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SEVENTH EDITION

EMERGENCY MEDICINE

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TOP SECRETS IN EMERGENCY MEDICINE

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EMERGENCY MEDICINE SECRETS

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SEVENTH EDITION

EMERGENCY MEDICINE SECRETS

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To Peter Bakes, my prince and the love of my life. Without you, there is nothing. With you, we have built a life of love, laughter, and support. I am forever grateful that God brought us together, and I love you more with each passing day.

Katherine M. Bakes

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PREFACE

This book is for all students and practitioners of emergency medicine, both novice and experienced. Because emergency medicine continues to mature as a specialty and our daily practice of it continues to evolve, we have reorganized some of the chapters and added appropriate content to reflect these changes. With difficulty we have also selected the Top 100 Secrets from more than 300 submitted by the chapter authors and editors. We hope that this book continues to be a concise, valuable, and enjoyable method of imparting information and knowledge. Knowing some of the most important questions about a particular presentation or problem is the first step to obtaining the answers needed at the patient's bedside. However, medicine being both an art and a science is nothing if not humbling, and knowledge alone does not treat all that ails. Listen to your patients and make them feel heard. Treat them all with the care and empathy you would wish for any member of your family. Getting to the correct diagnosis can be and is invigorating, but positively impacting a life confirms our calling.

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Last but certainly not least, we thank our mentors and heroes – Dr. Vince Markovchick, Dr. Steve Cantrill, and Dr. Peter Pons – GIANTS of emergency medicine. They solidified emergency medicine's position in health care, created a storied residency at Denver General, and spent tireless hours building and defending our specialty, all while supporting those of us following after them. Like so many others, we are honored to stand on their shoulders. We would like to take this opportunity to recognize the impact on innumerable clinicians who benefited from their enormous wealth of knowledge and the countless individuals healed by their wisdom and gracious hearts. They have taught us the privilege of taking care of patients during their most vulnerable moments. They have collectively elevated our field in ways we can never repay. We have been fortunate to call them colleagues and friends. We are humbled by the impact of these great men, and we hope that we will do them proud in continuing to champion their belief that every patient deserves exceptional, empathetic, evidence-based emergency care. This book, and the field we have dedicated our lives to, would not have been possible without each one of them. Thank you.

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TOP 100 SECRETS

1. Emergency treatment of preeclampsia includes:
   - Magnesium sulfate for patients who present with preeclampsia with severe features.
   - For patients with sustained systolic blood pressure (SBP) >160 mmHg or diastolic blood pressure (DBP) >110 mmHg, antihypertensives should be used in consultation with an obstetrician.
   - Administer corticosteroids to patients between 24 and 34 weeks' gestation to enhance fetal lung maturity.

2. A woman does NOT have to be pregnant to have preeclampsia or eclampsia. Twenty percent of cases occur in the postpartum period, when the most common presenting symptoms include headache, shortness of breath, blurry vision, nausea or vomiting, edema, or epigastric pain.

3. Button or disc batteries that are retained in the esophagus require emergent removal. Even for just a few minutes, button batteries can cause significant burns and erosions through the entire esophageal wall, leading to potentially massive bleeding, perforation, fistula formation, mediastinitis, and death.

4. With overdoses of drugs that cause sodium channel blockade, give sodium bicarbonate for the treatment of dysrhythmias or prolongation of the QRS duration. If the QRS duration does not narrow after administration of sodium bicarbonate, give a second bolus. Hyperventilation should be initiated to induce a serum pH of 7.5–7.55. Hypertonic saline can also be administered in a dose of 200 mL of 7.5% solution or 400 mL of a 3% solution. In addition to sodium bicarbonate, patients with cardiovascular toxicity often require fluids and vasopressors for hypotension, benzodiazepines for seizures, and endotracheal intubation for altered mental status.

5. Through stimulation of the cerebral respiratory center, salicylate overdose causes an acute respiratory alkalosis, without hypoxia. If the patient is hypoxic, coingestion of a sedative or salicylate-induced noncardiogenic pulmonary edema should be considered. Within 12–24 hours after ingestion, the acid-base status in an untreated patient shifts toward an anion-gap metabolic acidosis as a result of accumulation of lactic acid and ketoacids. A mixed respiratory alkalosis and metabolic acidosis typically is seen in adults. In patients with respiratory acidosis, concomitant ingestion of a central nervous system (CNS) depressant should be suspected. Metabolic acidosis is the predominant acid-base disturbance in children, patients who ingest massive amounts of salicylates, hemodynamically unstable patients, and patients of all ages who have chronic salicylate toxicity.

6. Standard indications for hemodialysis in salicylate poisonings include persistent, refractory metabolic acidosis (arterial pH < 7.10), renal failure with oliguria, cardiopulmonary dysfunction (e.g., pulmonary edema, dysrhythmias, cardiac arrest), CNS deterioration (e.g., seizures, coma, cerebral edema), and a serum salicylate level greater than 100 mg/dL at 6 hours post-ingestion in the acute setting. Because ingestion of more than 300 mg/kg predicts severe toxicity, a nephrologist should be consulted early in anticipation of the possible need for dialysis.

7. The acetaminophen nomogram can only be used with an accurate estimate of the time of ingestion. Treat patients with a credible history of acetaminophen exposure and unknown ingestion time by checking a single acetaminophen level and liver enzymes; if the acetaminophen level is >20 µg/mL or hepatic transaminases are elevated, treat with *N*-acetylcysteine (NAC) for 12 hours and then repeat the laboratory tests. If acetaminophen is undetectable and liver function has improved, NAC can be halted; otherwise, continue NAC and contact the regional Poison Control Center (1-800-222-1222).

8. The Glasgow-Blatchford score (GBS) is a risk stratification tool applied to patients with acute non-variceal upper gastrointestinal (GI) bleeding. The GBS is calculated by totaling points assigned for laboratory and clinical risk markers (Table 36.1). A score >0 is 99.6% sensitive in predicting high-risk patients who will require clinical interventions, including blood transfusion, endoscopy, or surgery. Low-risk patients with a score of 0 are candidates for outpatient management.

9. Peritoneal fluid criteria for SBP are:
   - Neutrophil count >250 cells/mm^3
   - Positive Gram stain
   - Positive culture result (gold standard)

10. Autonomic dysreflexia/hyperreflexia is an abnormality of the autonomic nervous system seen in patients with long-standing cervical or high thoracic spinal cord lesions (e.g., patients with quadriplegia and high paraplegia). It is caused primarily by unchecked reflex sympathetic discharge secondary to visceral or somatic stimuli below the level of the spinal injury. This potentially life-threatening syndrome includes severe paroxysmal hypertension, diaphoresis, tachycardia or bradycardia, anxiety, headache, and flushing. Uncontrolled, it may result in seizures and coma. Morbidity has resulted from cerebrovascular events, subarachnoid hemorrhage, and respiratory arrest. One of the most common precipitating stimuli is overdistention of the bladder from a plugged or kinked catheter; therefore always evaluate catheter patency in this population.

11. The inappropriate excretion of salt and water after relief of urinary obstruction is called postobstruction diuresis. Patients with abnormal renal function or chronic urinary retention are most susceptible. A physiologic diuresis is normal, because the kidneys excrete the overload of solute and volume retained while the urinary system is obstructed. If urine output persists at high levels, significant fluid and electrolyte abnormalities may develop. Patients who exhibit a continuous diuresis after clinical euvolemia is reached often require hospitalization for hemodynamic monitoring and fluid and electrolyte repletion.

12. BRASH stands for **b**radycardia, **r**enal failure, **A**V nodal blockers, **s**hock, and **h**yperkalemia. These patients present with bradycardia and hyperkalemia in the setting of renal failure, acidosis, and typically, the use of an AV nodal blocking agent such as a β-blocker or calcium channel blocker. In BRASH, renal failure causes hyperkalemia and accumulation of the patient's nodal blocking agent, both of which worsen bradycardia. In a dangerous cycle, bradycardia then leads to shock, further worsening renal perfusion, and so on.

13. In hyponatremia, there has been much debate over how rapidly Na^+ should be corrected, ranging from 0.5 to 2.0 mEq/L/h. In most patients, if serum Na^+ is <120 mEq/L, serum Na^+ should be corrected slowly, rising by no more than 0.5 mEq/h. This approach avoids the possible development of central pontine myelinolysis (which is also called osmotic demyelinating syndrome by some purists), a catastrophic neurologic illness seen with too-rapid Na^+ correction: coma, flaccid paralysis, and usually death.

14. Treatment of hyperkalemia is based on the presence or absence of electrocardiogram (ECG) changes, serum levels, and the patient's underlying renal function. If the patient has life-threatening ECG changes of hyperkalemia (widening QRS complex, a sine wave–like rhythm or bradycardia/heart block), 10% calcium chloride should be given (10 mL, preferably through a central line) to temporarily stabilize the myocardial cell membranes. Although calcium is relatively fast-acting, its effect lasts only 30–60 minutes, requiring additional measures to lower K^+ levels. However, most patients with hyperkalemia will not have QRS widening, and only require K^+ moved intracellularly, and then removed from the body.

