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<tr>
<td>Time pressure</td>
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<tr>
<td>Unknown anatomy and physiology</td>
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<tr>
<td>Full stomach</td>
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<tr>
<td>Lack of experienced assistants</td>
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<td>Lack of rescue equipment</td>
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<td>Difficult patient positioning</td>
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<td>Difficult access to the head of the bed</td>
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<td>Inability to pre-oxygenate</td>
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First Principle: Identify Mechanical Issues

Among the causes of sudden deterioration likely to lead to a preventable bad outcome are 2 mechanical issues. The first of these involves life-threatening anatomic abnormalities, cardiac tamponade, and tension pneumothorax. The second is iatrogenic errors in the delivery of medication. The experienced provider will consider the potential for either condition when assessing a patient who suddenly has become unstable, and maintain an index of suspicion for the complications until they are ruled out.

Cardiac tamponade and tension pneumothorax result from traumatic injury or iatrogenic damage during procedures in the chest or upper abdomen. Spontaneous medical disease can, in rare cases, cause either condition, although usually with a less acute course.

Tamponade presents with hypotension and tachycardia, and may progress rapidly to cardiac arrest. Tamponade can be caused by blunt or penetrating trauma, placement of a central line, or an errant subdiaphragmatic needle stick. Instability can be temporized with inotropic agents, and is dramatically worsened by any therapy—such as the induction of anesthesia—that reduces sympathetic tone.

In a trauma patient, a history of cardiac arrest that responds to cardiopulmonary resuscitation (CPR) and epinephrine with return of blood pressure is suggestive of tamponade. Patients who have exsanguinated typically will not respond to CPR. Echocardiography is the fastest and surest way to make the diagnosis. In the emergency department or OR, a witnessed cardiac arrest with a risk factor for tamponade—such as penetrating trauma anywhere in the thorax—should prompt an emergency thoracotomy.

Once the diagnosis of tamponade is made, treatment consists of opening or aspirating the pericardium. This step will enable the return of cardiac function, even following complete arrest, with long-term survival dependent on the duration of hypotension and the underlying cause of pericardial bleeding. Atrial injury is more likely to be repairable than ventricular injury, and lower-energy stab wounds or needle injuries more often are salvageable than high-energy gunshot wounds.

Tension pneumothorax also presents with hypotension. Hypoxia is common, but desaturation usually is a later and less specific sign than hemodynamic collapse. Pneumothorax should be suspected in the same circumstances as tamponade (ie, any thoracic trauma or procedure), and is a greater hazard after central-line placement or thoracic pain management procedures than tamponade.

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Diagnosis of tension pneumothorax depends on maintaining an index of suspicion. It is important to be aware that pneumothorax can develop as a secondary event due to interventions such as mechanical ventilation or chest compressions in a patient who is crashing for a different reason. Auscultation of the chest will reveal a difference in the timbre of breath sounds from the right to left fields of the lung, with “duller” sounds on the side with the pneumothorax. In cases of hemothorax, however, these findings will be reversed, with “sharper” sounds on the affected side.

If time allows, auscultatory abnormalities should lead to an urgent chest radiograph; hemodynamically significant pneumothorax or hemothorax will be obvious on the image. If the patient is deteriorating quickly, bilateral chest decompression using tube thoracostomy is...
Case Study

The anesthesiologist on call was summoned to the bedside of a 64-year-old man. The patient had undergone an uneventful right total hip arthroplasty, performed under epidural anesthesia and intravenous sedation, but had become acutely agitated and cyanotic. Measurement of arterial oxygen saturation was intermittent, due to patient movement, but the last recorded value was 88%. Noninvasive measurement of blood pressure (BP) was not possible; BP 30 minutes ago was 120/80 mm Hg. The patient had removed his electrocardiogram (ECG) leads and was attempting to get out of bed, but was being actively restrained by staff. A left internal jugular central line was in place but threatened by his movement.

Differential Diagnosis

Acute hypoxia is the most urgent cause of patient anxiety and in this situation could be caused by pulmonary embolus, pulmonary edema, airway obstruction or tension pneumothorax secondary to the presence of the central line. Other possibilities are severe pain, acute situational delirium (“sundowning”), toxic reaction to a pharmaceutical, acute psychosis or rare presentations of stroke or seizure.