15. Kidney stones reaching the distal ureter are more likely to pass than those impacting proximally. Stones 2–4 mm pass 95% of the time; stones 4–6 mm pass 50% of the time, and stones >6 mm pass 10% of the time. When estimating stone size, the x-ray image is magnified; the actual size is 80% of what is measured on the films.

16. Two potentially reversible entities should be considered in a patient with renal failure and cardiac arrest:
   - *Hyperkalemia.* When a patient suffers an arrest from whatever cause, respiratory and metabolic acidosis and the efflux of potassium from cells can be expected to produce hyperkalemia secondarily. In the patient who already may have a tendency toward hyperkalemia, this further increase could cause the patient to be refractory to standard advanced cardiac life support (ACLS) interventions. Patients with chronic kidney disease (CKD) who are in cardiac arrest should receive intravenous (IV) calcium if they do not respond immediately to the first round of ACLS measures.

- *Acute pericardial tamponade.* Cardiac arrest may result from the accumulation of pericardial fluid or spontaneous bleeding into the pericardial sac, and patients with uremia are more susceptible. Patients with tamponade tend to display refractory hypotension, pulseless electrical activity, or both. Bedside ultrasound may be diagnostic, and emergency pericardiocentesis may be life-saving.

17. Patients with metformin-associated lactic acidosis (MALA) have a very high lactate and a low pH, out of proportion to clinical presentation. Chronic toxicity usually arises from patients who develop renal failure for some reason and continue to take therapeutic doses of their metformin. Because metformin is cleared by the kidneys, it starts to accumulate, resulting in a severe lactic acidosis. The mechanism for the lactic acidosis is unknown. The treatment is emergent dialysis for patients with renal failure and a severe acidosis.

18. In primary adrenal insufficiency, hyperkalemia may be present from lack of aldosterone as well as cortisol deficiency. Hyponatremia may be present from lack of aldosterone and the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). Cortisol is one of the counter-regulatory hormones that increase liver glucose production with fasting. In the setting of adrenal insufficiency, hypoglycemia may develop if the patient has not eaten. Anemia and an increase in eosinophils may be seen. Rarely, adrenal insufficiency causes hypercalcemia.

19. In alcoholic ketoacidosis (AKA), patients are in a redox state that favors the formation of lactate from pyruvate. Dextrose-containing fluids reduce fatty acid oxidation (thus limiting acetoacetate generation) and provide another source of adenosine triphosphate (ATP), shifting the nicotinamide adenine dinucleotide/reduced nicotinamide adenine dinucleotide (NAD^+/NADH) ratio to reduce lactate generation.

20. Isopropyl or rubbing alcohol is metabolized in the liver to acetone, which results in measurable ketonemia in the serum. Acetone is excreted by the kidney, resulting in ketonuria, and is exhaled through the lungs, giving the breath an acetone aroma. Because acetone is not acidic, isopropyl alcohol poisoning does not cause metabolic acidosis and is far less toxic than either methanol or ethylene glycol, although it may still result in depressed mental status, gastritis, or gastrointestinal bleeding.

21. Clinical manifestations of ethylene glycol and methanol overdose are often delayed 6–12 hours, causing symptoms only once sufficient quantities of the toxic metabolites accumulate. The delay in symptoms is even greater with concurrent ethanol intoxication, because ethanol stops or slows the rate of methanol and ethylene glycol metabolism.

22. Ventilation and oxygenation remain an essential component of adult cardiopulmonary resuscitation (CPR). While various ventilation and oxygenation strategies have been studied for many years, there is still incomplete evidence regarding optimal: type of delivery used (i.e., passive oxygen insufflation, bag mask ventilation (BVM), advanced airway placement); timing of advanced airway placement in relation to other interventions; or type of advanced airway device used (supraglottic airway [SGA] vs. endotracheal tube [ETT]). What is known to improve survival in adults experiencing nontraumatic out-of-hospital cardiac arrest (OHCA) is early high-quality CPR (and for VF/pVT – defibrillation). Therefore the tasks and timing of ventilation and oxygenation need to be integrated in a way that minimizes interruptions or quality of chest compressions.

23. Even when performed by experts, CPR provides only approximately 30% of normal blood flow to the brain and 10%–20% of normal blood flow to the heart. Blood flow to the heart occurs during the relaxation phase of CPR, whereas blood flow to the brain occurs during the compression phase of CPR. This is the foundation for the American Heart Association's recommended CPR "duty cycle" of 50% (the proportion of the compression/decompression cycle spent in compression).

24. Targeted temperature management (TTM) refers to induced core hypothermia (32°C–36°C) and active temperature control (i.e., prevention of hyperthermia) for approximately 24 hours in comatose OHCA survivors. Two landmark studies published in the early 2000s showed this intervention to be highly efficacious for improving neurologic function, and a study published in 2013 showed similar results to the original studies but no advantage of cooling to 33°C versus 36°C.

25. Passive apneic oxygenation is a technique for preventing hypoxia during rapid-sequence intubation (RSI), where a nasal cannula with high-flow oxygen is placed on the patient during

the preoxygenation phase of RSI and left in place throughout the intubation attempt. Studies have shown that despite apnea during paralysis, passive oxygenation can greatly extend the time it takes for a patient to become critically hypoxic. To prevent harm, care should be taken with pediatric patients, as appropriate flow rates are based on size.

26. The lack of a gag reflex is an unreliable marker for airway collapse, as up to 25% of the population lacks a gag reflex at baseline. Also, the presence of a gag reflex does not guarantee the ability to protect the airway.

27. Serum lactate concentration rises as cells convert to anaerobic metabolism and, thus, is commonly used as a marker to assess the extent of systemic hypoperfusion and tissue hypoxia. An elevated serum lactate concentration is an early marker of systemic hypoperfusion and often precedes overt changes in a patient's vital signs. Higher serum lactate concentrations are associated with higher morbidity and mortality, and a lactate concentration >4 mEq/L has been used to define those with the highest mortality rates. A decrease in serial serum lactate measurements, or lactate clearance, indicates effective shock resuscitation.

28. The systemic inflammatory response syndrome (SIRS) is classically defined by two or more of the following: temperature >38°C or <36°C, heart rate >90 beats per minute (bpm), respiratory rate >20 breaths per minute, or partial pressure of carbon dioxide <32 mmHg, serum white blood cell count >12,000 mm^3 or <4000 mm^3 or >10% band forms. The SIRS response contributes to distributive shock, as inflammation causes reduced systemic vascular resistance and increased capillary permeability. This definition, although standardized, is not specific for defining serious illness or shock. Although most commonly described in the context of sepsis, a systemic inflammatory response may result from a variety of noninfectious insults, including trauma, burns, pancreatitis, or overdose.

29. An intrauterine pregnancy (IUP) may be detectable as early as 4 weeks' gestational age by transvaginal ultrasound (6 weeks using transabdominal ultrasound). The discriminatory zone is the level of β-human chorionic gonadotropin (β-HCG) at which one would expect to see evidence of an IUP by ultrasound. Although this depends on the institution where the patient is being seen, it is typically at a β-HCG level of 1000–2000 mIU/mL by transvaginal ultrasound and 5000 mIU/mL by transabdominal ultrasound. A gestational sac is seen at approximately 4–5 weeks' gestational age, and cardiac activity can be measured as early as 6 weeks' gestational age.

30. A combination of ultrasound evaluations can be used to differentiate the etiology of shock in emergency department (ED) patients. There are multiple protocols used to perform resuscitative ultrasound. However, most include the following: a subcostal cardiac view to evaluate for pericardial effusion and tamponade; an inferior vena cava (IVC) view looking for greater than 50% collapse with inspiration, indicating low intravascular volume; a parasternal long-axis cardiac view to estimate overall left ventricular function; a parasternal short-axis view to evaluate right ventricular enlargement; an apical four-chamber cardiac view to estimate overall function and evaluate relative chamber size (right vs. left); a view of the hepatorenal recess to evaluate for free intraperitoneal fluid; a view of the pelvis and retrovesical area to evaluate for free intraperitoneal fluid; lung views bilaterally in the second intracostal space to rule out pneumothoraces; and finally, a view of the abdominal aorta to evaluate for aneurysm. Use of this systematic, goal-directed protocol in patients in the ED, who have undifferentiated nontraumatic hypotension, allows physicians to narrow their differential diagnosis sooner and gives them a more accurate impression of the class of shock and final diagnosis.

31. The confidence interval is the expected range of results in the study population. A 95% confidence interval means that if you repeated the study 100 times, the result would fall within the 95% confidence interval 95 of the 100 times. The confidence interval is a measure of precision, where a narrower interval is considered more precise and is associated with larger sample sizes. Confidence intervals provide more detail than *P* values, as they not only demonstrate statistical significance, but also help determine clinical significance. The wider the confidence interval, the more likely the study results are not clinically meaningful. Look at the upper and lower boundaries of the confidence interval and determine whether both values still would hold clinical significance. If only the upper boundary value would have significance, there may not be broad clinical benefit.