Treatment

The best outcome will follow from immediate control of a situation that is dangerous for both staff and patient. Rapid sequence induction with a sedative and succinylcholine should be followed by 3 to 4 assisted breaths of 100% oxygen delivered by bag-valve-mask as the patient relaxes, and then endotracheal intubation. The dose of sedative administered—typically propofol or etomidate—should be decreased from normal in deference to the patient’s age and potential hemodynamic instability.

Following confirmation of exhaled carbon dioxide, the endotracheal tube should be secured and preparations made for ongoing mechanical ventilation, including patient transfer to the postanesthesia care unit or an intensive care unit. A more detailed medical history should be gathered, including any recent drug administration or other nursing event. Hemodynamic stability should be monitored closely, and further diagnostic assessments should be ordered. Depending on the patient’s response to intubation, these tests might include a 12-lead ECG, toxicology, chest radiograph, or computed tomography. The most likely and serious potential problem—pulmonary embolus—will respond only transiently to intubation; continued care may be required, up to and including advanced cardiac life support and pulmonary or systemic thrombolysis.

Airway and Breathing Management

Treatment of the crashing patient begins with a 1-second assessment of neurologic status and respiratory effort. Ask the patient, “How are you doing?” The answer (if any) will determine the next few steps. If the patient is comatose or unable to breathe, airway support and assisted ventilation with a bag-valve-mask system connected to 100% oxygen is indicated. The ability to perform this procedure is a fundamental skill of anesthesiologists and an important reason for inclusion on the team. An experienced provider will know in moments whether airflow is adequate.

If airflow is not occurring, the immediate priority becomes obtaining an adequate airway. Placement of an oral or nasopharyngeal airway is a temporizing maneuver that may allow oxygenation. Many institutions now include a laryngeal mask airway (or related device) in their crash cart or emergency equipment box. Although familiar to anesthesiologists as a tool for healthy elective surgery patients, experience in Europe and in prehospital care would suggest that supraglottic airway (SGA) devices are more useful in emergency airway management than previously recognized. An SGA can quickly and reliably be placed to enable positive pressure oxygenation. The relative risks for gastric distention or aspiration are trivial in the crashing patient with an obstructed airway. Furthermore, the ability to intubate the patient through the SGA has been enhanced in...
video laryngoscope to all off-floor intubations can make ventilation (PPV) generally will improve the condition of the patient, except in the presence of laryngospasm or ketamine that may still lower the endogenous catechol level, and by applying PPV with caution: low volumes, low pressure, no positive end-expiratory pressure in fragile patients. Ventilation also can convert a simple pneumothorax into a hemodynamically significant tension pneumothorax; this diagnosis should be considered in any patient with an obvious risk factor for thoracic injury. The final cause of instability following successful intubation is a pathophysiologic process that manifests as hypoxia or tachypnea but is not directly related to respiration. Such conditions might include hemorrhage, pulmonary embolus, myocardial infarction, or increased intracranial pressure. Whatever the case, airway support, up to and including intubation, is still indicated, because protection of the airway and control of ventilation will facilitate patient care regardless of the cause of instability.

### Hemorrhage and Resuscitation

Life-threatening hemorrhage occurs in 1 or more of 5 anatomic compartments: the thorax, the peritoneum, the pelvis, the long-bone compartments of the thigh, and the street. In most patients crashing from hemorrhage, the source of bleeding will be obvious. If not, the diagnostic steps of the Advanced Trauma Life Support course should be followed: chest radiograph to eliminate significant hemothorax, abdominal ultrasound to eliminate free intraperitoneal fluid, and physical examination to indicate instability of the pelvis or femurs. Bleeding outside of the body should be apparent by inspection. Although the use of early rapid computed tomography (CT) scans are gaining favor in trauma management—even in marginally unstable patients—the cause of bleeding is already known in most cases and clinicians must weigh the time required to obtain a CT study against a delay in achieving hemostasis.