32. Clinical signs of child abuse (abdominal bruising or tenderness) are very specific, but not sensitive for identifying abdominal injuries. We recommend obtaining aspartate transaminase (AST) and alanine

transaminase (ALT) for all children with concern for physical abuse and a significant injury (like a long-bone fracture), and abdominal computed tomography (CT) for those with AST or ALT >80 IU/L, or for those with abdominal bruising, tenderness, distention, or with a report of abusive abdominal trauma. The yield of CT using this protocol is >20%, and outweighs the risk from radiation. AST and ALT normalize over hours or days, regardless of whether an injury is present, so rechecking these tests is not a good alternative. Ultrasound has limited sensitivity and should not be used in place of CT.

33. What is the sensitivity of noncontrast CT of the head for subarachnoid hemorrhage (SAH)?
There have been several studies that have shown that a negative CT completed within 6 hours of symptom onset has a 100% sensitivity to rule out SAH and that no further testing needs to be performed. However, the literature is consistent in demonstrating a decreasing sensitivity of head CT from headache onset time. A reasonable estimate is 95% within 24 hours, 80% at 48 hours, 70% at 72 hours, and 50% at 5 days.

34. Alteplase (also called tissue plasminogen activator [tPA]) is the only thrombolytic currently approved by the US Food and Drug Administration (FDA) for acute stroke. In 1995, the National Institute of Neurological Disorders and Stroke (NINDS) trial showed that tPA improved functional outcome (modified Rankin scale) at 3 months, if given within 3 hours of symptom onset, with a number needed to treat (NNT) of 6. In 2008, ECASS III showed similarly improved functional outcome within the 3- to 4.5-hour timeframe (NNT = 14). In all the stroke literature thus far, there has never been shown to be a mortality benefit for systemic administration of tPA in patients with suspected stroke. Recently, the PRISMS trial demonstrated that for patients with a National Institutes of Health Stroke Scale (NIHSS) <5, there was no benefit of tPA over aspirin alone.

35. The primary risk of tPA is systemic bleeding, particularly intracerebral hemorrhage (ICH). For the NINDS trial, ICH with tPA was 6.4% versus 0.6% for the non-treatment group, or a number needed to harm (NNH) of 17. For ECASS III, ICH occurred in 7.9% of the receiving tPA versus 3.5% in the placebo arm, absolute risk difference of 4.4% and NNH of 23. The SITS-ISTR has shown a similar rate of about 2% ICH in their extended time window of 4.5 hours since last normal. The factors that appear associated with increased risk of hemorrhage are older age, brain edema or mass effect on CT, and higher baseline stroke severity. Angioedema may occur in 1%–5% of patients.

36. The most common error in the management of meningitis is delaying administration of antibiotics until the lumbar puncture (LP) is done. If there is a clinical suspicion of bacterial meningitis, antibiotics should be administered promptly. Intravenous antibiotics given <2 hours before the LP (and ideally after blood and urine cultures are obtained) will not affect the results of the cerebrospinal fluid (CSF) analysis.

37. Compared with that of younger patients, elderly patients with acute abdominal pain have mortality rates that are six to eight times higher, with two times the surgery rates. Elderly patients have decreased pain perception and are more likely to have normal vital signs with significant intraabdominal pathology; 80% of older patients with a perforated ulcer do not have rigidity. In one study, 30% of older adults who had surgical abdominal pain did not show signs of either a fever or leukocytosis. These factors cause delays in diagnosis, higher perforation rates, and higher mortality rates in the elderly. Keep a broad differential diagnosis and consider the common disorders such as appendicitis and cholecystitis, but also diseases specific to older patients, such as diverticulitis, volvulus, mesenteric ischemia, abdominal aortic aneurysm, and carcinomas.

38. Children with simple febrile seizures should be evaluated and treated based on fever alone, because the concomitant presentation of the seizure does not increase the risk of serious bacterial illnesses above baseline.

39. There are several important differences between the adult and the pediatric airway that make airway problems more serious in children. The child's tongue is large and is the most common cause of airway obstruction in the obtunded child. The narrowest portion of the pediatric airway is at the cricoid ring, making obstruction with subglottic pathology more likely than in adults. The small size of the pediatric airway (approximately one-third the diameter of an adult's at birth) means that small changes in diameter cause significant increases in resistance. (Resistance is inversely related to the fourth power of the radius.) Higher oxygen consumption in children contributes to more rapid decrease in arterial oxygen levels after airway obstruction.

40. In children with croup, aerosolized epinephrine decreases airway obstruction. It is indicated for children with stridor at rest or increased work of breathing (e.g., tachypnea, retractions). Racemic epinephrine (0.5 mL of a 2.25% solution) is used most commonly, but L-epinephrine alone (5 mL of a 1:1000 solution to maximum of 5 mL) is equivalent. Maximal effect is seen within 30 minutes, with potential to return to increased work of breathing and stridor at rest within 3 hours. Patients with unlabored breathing and without resting stridor after 3 hours can be safely discharged home.

41. In children, maintenance fluids per hour are calculated based on weight in kilograms using the 4-2-1 rule: 4 mL/kg for the first 1–10 kg, an additional 2 mL/kg for the next 11–20 kg, and 1 mL/kg for every additional kg. For example, a 32-kg child should receive (4 mL/kg $\times$ 10 kg) + (2 mL/kg $\times$ 10 kg) + (1 mL/kg $\times$ 12 kg) = 72 mL/h of maintenance fluids.

42. Bilious emesis in a neonate could represent malrotation with midgut volvulus. Congenital malrotation of the midgut predisposes the bowel to twisting on itself, leading to bowel obstruction and vascular compromise, with bowel necrosis of the entire involved segment developing in as little as 2 hours. All neonates with bilious emesis, irrespective of their appearance, require emergent pediatric surgical consultation.

43. For a typical Kawasaki's disease diagnosis, in addition to having a fever for 5 days, children must display four of the five following features described by the CRASH mnemonic:
    - **C**onjunctivitis: bilateral, nonexudative, bulbar conjunctival injection
    - **R**ash: polymorphous, generalized rash
    - **A**denopathy: cervical node >1.5 cm
    - **S**trawberry tongue: pharyngeal erythema, or red and cracked lips
    - **H**ands and feet: erythema and swelling

44. The combination of a fever and petechiae in a child can arise with several emergent diseases, including meningococcemia, disseminated intravascular coagulation (DIC), Rocky Mountain spotted fever, pneumococcal bacteremia, or leukemia. A complete blood count (CBC), C-reactive protein (CRP), coagulation studies, and blood culture should be obtained on nearly all children with fever and petechiae. Well-appearing patients with normal laboratory values can be discharged with close follow-up. Children with a single petechial lesion or petechiae distributed only above the nipple after coughing or vomiting are less likely to have serious bacterial infections and do not require testing if they are well appearing.

45. Ketamine, a dissociative agent causing a trancelike cataleptic state, is a commonly used medication for pediatric procedural sedation and analgesia (PSA). It provides strong sedation, analgesia, and amnesia while maintaining cardiovascular stability and protective airway reflexes. Ketamine onset is within a few minutes IV and 5–10 minutes intramuscularly (IM). Ketamine can increase salivation; however, coadministration with an antisialagogue, such as atropine, is no longer recommended because it does not decrease adverse respiratory events. Recovery agitation or emergence phenomenon consisting of vivid dreams, hallucinations, or delirium may occur. Coadministration of midazolam has not been shown to reduce its occurrence, but can be used to treat severe emergence phenomenon that occurs in 1.4% of patients. These patients are typically older or have a history of psychiatric illness. Ondansetron has been shown to reduce recovery emesis associated with ketamine that occurs in approximately 8% of patients. Ketamine, although protective of airway reflexes, is associated with such airway or respiratory complications as oxygen desaturation or airway obstruction in 2.8%, transient apnea in 0.8%, and transient laryngospasm in 0.8%.

46. To determine ETT size in children between the ages of 1 and 8 years, the simple formula (age/4) + 4 can be used for uncuffed tube sizes, and (age/4) + 3.5 for cuffed tubes. More and more evidence suggests that cuffed tubes are preferred to prevent air leaks that can require risky tube changes and limit effective ventilation (particularly in children requiring high ventilator pressures). Cuff pressures must be checked with a manometer and should not exceed 20 cmH_2O, as even 30 minutes of higher pressures can lead to permanent airway injury.

47. The cricothyroid membrane is too small for an open cricothyrotomy in children under ~10 years old; needle cricothyrotomy is recommended for infants and children after failed endotracheal intubation, failed supraglottic airway placement, and ineffective bag-valve mask ventilation. This is a temporizing measure to provide some oxygenation while awaiting definitive operative intervention; ventilation is passive through the mouth, and not effectively provided through needle cricothyrotomy.

48. In neonatal resuscitation, compressions should be started if the heart rate is <60 bpm. The compressor should stand at the head of the warmer and the person delivering positive pressure ventilation (PPV) should stand to the side. Compressions should be delivered by encircling the baby's chest with the hands and placing thumbs side by side or on top of each other in the center of the sternum at the nipple line. Compress approximately one-third the anterior-posterior diameter of the chest at a rate of 90 compressions per minute. Coordinate compressions with ventilations by repeating out loud "one-and-two-and-three-and-breath." Allow recoil of the chest during the "ands." Increase the fraction of inspired oxygen (FiO_2) to 100% after the infant is intubated and compressions initiated. Recheck the heart rate using a cardiac monitor every 60 seconds, and stop compressions if the heart rate reaches 60 bpm. If the heart rate does not improve, prepare for umbilical vein catheter placement and epinephrine.

49. A prior history of suicide attempt is the single greatest predictive factor of suicide. Patients who continue to express suicidal ideation after an attempt are especially at risk for a subsequent attempt. Previous suicide attempts should always be inquired about, as these patients are 100 times more likely to die from suicide than the general public. The risk of completed suicide is much higher in the first year after an attempt, particularly for people >45 years of age.

50. If a patient has an afferent pupillary defect (APD; Marcus Gunn pupil), it confirms damage to the retina or optic nerve. To perform the swinging flashlight test, shine a light toward the normal eye, and after several seconds swing it to the other eye. After a brief pupillary constriction in the abnormal eye, the redilation in response to light reflects afferent deprivation; this response may only be appreciated in a dark room.

51. In a patient on a ventilator with acutely worsening oxygenation or ventilation, first remove the patient from the ventilator and provide manual ventilation to the patient by using a bag ventilatory device. The **DOPE** mnemonic taught in pediatric life support can be helpful in remembering the remainder of the approach:
    - ***D****isplacement.* Confirm that the ETT is in the proper place by using some combination of auscultation, measurement of CO_2 exchange, radiography, and direct visualization.
    - ***O****bstruction.* Confirm that the ETT is patent by passing a suction catheter down the lumen. Sometimes an ETT can become kinked simply as a result of patient positioning.
    - ***P****atient.* Consider various causes within the patient. First and foremost is the possibility of secretions obstructing large bronchi. Vigorous suctioning may remedy this situation. Next, consider pneumothorax. Confirm that there is no evidence of barotrauma, usually by a combination of physical examination and a chest radiograph.
    - ***E****quipment.* Confirm that the ventilator circuit and ventilator itself are functioning properly.

52. Massive transfusion is defined as the transfusion of 10 units of red cells in 24 hours or more than three units over an hour in a patient with ongoing blood loss. A massive transfusion protocol (MTP) is the delivery of a prespecified ratio of blood and blood products (platelets and fresh frozen plasma). Blood that is universally compatible is Type O (Rh− should be reserved for childbearing-aged females; otherwise, use Rh+). Blood should be transfused in a 1:1:1 ratio (1 unit of blood, 1 unit of platelets, and 1 unit of fresh frozen plasma, infused at the same time if possible). In practice, a cooler for trauma may contain six units of packed red blood cells (PRBCs), 4–6 units of thawed plasma, and six units of pooled platelets, or one apheresis unit. Tranexamic acid (TXA) is an antifibrinolytic agent often transfused with MTP, as it may decrease mortality when given within 3 hours of injury. MTP is activated, based on obvious severe hemorrhage, or a combination of factors that predict the need for significant blood transfusion. While clinical judgment may be used, objective scoring systems may be best to activate the protocol. The ABC scale is a scoring system that gives points for penetrating mechanism, hypotension, tachycardia, and a positive focused assessment with sonography for trauma (FAST). Scoring ≥2 predicts increasing likelihood to require the protocol. Using the shock index (HR/SBP) ≥1 is also common.

53. Cardiovascular collapse may occur during intubation in patients that are elderly, hypotensive, or hemorrhaging. In these high-risk patients, consider lower dose induction agents, ketamine instead of opioids or etomidate, blood prior to intubation (when safe), and push-dose peri-intubation pressors.

54. If a patient sustains trauma to the orbit resulting in a retrobulbar hematoma, the buildup of pressure behind the globe can lead to ischemia of the optic nerve and retina, and permanent blindness

in as little as 90 minutes after injury. The ocular compartment syndrome can present as proptosis, impaired extraocular movement, decreased vision, and increased intraocular pressure; retrobulbar hematoma can by confirmed by CT scan. In order to prevent optic nerve or retinal ischemia, a lateral canthotomy is performed by incising the lateral canthal ligaments of the orbit to relieve intraocular pressure, in conjunction with emergent ophthalmology consultation.

55. Blunt cerebrovascular injury (BCVI) to the carotid or vertebral artery is found in nearly 0.5% of all blunt craniomaxillary facial trauma patients, and may cause significant morbidity if left untreated. These injuries may initially be silent, without focal neurologic deficits noted. Computed tomography angiography (CTA) should be performed in patients suspected of having a BCVI based on signs or symptoms, or focal neurologic deficits. High-risk patients that should be screened include those with a Le Fort II or III fractures, mandibular fractures, certain cervical spine fracture patterns (subluxation, fractures extending into the transverse foramen, vertebral body fractures of C1, C2, or C3), basilar skull fracture with carotid canal involvement, traumatic brain injury with Glasgow Coma Scale (GCS) scores <6, near-hangings with anoxic brain injury, or seat belt sign of the neck.

56. All patients with nasal trauma and suspicion of a nasal fracture require inspection of the nasal septum for a septal hematoma. This is a collection of blood between the mucoperichondrium and the cartilage of the septum. It appears as a grapelike swelling over the nasal septum. If left undrained, it may result in septal abscess, necrosis of the nasal cartilage, and permanent saddle nose deformity. If a septal hematoma is identified, incision and drainage, with suction and irrigation, is indicated in the ED. This is followed by nasal packing (similar to epistaxis), antistaphylococcal antibiotics (prophylaxis for toxic shock syndrome), and urgent follow-up.

57. Alkali burns (fertilizers, drain cleaners, airbag propellants, cement and plaster components, and some detergents) are more dangerous than acid burns, quickly penetrating ocular tissues through liquefactive necrosis. Acid burns (car batteries, refrigerants, and pool cleaners) cause coagulation necrosis, with depth of penetration limited by eschar formation. Following irrigation, perform a slit lamp and fluorescein examination, evaluating for extent of corneal damage and perforation. If perforation is excluded, measure intraocular pressure, as pressures can spike following chemical injuries. Obtain emergent consultation for severe exposures (pH <2 or >12), severe injuries (corneal perforation or ischemia), or elevated intraocular pressure.

58. Entrapment of the inferior rectus muscle stimulates the oculocardiac reflex, vagal stimulation from ciliary ganglion via the trigeminal nerve, which can cause severe sinus bradycardia, heart block, hemodynamic collapse, asystole, and death. The reflex is exacerbated by strain on the entrapped inferior rectus muscle. Having the patient look down can relieve some of this stimulus. Atropine may be effective in cases of oculocardiac reflex–induced bradycardia. In severe cases, emergent operative intervention is indicated to release the entrapped muscle.

59. Any hemodynamically unstable patient with ongoing chest hemorrhage should be emergently taken to the operating room. In the hemodynamically stable patient having sustained blunt trauma, immediate drainage of 1500 mL of blood with ongoing bleeding is an indication for operative intervention. For penetrating trauma, surgical intervention is indicated after immediate drainage of 1000 mL of blood with ongoing bleeding. Finally, drainage greater than 200 mL/h of gross blood for more than 2 hours is an indication for exploration, mechanism of injury notwithstanding.

60. An ECG should be performed on any patient with suspected blunt cardiac injury (BCI). The most common ECG findings after BCI are sinus tachycardia or premature contractions. ECG alone does not rule out BCI. However, a normal ECG combined with a normal troponin has a negative predictive value of 100%. If an ECG shows a new dysrhythmia, new heart block, or ischemic changes, the patient should be admitted for continuous cardiac monitoring. Echocardiography (ECHO) should be performed on any symptomatic patient or any patient with any ischemic changes on ECG, new dysrhythmias, or hypotension.

61. Neurogenic shock occurs after cervical or high thoracic injuries cause impairment of sympathetic stimulation to the heart and peripheral vasculature. Signs and symptoms include vasodilatation resulting in flushing, warm extremities, hypotension, and bradycardia (i.e., absence of compensatory tachycardia). Neurogenic shock is treated with volume resuscitation and peripheral vasoconstrictors, such as dopamine or norepinephrine.

62. In pregnant patients with abdominal trauma, any viable (>23–24 weeks' gestation) fetus requires continuous electronic fetal monitoring (EFM). EFM is recommended even for patients without external evidence of trauma, because it has been well documented that these patients are at risk of placental abruption. Current guidelines suggest that these patients be observed for a minimum of 4 hours with a cardiotocograph. If any abnormalities are discovered, including contractions, amniotic membrane rupture, vaginal bleeding, serious maternal injury, significant abdominal pain, and concerning fetal HR variability, the patient should be hospitalized and monitored for 24 hours.

63. Children with neck pain, midline bony tenderness, decreased range of motion, torticollis, altered mental status (GCS score <14), focal neurologic abnormality, predisposing conditions, or transient spinal cord symptoms should have their cervical spine imaged. Plain radiographs (anteroposterior [AP], lateral, and odontoid views) are preferred for most patients. Patients with abnormal plain radiographs or concerning physical examination findings should undergo CT or MRI, and often require both.

64. Asymptomatic microscopic hematuria in adults is not a sensitive predictor of significant urologic injuries in patients with blunt trauma mechanisms, and the amount of blood in the urine does not correlate with the severity of injury. The relatively low incidence of positive studies requiring surgery does not justify an extensive radiographic evaluation. Repeat urinalysis is justified, with advanced imaging for persisting hematuria.