Recommendations for resuscitation of the bleeding patient have undergone significant revision over the past 2 decades. The current concept and goals of hemostatic resuscitation are based on the newly recognized need for focused support of coagulation from the start of treatment (Table 2). The first principle is obvious: The sooner the source of hemorrhage is controlled, the better the patient will do. Control might be achieved by external compression of an open wound, application of a tourniquet, or by rapid transport to the OR or angiography suite. In many institutions, the anesthesiologist is in a unique position to facilitate this step, thanks to his or her local knowledge of how to activate OR resources and experience with the

<table>
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<tr>
<th>Table 2. Goals of Hemostatic Resuscitation in the Actively Bleeding Patient</th>
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<tr>
<td>Facilitate anatomic control of hemorrhage (expedite patient transfer to OR/angiography).</td>
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<tr>
<td>Intubate and anesthetize (gently!).</td>
</tr>
<tr>
<td>Maintain deliberate hypotension (systolic blood pressure 80-90 mm Hg).</td>
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<tr>
<td>Activate massive transfusion protocol.</td>
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<tr>
<td>Limit crystalloid infusion; resuscitate with red blood cells and plasma until cause and duration of bleeding is known.</td>
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<tr>
<td>Administer an antifibrinolytic (eg, tranexamic acid or aminocaproic acid) early in resuscitation.</td>
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essential logistics of moving unstable patients through the hospital.

Anesthesia treatment of bleeding once consisted of the rapid and continued administration of whatever fluids were closest at hand. This approach now is recognized as counterproductive. Rapid administration of fluid has the potential to raise blood pressure, increase hemorrhaging, and wash away fragile early clots. Furthermore, administration of non-blood products will dilute normal components of coagulation that already are depleted by hemorrhage.

The new understanding leads to 2 important recommendations: First, blood pressure should be maintained at a level below normal; and second, blood products—including plasma and platelets—should be administered as early as possible in the course of treatment. The exact ratio of red blood cells (RBCs) to plasma for optimal resuscitation is controversial, but the goal is to stay ahead of coagulopathy whenever possible. A general principle is the more rapid and undefined the hemorrhage, the closer the provider should aim for a 1:1 ratio of plasma to RBCs. Any hospital that expects to care for hemorrhaging patients should develop a massive transfusion protocol to ease the logistic burden of relaying products from the blood bank to the bedside. The transfusion protocol for a level 1 trauma center also will include provisions for delivery of type O RBCs to patients who have not been cross-matched, and for the rapid preparation and delivery of plasma.9

Early administration of an antifibrinolytic agent—tranexamic acid or aminocaproic acid (Amicar, Pfizer)—has been shown to improve survival from hemorrhagic shock in both trauma and non-trauma settings. This standard is emerging in military and civilian trauma care and is under study for other causes of hemorrhage.10 Development of IV procoagulant compounds is an active research area at present, but to date the evidence of efficacy is limited and the potential benefits of off-label use must be weighed against the risk for thromboembolic complications. Available compounds include cryoprecipitate, factor VIIa, prothrombin complex concentrates, and recombinant fibrinogen. Clinicians should know which of these are available in their institution, and have an understanding of when their use might be indicated. In general, the greater the patient’s risk for dying from hemorrhage, the more reasonable it is to use one of these agents.

**Cardiac Support**

Assessment of the circulation should follow airway management and control of hemorrhage in any unstable patient. Every anesthesiologist should be familiar with the basic principles of advanced cardiac life support (ACLS).11 The ACLS course, which teaches initial steps for management of patients in cardiac arrest, is designed to restore functional cardiac rhythm and perfusing blood pressure. Once these basic goals are accomplished, three broad considerations should guide further management.

**Rhythm Control**

The goal of rhythm control is to return the patient to a normal sinus cardiac rhythm as fast as possible. Urgency depends on the patient’s blood pressure. Defibrillation is indicated in any unstable patient. Chemical intervention with adenosine is appropriate in semi-stable patients with a rapid supraventricular tachycardia. Sedation may be indicated in conscious patients who require defibrillation but should be administered cautiously as cardiac output is already low.