65. It is difficult to predict which burn patients will develop laryngeal edema and airway obstruction. Concerning history includes an enclosed space burn. Additionally, there is an increased risk with larger burns, typically greater than 40%. Physical examination findings suggestive of airway injury include soot around the mouth or nares, hoarseness or stridor, facial burns, or carbonaceous sputum. Since not all patients with carbonaceous sputum, oral or nasal soot, and/or facial burns have significant inhalation injury, clinical judgment must be used to decide on the need for intubation. Stridor, increased work of breathing, and hypoxemia are more reliable indicators of significant inhalational injury. However, decreased oxygen saturation occurs late in patients with significant airway injury. Patients who present with facial burns from smoking on oxygen have a low risk of significant inhalation injury, and normally have a baseline oxygen requirement.

66. To transport amputated body parts:
    - Remove gross contamination with saline irrigation.
    - Wrap the part in a saline-moistened (not soaked) sterile gauze.
    - Place the wrapped part into a sealed plastic bag or container.
    - Place the bag or container into an ice water bath.
    - Never put the amputated part directly onto ice or immerse it in disinfection solution.

67. The ankle-brachial index (ABI) is calculated by dividing the Doppler systolic arterial pressure measured in the injured leg by the pressure measured in an uninjured arm. An ABI value >0.9 is considered normal. The ABI measurement may be inaccurate in patients with risk factors for peripheral arterial disease, such as diabetes and hypertension. Vessel calcification in the elderly can also increase the risk of a false-positive result. An ABI <0.9 should trigger further diagnostic workup or surgical exploration in the operating room.

68. Fractures involving the physeal zone may result in growth disturbance, and parents must be informed accordingly. About 80% of these injuries are Salter-Harris types I and II, both of which have a low complication rate. Salter-Harris types III, IV, and V injuries have a worse prognosis. Displaced Salter-Harris types III and IV fractures may require open reduction to restore the normal anatomic physeal relationship. The five types of fracture according to the Salter-Harris classification are (Fig. 93.5):
    - Type I: physeal separation; this may appear as a widening of the radiolucent area representing the growth plate.
    - Type II: fracture traverses the physis and exits on the metaphyseal side.
    - Type III: fracture traverses the physis and exits on the epiphyseal side.
    - Type IV: fracture traverses through epiphysis, physis, and metaphysis.
    - Type V: physeal crush injury; this may be difficult to determine on plain radiographs.

69. The tumor lysis syndrome (TLS) is characterized by hyperkalemia, hyperphosphatemia, hypocalcemia, and hyperuricemia. TLS is commonly seen in cancer patients with a large tumor burden,

such as leukemia or lymphoma. Additional risk factors include baseline kidney injury and dehydration. Although it can occur spontaneously, it is more common in the week following treatment. Treatment is targeted at reversing metabolic abnormalities. Those with significant kidney injury may require dialysis.

70. The pulmonary embolism rule-out criteria (PERC) is used in patients with a low gestalt clinical suspicion for pulmonary embolus (PE). If all criteria are met, the patient has a less than 2% risk of PE, and further workup is not indicated. Patients with clinical suspicion who do not meet all criteria may require further evaluation. In patients with moderate or high clinical suspicion for PE, the PERC rule does not exclude PE.

71. D-dimer, a degradation product of cross-linked fibrin, is found in increased levels of the circulation of patients with acute venous thromboembolism (VTE). There are different assays available for measuring D-dimer with different sensitivities and specificities, including enzyme-linked immunosorbent assay (ELISA), rapid ELISA, turbidimetric assay and whole-blood agglutination D-dimer assay. Different tests can report results as either fibrin equivalent units or D-dimer units. Older latex agglutination tests cannot be used in these algorithms because of poor negative predictive values. Although useful in ruling out VTE disease in select populations, owing to a lack of specificity, D-dimer has not proven useful at ruling in the diagnosis.

72. In patients older than 50 years, with low to low-moderate pretest probability for VTE, PE can be ruled out with an age-adjusted D-dimer. For D-dimer assays using fibrin equivalent units, use a cut-off of age $\times$ 10 μg/L. For assays using D-dimer units, use age $\times$ 5 μg/L.

73. Platelet transfusion should be delayed in idiopathic thrombocytopenic purpura (ITP) and thrombotic thrombocytopenic purpura (TTP) to avoid disease-specific complications and alloimmunization. It is more commonly indicated for primary bone marrow problems. Each bag of random donor platelets raises the platelet count ~5000/mL. They are usually ordered six at a time.
    - Platelet count >50,000/mL – hemorrhage unlikely.
    - Platelet count 10,000–50,000/mL – variable risk of bleeding with trauma, ulcer, and invasive procedures. Choosing when to transfuse at these levels is not an exact science.
    - Platelet count <10,000 – platelet transfusion is indicated because there is a significant risk of spontaneous hemorrhage.

74. Incomplete cord syndromes are as follows:
    - Anterior cord syndrome results in loss of function in the anterior two-thirds of the spinal cord from damage to the corticospinal and spinothalamic pathways. Findings include loss of voluntary motor function as well as pain and temperature sensation below the level of the injury, with preservation of the posterior column functions of proprioception, pressure, and vibration. The key issue is the potential reversibility of this lesion if a compressing hematoma or disk fragment can be removed. This condition requires immediate neurosurgical evaluation.
    - Central cord syndrome results from injury to the central portion of the spinal cord. Because more proximal innervation is placed centrally within the cord, this lesion results in greater involvement of the upper extremities than of the lower extremities. Bowel or bladder control is usually preserved. The mechanism of injury is hyperextension of a cervical spine with a cord space narrowed by congenital variation, degenerative spurring, or hypertrophic ligaments. This syndrome can occur without actual fracture or ligamentous disruption.
    - Brown-Séquard syndrome is an injury to a hemisection of the spinal cord, usually from penetrating trauma. Contralateral sensation of pain and temperature is lost, and motor and posterior column functions are absent on the side of the injury.
    - Cauda equina syndrome is an injury to the lumbar, sacral, and coccygeal nerve roots, causing a peripheral nerve injury. There can be motor and sensory loss in the lower extremities, bowel and bladder dysfunction, and loss of pain sensation at the perineum (saddle anesthesia).

75. Typical synovial fluid counts in septic arthritis are >50,000 white blood cells (WBCs)/mm^3, with predominantly polymorphonuclear neutrophilic (PMN) WBCs, and a Gram stain positive for bacteria. However, some patients with septic arthritis have synovial fluid counts <50,000 cells/mm^3, particularly those with prosthetic joints. Synovial fluid lactate levels >10 mmol/L are highly suggestive of septic arthritis, whereas levels lactate levels <4.3 mmol/L make septic arthritis very unlikely.

76. The rash of erythema multiforme is morbilliform and usually on the palms and soles, resulting from viral/bacterial infections or medications. Erythema migrans is a bull's-eye rash that usually expands (rather than migrates), and is a common manifestation of early Lyme disease. Erythema marginatum, part of the Jones' criteria for diagnosis of rheumatic fever, is an evanescent, nonpruritic rash that typically involves the trunk and extremities while sparing the face. Erythema nodosum (EN) is an acute inflammatory reaction leading to appearance of red, painful, tender nodules on the lower legs; etiologies are diverse and often idiopathic.

77. The mainstay of therapy for nerve agents is atropine, combined with decontamination, pralidoxime, and benzodiazepines (for seizure).

78. The most appropriate course of action for a patient with both a radiation exposure and associated major trauma or medical issues is to care for life-threatening trauma or medical problems first; these life threats always take precedence over management of the radiologic exposure.

79. Infection causes the vast majority of fevers, but the following other causes are included in the differential diagnosis:
    - Neoplastic diseases (e.g., leukemia, lymphoma, or solid tumors)
    - Autoimmune disease (e.g., giant cell arteritis, polyarteritis nodosa, systemic lupus erythematosus, or rheumatoid arthritis)
    - Endocrine disorders (e.g., thyroid storm)
    - CNS lesions (e.g., stroke, intracranial bleed, or trauma)
    - Illicit drug use (e.g., cocaine, 3,4-methylenedioxymethamphetamine [MDMA, ecstasy], or other methamphetamines)
    - Withdrawal syndromes (e.g., delirium tremens or benzodiazepine withdrawal)

80. Life-threatening causes of acute chest pain include:
    - Acute coronary syndrome (ACS; unstable angina and myocardial infarction)
    - Pulmonary embolism (PE)
    - Pneumothorax
    - Aortic dissection
    - Pericarditis
    - Myocarditis
    - Cardiac tamponade
    - Mediastinitis/esophageal rupture
    - Trauma

81. The American College of Cardiology Foundation/American Heart Association (ACCF/AHA) guidelines suggest that the most important factor in predicting acute coronary syndrome in a patient with chest pain is the history of present illness, rather than cardiac risk. Classic risk factors for cardiac ischemia have had very limited utility in the ED setting when trying to determine the immediate risk of ACS. Nevertheless, various clinical decision rules have been developed to generate a risk profile for coronary ischemia in patients presenting with chest pain. The **HEART** score is perhaps the most useful to date: **H**istory, **E**CG, **A**ge, **R**isk factors, and **T**roponin. The low-risk cohort was associated with 0.99% risk of an adverse cardiac event in a retrospective study and 1.7% in a prospective study. When combined with a repeat troponin 3 hours later to form the HEART pathway, the HEART score can identify patients in a low-risk cohort who may be safely discharged with outpatient follow-up. Clinical decision rules are no substitute for physician judgment. Clinical gestalt can help identify cases that would otherwise be missed by clinical decision rules.

82. Factors associated with failure to diagnose acute myocardial infarction (MI) (1%–2% of ED chest pain patients) include:
    - Young age group
    - Non-White race
    - Failure to obtain an accurate history
    - Incorrect interpretation of the ECG
    - Failure to recognize atypical presentations
    - Hesitance to admit patients with vague symptoms
    - Reliance on laboratory assays, such as cardiac enzymes
    - Insufficient experience or training

83. Improvement in headache with sumatriptan or ketorolac does not mean that the diagnosis is migraine. Because the final common pathway for most pain in the head is limited, and vasogenic inflammation probably plays a role, the response to any analgesic or antimigraine medication is of no etiologic significance. This includes triptans, which have been documented to improve the headaches of patients with SAH and cervical artery dissections.

84. Cardiac causes of syncope comprise the riskiest group of patients and include ACS, PE, physical aortic outflow obstructions (e.g., hypertrophic obstructive cardiomyopathy (HOCUM), aortic stenosis, or atrial myxoma), slow rhythms such as sick sinus syndrome, and tachyarrhythmias. Brugada syndrome, preexcitation, arrythmogenic right ventricular dysplasia (ARVD), and long QT syndrome can precipitate lethal dysrhythmias.

85. Suspect necrotizing fasciitis in patients with severe pain and tenderness that is out of proportion to the degree of visible cellulitis. Feel for crepitus, sometimes also appreciated on plain radiographs. Outline the area of visible infection to monitor for rapidly expanding signs of infection. CT or MRI can help evaluate the extent of the disease. Patients may appear septic, but this can occur later in the course of the disease.

86. Involvement of at least two of the following are needed to diagnose anaphylaxis:
    - Cutaneous manifestations (urticaria or rash)
    - Mucous membrane involvement (angioedema)
    - Upper respiratory tract involvement (edema and hypersecretion)
    - Lower respiratory tract involvement (bronchoconstriction)
    - GI symptoms (nausea, vomiting, or abdominal cramping)
    - Cardiovascular system effects (tachycardia, hypotension, and cardiovascular collapse)

    Hypotension in a patient who is exposed to a known allergen is adequate to make the diagnosis of anaphylaxis.

87. Initial treatment for life-threatening anaphylaxis includes:
    - Upper airway obstruction with stridor and edema is treated with high-flow nebulized oxygen, racemic epinephrine, and IV epinephrine. If airway obstruction is severe or increases, perform endotracheal intubation or cricothyroidotomy.
    - Acute bronchospasm is treated with epinephrine. Mild to moderate wheezing in patients with normal blood pressure may be treated with 0.01 mg/kg of 1:1000 epinephrine administered IM. If the patient is in severe respiratory distress or has a silent chest, administer IV epinephrine via a drip infusion: 1 mg of epinephrine in 250 mL of dextrose 5% in water (D5W) at an initial rate of 1 μg/min, with titration to desired effect. Bronchospasm refractory to epinephrine may respond to a nebulized β-agonist, such as albuterol or metaproterenol.
    - Cardiovascular collapse presenting with hypotension is treated with a constant infusion of epinephrine, titrating the rate to attain a systolic blood pressure of 100 mmHg or mean arterial pressure of 80 mmHg.
    - For patients in full cardiac arrest, administer 1:10,000 epinephrine, 1 mg slow IV push, or 2.0–2.5 mg epinephrine diluted in 10 mL NS via an ETT. Immediate endotracheal intubation or cricothyroidotomy should be performed as needed to secure the airway.

88. While early goal-directed therapy (EGDT) as a protocol-driven strategy is no longer recommended by the Surviving Sepsis Campaign guidelines, the general principles still apply. For example, multiple studies have shown an association between early antibiotics and improved outcomes. However, it is essential to note that source control often requires more than just appropriate antimicrobial coverage, and may require procedural intervention in some cases. Other studies have shown an association of adherence to the 3-hour sepsis "bundle" with improved outcomes.

89. Sepsis is now defined as a life-threatening organ dysfunction caused by a dysregulated host response to infection. There are two working definitions for the diagnosis of sepsis in clinical use. The first, and older definition, is the combination of SIRS criteria with suspicion for an infectious source. The second, recommended by the Surviving Sepsis Campaign, and published in 2016, is two out of the three qSOFA (quick sequential organ failure assessment) criteria (see Chapter 50).

90. Empiric antibiotics should NOT be used in well-appearing immunocompetent patients with infectious diarrhea. The treatment of certain bacterial pathogens with antibiotics may decrease duration

of symptoms by one day. However, the benefits are typically outweighed by risk of increasing antibiotic resistance and risk of hemolytic-uremic syndrome (HUS) with antibiotic-induced lysis of Shiga toxin–producing *Escherichia coli* (STEC)-releasing endotoxins. Empiric antibiotics may be considered in the following patient populations:

- Infants <3 months old with suspected bacterial source.
- Ill-appearing patients with dysentery.
- Patients who have travelled internationally with fever or sepsis.

Antimicrobial choice: First-line treatment is a single oral dose of ciprofloxacin 2 g or 3-day regimen of ciprofloxacin 500 mg twice daily. Alternative regimen (for children and pregnant women) is azithromycin 1 g (5–10 mg/kg) orally, as a single dose. In areas where amebiasis and giardiasis are endemic, consider metronidazole 500–750 mg orally three times daily for 10 days for amebiasis or 250 mg orally three times daily for 5 days for giardiasis (Fig. 53.1).

91. For treatment of acute pulmonary edema, follow the ABCs (airway, breathing, and circulation). In severe hypoxia, airway and breathing may be compromised, requiring intubation. The use of noninvasive mask continuous or bilevel positive airway pressure (CPAP/BiPAP) has decreased the need for intubation in patients with pulmonary edema; however, this has not significantly affected in-hospital mortality. Intubation may also be avoided with prompt medical treatment (see later). Patient symptoms can be dynamic, requiring frequent reevaluations for further medical management. Administer oxygen to maintain sufficient oxygen saturation (>90%), either by nasal cannula or nonrebreather mask, CPAP, or BiPAP. Continuously monitor oxygen saturation with pulse oximetry.

92. Drug therapy for acute pulmonary edema is aimed at decreasing preload. Nitrates are first-line drugs and are useful in the form of sublingual nitroglycerin (NTG) or an IV NTG drip. NTG is predominantly a venodilator, reducing preload. However, NTG also dilates coronary arteries, and may be especially helpful in the setting of coronary artery disease. Diuretics should only be administered to patients with signs of obvious fluid overload (e.g., peripheral edema, elevated jugular venous distention). When indicated, furosemide is given as a 40-mg IV bolus (larger amounts if the patient is already taking diuretic medication, although high-dose diuretics are associated with worse outcomes). Initially, within 5–15 minutes of the injection, venodilation occurs, although this is of limited clinical benefit in the setting of nitrates. This action is followed within 30 minutes by diuresis.

93. The most common presenting symptom in patients with acute ischemic heart disease is chest pain. Typically, ischemic pain is described as heaviness, pressure, tightness, or squeezing. It is less commonly described as sharp or aching. Usually the pain lasts minutes, as opposed to seconds or hours. Patients may describe their pain as indigestion, which can lead to the misdiagnosis of reflux esophagitis. Although precordial discomfort is most common, it may be felt anywhere on the chest, the right or left shoulder or arm, the throat or jaw, or the upper abdomen. The pain often radiates to one of these locations as well. Less commonly, patients may simply have pain in other more unusual locations (e.g., left ear or upper back). Associated symptoms include dyspnea, nausea, vomiting, diaphoresis, lightheadedness or syncope, palpitations, or malaise. Diaphoresis and vomiting, in particular, increase the likelihood of ACS. One-third of patients may not experience pain and just present with associated symptoms – in this case, it is termed an anginal equivalent or atypical angina. The most common symptom is shortness of breath. This is more common in the elderly, women, and patients with diabetes.

94. ST elevation caused by infarction may display one or more of the following:
    - *Morphology.* Convex upward ST elevation ("tombstone" morphology) is highly predictive of injury. However, the morphology may also be concave upward or horizontal.
    - *Reciprocal changes.* The presence of ST depression in another area of the ECG is highly predictive of injury, but is not always present.
    - ST elevation caused by injury is dynamic; serial ECGs usually show changes over time.

95. Hypertensive emergency is characterized by severely elevated blood pressure (BP; typically >220/120 mmHg) with acute end-organ (brain, heart, kidneys) damage. The absolute BP is not a criterion. The terms "malignant hypertension (HTN)" and "accelerated HTN" are used in the International Classification of Diseases, 10th edition (ICD-10). Examples include:
    - Hypertensive encephalopathy
    - Ischemic and hemorrhagic stroke

- Subarachnoid hemorrhage (SAH)
- Acute myocardial infarction (AMI)
- Decompensated congestive heart failure (CHF) with pulmonary edema
- Aortic dissection
- Preeclampsia/eclampsia
- Acute kidney injury (AKI): a controversial topic that requires special consideration, as isolated elevations in creatinine may reflect chronic kidney disease (CKD) rather than an acute process.