**Fluid Volume Control**

Fluid volume status is a consideration in patients with hemodynamic instability. Bleeding should be diagnosed and managed as above, but this is not the only concern. Hyper- or hypovolemia may lead to cardiovascular compromise even in the absence of hemorrhage. In the acute setting, the experienced anesthesiologist will assess volume status through controlled experimentation: administration of a small dose of fluid or anesthetic and observing the patient’s response. Data from patient monitors may be of assistance; widened pulse variation and low central venous pressure are associated with hypovolemia. These monitors are even more useful when followed as trends over time in a given individual. Single snapshot values do not correlate as well, especially in the middle of a crisis. Transesophageal echocardiography is the most rapid and precise way to define the patient’s volume status; its use should be strongly considered in any patient with ongoing hemodynamic instability of uncertain origin.5

**Cardiac Function**

Cardiac function is the third major concern, especially in patients in whom the more treatable causes of hemodynamic instability have been excluded. Ability of the heart to generate adequate output may be impaired from chronic disease, an acute cardiac event, or from noncardiac factors such as sepsis, poisoning, or medication overdose. Anesthesiologists are not expert at managing coronary ischemia, but they should be able to diagnose this cause of instability. After immediate symptomatic therapy according to the ACLS, a 12-lead electrocardiogram should be obtained if ischemia or primary dysrhythmia is in the differential diagnosis. Ischemia should be addressed by early consultation with a cardiologist and then expedited patient transfer for angiography or thrombolytic therapy. Hemodynamically significant dysrhythmias should be managed in accordance with ACLS.

Impaired cardiac output resulting from noncardiac disease is treated symptomatically at first, with intubation (if indicated) and hemodynamic support. Norepinephrine is the pressor of choice for patients in septic shock, although vasopressin, epinephrine, and dopamine also may have a role.12 Potential sources of poisoning should be considered, and toxin-specific therapy initiated as necessary. The hospital pharmacy,
the emergency department, and the local poison control center all can provide advice.

**Neurologic Injury**

Stroke or seizure, in rare cases, may cause sudden hemodynamic instability. Emergency treatment is supportive, beginning with airway management and administration of oxygen. Any anesthetic induction agent will end seizure activity. In the fragile or unstable patient, it is better to induce anesthesia and intubate than to try to arrest the seizure while preserving spontaneous ventilation and an open airway. The post-ictal period following a grand mal seizure will delay recovery of normal neurologic function and may contribute to slow arousal after induction of anesthesia.

Diagnosis of stroke is based on both lateralizing neurologic deficits and CT imaging. If stroke is suspected, the anesthesiologist should take steps to facilitate early imaging, as optimal outcomes from thrombolytic therapy are highly time-dependent. Early intubation will improve patient safety during the scan and may help to expedite therapy.

Patients with quadriplegia or high paraplegia after injury to the cervical spine will require intubation within the first 24 hours. Even previously fit patients who appear to have good diaphragmatic function may experience sudden respiratory arrest resulting from ischemia of the ascending spine, atelectasis, and the inability to clear secretions. Emergency management is complicated by the need to protect the cervical spine from further injury during intubation. The recommended approach is an awake fiber-optic intubation if the patient is stable, or rapid sequence induction and video laryngoscopy with continuous manual inline cervical stabilization if the patient is hypoxic, hypotensive, or agitated.

**Rare Causes of Instability**

Poisoning, in various forms, can produce sudden decompensation. Polypharmacy related to pain medications, sedatives, and even drugs of abuse is a leading cause of respiratory arrest in the hospital. In addition to providing airway management and ventilatory support in this situation, the anesthesiologist should consider possible iatrogenic sources of arrest and should request a toxicology screen in any suspicious situation. Any patient-controlled analgesia pump or other drug-delivery device should be tested for proper functioning and programming.

Drug abuse also can lead to sudden psychotic debility with agitation and violent behavior, either as a direct effect or as withdrawal syndromes. When summoned to this kind of emergency, the first priority for the anesthesiologist should be protection from personal injury. The patient should not be approached until sufficient personnel are available to provide adequate restraint. Simple sedation may be effective management, but some patients will require anesthetic induction and intubation. There should be no hesitation to induce anesthesia if the patient is threatening harm to staff, other patients, or himself or herself.

**Conclusion**

Anesthesiologists provide an important safety net in the hospital through their ability to rescue patients from the brink of death. As a core skill, every anesthesiologist should understand how to diagnose and treat patients with sudden life-threatening conditions.

**References**