96. Most patients with abdominal aortic aneurysms (AAAs) are asymptomatic, and the aneurysm is found incidentally. Patients with symptoms of acutely expanding AAA may have gradually increasing abdominal pain, low back pain, or flank pain radiating to the groin. It is often described as dull, throbbing, or colicky.
    - 3% of men >50 years old have an occult AAA.
    - 75% of aneurysms are >5 cm and can be palpated.
    - 5%–10% of patients with an AAA have an abdominal bruit.

97. Patients with thoracic aortic dissections experience a sudden onset (85%) of severe chest pain with radiation to the jaw, neck, or back in the intrascapular region. Clinical patterns include:
    - Pain is most severe at onset and is often described as sharp, ripping, or tearing in quality.
    - Pain starts in one region and moves to another (abdomen to chest or chest to abdomen).
    - The patient may have nausea, vomiting, diaphoresis, lightheadedness, and apprehension or a sense of impending doom.
    - Syncope can also be the presenting complaint, and in some cases may be the only symptom.
    - Proximal dissections may cause aortic regurgitation and pericardial effusion with tamponade (16%).
    - Occlusion of aortic branches may cause AMI (coronary artery involvement); stroke (carotid or vertebral artery involvement); or paresthesias and arm pain (subclavian artery involvement), which may be suggested by unequal BPs or pulses.
    - Spinal artery occlusion can cause neurologic compromise.
    - Hoarseness may result from recurrent laryngeal nerve compression.
    - Chest pain unrelieved by large doses of narcotic analgesics should raise the concern for this diagnosis.

98. In general, if the patient is hemodynamically unstable, a specific dysrhythmia does not usually need to be diagnosed prior to treatment. In the unstable patient with a dysrhythmia, a general rule of thumb is to provide electricity (perform electrical cardioversion) if the heart rate is fast, and if the heart rate is slow, pace the patient with a pacemaker.

99. Differentiate ventricular tachycardia (VT) from supraventricular tachycardia (SVT) with aberrancy based on findings on the 12-lead ECG. In unstable patients, assume that the rhythm is VT and treat accordingly whenever there is any question. Treating SVT with aberrancy as if it were VT is less problematic than treating VT as if it were SVT with aberrancy. Any of these findings on the 12-lead ECG strongly suggest VT:
    - Atrioventricular (AV) dissociation
    - Fusion or capture beats
    - Left- or right-axis deviation
    - QRS width of >140 msec
    - Concordance of QRS complexes
    - Monophasic or biphasic QRS in lead V1
    - RS or QS in lead V6
    - History of coronary artery disease or CHF
    - Evidence of AV dissociation on physical examination (cannon A waves)

100. A job won't make you happy. You can find someone miserable and someone joyful in any profession. Be kind, listen with intention, work hard toward something meaningful, stay curious, respect wisdom, be humble, laugh often, and love easily. Emergency medicine affords us the unique opportunity to share in patients' most profound moments, teaching us that, in the end, happiness is more about gratitude and human connection. Relish the privilege.

DECISION MAKING IN EMERGENCY MEDICINE

CHAPTER 1

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1. **Is there anything unique about emergency medicine?**
   Although there is significant crossover between emergency medicine and all other clinical specialties, emergency medicine approaches to patient care and the decision-making processes are unique. Emergency medicine physicians must be knowledgeable about all aspects of medical care, with an emphasis on identifying and treating acute life threats.

2. **Describe the conventional method of evaluating a patient.**
   A comprehensive history, physical examination including vital signs, routine laboratory diagnostic studies, special diagnostic procedures, and the formulation of a problem-oriented medical record and rational course of therapy constitute the ideal approach to patient care, because it is so comprehensive.

3. **Why is the conventional methodology not ideal for use in the ED?**
   Even though in retrospect only 10% to 20% of patients presenting to an ED truly have emergent problems, it must be presumed that every patient who comes to an ED has an emergent condition. Therefore the first and most important question that must be answered is, "What is the life threat?" The conventional approach does not ensure an expeditious answer to this question. Time constraints, multitasking, and limited resources also impede the use of conventional methodology in the ED.

4. **How do I identify the patient with a life-threatening condition?**
   Three components are necessary to quickly identify the patient with a life-threatening condition:
   1. A chief complaint and a brief, focused history relevant to the chief complaint
   2. A complete and accurate set of vital signs in the field and in the ED
   3. A rapid, focused physical examination that includes visualization, auscultation, palpation, and observation

5. **What is so important about the chief complaint?**
   The chief complaint, which sometimes cannot be obtained directly from the patient but must be obtained from family members, observers, emergency medical technicians (EMTs), or others at the scene, will immediately help categorize the general type of problem (e.g., cardiac, traumatic, respiratory, or psychiatric).

6. **Why are vital signs important?**
   Vital signs are the most reliable objective data that are immediately available to ED personnel, provided they are accurately taken and critically interpreted. Vital signs and the chief complaint, when used as triage tools, will identify the majority of patients with life-threatening conditions. Familiarity with normal vital signs for all age groups is essential.

7. **What are the determinants of (normal) vital signs?**
   Age, underlying physical condition, medical problems (e.g., hypertension), and current medications (e.g., β-blockers) are important considerations in determining normal vital signs for a given patient. For example, a well-conditioned, young athlete who has just sustained major trauma and arrives with a resting supine pulse of 80 beats per minute may have significant blood loss because the normal pulse is probably in the range of 40 to 50 beats per minute.

8. **What is the most inaccurate vital sign taken in the field and ED?**
   In the field the most common inaccurate vital sign is the respiratory rate, because it is sometimes estimated rather than counted. In the ED, the temperature may be inaccurate if a temporal or tympanic thermometer was used or if the patient was hyperventilating or mouth breathing when the oral temperature was taken. When either fever or hypothermia is suspected, measure a rectal temperature.

9. **Why do I need to compare field vital signs with ED vital signs?**
   Most prehospital care systems with a level of care beyond basic transport also provide therapy to patients. Because this therapy usually makes positive changes in the patient's condition, the patient may look deceptively well on arrival in the ED. For example, a 20-year-old woman is found in the field with acute onset of left lower quadrant abdominal pain. She is cool, clammy and diaphoretic, with a pulse of 116 beats per minute and blood pressure of 78 palpable. She receives 1000 mL of intravenous (IV) fluid on route to the ED. She may arrive with

normal vital signs and no skin changes. If one does not read and pay attention to the EMT's description of the patient and the initial vital signs, the presumption may be made that this is a stable patient.

10. When are normal vital signs abnormal?

This is when the vital signs, although in the normal range, are inconsistent with the patient's chief complaint and overall clinical appearance. For example, a 20-year old man with severe asthma who presents with hours of dyspnea and poor air movement may have a "normal" respiratory rate of 14 breaths per minute. For this patient one would expect a respiratory rate of 20 to 30 breaths per minute, and thus a respiratory rate of 14 is abnormal, indicating fatigue and impending respiratory failure. This is a classic example of when "normal" is abnormal.

11. Why do I need to visualize, auscultate, and touch the patient?

In many instances these measures help to identify the life threat (e.g., Is it the upper airway, lower airway, or circulation?). Touching the skin is important to determine whether shock is associated with vasoconstriction (i.e., hypovolemic or cardiogenic) or with vasodilatation (i.e., septic, neurogenic, or anaphylactic). Auscultation will identify life threats associated with the lower airway (e.g., bronchoconstriction, tension pneumothorax).

12. Once I have identified the life threat, what do I do?

Stop immediately and intervene to reverse the life threat. For example, if the initial encounter with the patient identifies upper-airway obstruction, take whatever measures are necessary to alleviate upper-airway obstruction such as suctioning, positioning, or intubating the patient. If the problem is hemorrhage, volume restoration and hemorrhage control are indicated.

13. I have identified and stabilized or ruled out an immediate life threat in the patient. What else is unique about the approach to this patient in the ED?

The differential diagnosis formulated in the ED must begin with the most serious condition possible to explain the patient's presenting symptoms and be continued from there. An example is a 60-year-old man who exhibits nausea, vomiting, and epigastric pain. Instead of assuming the condition is caused by a gastrointestinal disorder, an acute myocardial infarction (MI) must first be considered and appropriate steps must be taken to stabilize the patient (i.e., start an IV, initiate oxygen [O_2], and place a cardiac monitor). Then, rule out an MI, aortic dissection, or surgical or other acute abdominal pathology, by completing an adequate history and physical examination, an electrocardiogram (ECG), and appropriate laboratory studies.

14. Why does formulating a differential diagnosis sometimes lead to problems?

The natural tendency in formulating a differential diagnosis is to think of the most common or statistically most probable condition to explain the patient's initial presentation. This approach may overlook the most serious, albeit sometimes a very uncommon, problem. Therefore the practice of emergency medicine involves some degree of healthy paranoia to consider the most serious conditions compatible with the patient's presenting symptoms. Through a logical process of elimination, first rule in or out the life threats before gravitating to the more likely diagnoses.

15. Is a diagnosis always possible or necessary in the ED?

No. Patients should be informed of goals in the ED. Sometimes, the most important thing is to know that they don't have a life-threatening condition. It may take days, weeks, or months for a final diagnosis to be made. It is unreasonable to expect that every patient should or must have a diagnosis made in the ED.

16. If I cannot make the diagnosis, what do I do?

It is the role of the ED physician to rule out and stabilize serious or life-threatening conditions, not to always arrive at a definitive diagnosis. For example, a patient who comes to the ED with abdominal pain; who has had an appropriate history, physical examination, and diagnostic studies; and who in your best judgment does not have a life-threatening or acute surgical problem should be so informed. The discharge diagnosis would be abdominal pain of unknown etiology. This avoids the trap of labeling the patient with a benign diagnosis such as gastroenteritis or gastritis that is not supported by the medical record. More important, it avoids giving the patient the impression that there is a totally benign process occurring and will help to avoid the medical (and legal) problem of the patient returning 1 or 2 days later with something more serious, such as a ruptured appendix.

17. What is the most important question to ask a patient who comes to the ED with a chronic, persistent, or recurrent condition?

"What's different now?" This question should be asked of all patients who have a chronic condition that has resulted in a visit to the ED. The classic example is migraine headache. The patient with a chronic, recurrent migraine headache who is not asked this question may on this occasion have had an acute subarachnoid bleed. Such a patient may not volunteer that this headache is different from the pattern of chronic migraines unless asked.

18. How do I decide if the patient needs hospitalization?

The medical condition is the first obvious factor to consider. Beyond this, ask yourself the following questions: Is there a medical need that can be fulfilled only by hospitalization, or can the patient be safely observed in the outpatient setting? For example, does the patient need oxygen therapy or cardiac monitoring? Can patients who

have sustained head trauma adhere to head trauma precautions, or do they require in-patient care because of homelessness or living alone? The patient's ability to pay for services should not determine disposition decisions.

19. If the patient does not need admission, how do I arrange a satisfactory disposition?
All patients should be instructed to follow up or return to the ED for new or worsening symptoms. Failure to do so constitutes patient abandonment. Specific verbal and written follow-up instructions should be given to all patients.

20. What is the most important thing to document in the ED discharge instructions?
All follow-up instructions must include specific mention of the most serious potential complications of the patient's condition. For example, a patient who is being discharged home with the diagnosis of a probable herniated L4-5 intervertebral disk should be instructed to return immediately if any bowel or bladder dysfunction develops. This takes into account the most serious complication of a herniated lumbar disk, which is a central midline disk herniation (i.e., cauda equina syndrome) with bowel or bladder dysfunction, an acute neurosurgical emergency.

21. What three questions should always be asked (and answered) before a patient is discharged from the ED?
1. Why did the patient come to the ED?
2. Have all specific concerns or fears been addressed?
3. Have I made the patient feel better?

Generally, most patients come to the ED because of pain, somatic or psychological, and a reasonable expectation is that this pain will be acknowledged and appropriately treated. If such pain cannot be alleviated, a thorough explanation should be given to the patient regarding the reasons why analgesics cannot be provided. Reassurance is sometimes all that is needed to relieve anxiety about serious medical conditions.

22. Why is the previous question and answer one of the most important in this chapter?
Attention to treating and alleviating a patient's pain will dramatically reduce subsequent complaints concerning care in the ED and remove one of the significant risk factors for initiation of a malpractice suit. It may also decrease the likelihood of an unnecessary return visit to the ED. It is also how you would want to be treated.

23. What about the chart?
The chart must reflect the answers to the preceding questions in this chapter. It need not list the entire differential diagnosis, but one should be able to ascertain from reading the chart that the more serious diagnoses were indeed considered. It also must contain appropriate follow-up instructions.

24. What role do clinical decision rules have in decision making in the ED?
Evidence-based clinical decision rules (such as pulmonary embolism rule-out criteria [PERC]) should be followed unless specific circumstance makes deviation from the rule in the best interest of the patient. In such cases, document the reasoning for deviation.

25. What is the role of shared decision making in the ED?
Shared decision making can be useful in a day and age of access to too much information via the Internet. It is important to get agreement from the patient as to their course of treatment. This can be accomplished by explaining the reasons that a particular study does or does not need to be done. The patient can then feel more comfortable with the decisions made.

KEY POINTS: DECISION MAKING IN EMERGENCY MEDICINE

1. Stabilize the patient before performing diagnostic procedures.
2. Always consider the most serious possible cause of the patient's signs and symptoms.
3. Always inquire about a patient's social situation before ED discharge.
4. Remember to focus on alleviating the patient's somatic or psychological pain.

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QUESTIONS

1. When formulating a differential diagnosis on an ED patient, one should ask what is:
   a. The most likely diagnosis
   b. The most serious diagnosis
   c. The most uncommon diagnosis
   d. The most benign diagnosis
   The correct answer is *b.*
2. What is the top priority in the initial encounter with a patient in the ED?
   a. Introduce yourself.
   b. Order appropriate diagnostic studies.
   c. Identify and stabilize the life threat.
   d. Obtain a past medical history.
   The correct answer is *c.*
3. What is the most often inaccurate vital sign obtained by EMTs in the prehospital setting?
   a. Blood pressure
   b. Pulse
   c. Respiratory rate
   d. Temperature
   The correct answer is *c.*

CHAPTER 2

ADULT CARDIAC ARREST

Ryan Murphy, MD and Jason Haukoos, MD, MSc

1. What is cardiac arrest and what is its incidence?

Cardiac arrest is defined by the triad of unconsciousness, apnea, and pulselessness.

In 2017, incidence of EMS–assessed out-of-hospital cardiac arrest (OHCA) in people of any age was 111 per 100,000 population, based on extrapolation from the Resuscitation Outcomes Consortium.

2. What are the most common causes of nontraumatic OHCA in adults?

The most common etiology of nontraumatic OHCA in adults is coronary heart disease (CHD). Other causes include respiratory failure, sepsis, circulatory obstruction, hypovolemia, electrolyte disturbances (most commonly hyperkalemia), drug toxicity, electrocution, and hypothermia.

3. What are the four initial rhythms seen in cardiac arrest? Which is most common?

The four initial rhythms are ventricular fibrillation (VF), pulseless ventricular tachycardia (pVT), pulseless electrical activity (PEA), or asystole. These rhythms are generally categorized as shockable (VF, pVT) or nonshockable (PEA, asystole). As most arrests are initially unmonitored, the prevalence of each initial rhythm is unknown. The 2019 American Heart Association (AHA) Heart Disease Statistic Update reported that initial recorded cardiac rhythm was shockable in 19% of EMS-treated OHCAs in 2017. Underlying CHD accounts for the majority of VF arrests. PEA and asystole can be due to various underlying causes but is also commonly the result of prolonged or untreated VF/pVT.

4. What are the "H's and T's" of treating cardiac arrest?

A mnemonic is used to remember reversible causes of cardiac arrest (especially in patients with initial rhythms of PEA or asystole). H's and T's: **h**ypovolemia, **h**ypoxia, **h**ydrogen ion (acidosis), **h**ypo/**h**yperkalemia, **h**ypothermia, **t**ension pneumothorax, **t**amponade, **t**oxins, **t**hrombosis (cardiac or pulmonary).

5. Why are the ABCs of cardiac arrest better represented as CAB?

In 2010, the AHA recommended the basic life-support sequence for all *adult* patients experiencing cardiac arrest be changed from airway-breathing-chest compressions (ABC) to chest compressions, airway, and breathing (CAB). (See Chapter 69 for details related to management of pediatric cardiac arrest.) All rescuers (regardless of training) are recommended to initiate chest compressions before giving rescue breaths. In nontraumatic adult OHCA, oxygen delivery to the heart and brain during the *first few minutes* of cardiopulmonary resuscitation (CPR) is limited by blood flow, rather than by arterial oxygen content. This sequence change was recommended to prioritize the early initiation of chest compressions, which are often delayed when opening the airway and rescue breaths are prioritized.

6. Is ventilation and oxygenation still important during adult CPR?

Yes, ventilation and oxygenation remains an essential component of adult CPR. While various ventilation and oxygenation strategies have been studied for many years, there is still incomplete evidence regarding optimal procedures: type of delivery used (i.e., passive oxygen insufflation, bag mask ventilation [BVM], advanced airway placement); timing of advanced airway placement in relation to other interventions; or type of advanced airway device used (supraglottic airway [SGA] vs. endotracheal tube [ETT]). What is known to improve survival in adults experiencing nontraumatic OHCA is early high-quality CPR (and, for VF/pVT, defibrillation). Therefore the tasks and timing of ventilation and oxygenation need to be integrated in a way that minimizes interruptions or quality of chest compressions.

7. How should a healthcare provider perform cardiopulmonary resuscitation (CPR) on an adult as described by the AHA?

Simultaneously check for pulse (<10 seconds allowed for pulse check) while checking to see if patient is not breathing or having only agonal respirations.

1. If the arrest occurs in the:
   a. out-of-hospital setting → call 911 (activate EMS)
   b. in-hospital setting → activate the hospital's cardiac arrest team
2. Begin high-quality CPR for 2 minutes
   a. Chest compression technique guidelines for adults in ALL settings:
      i. compression rate = 100–120 compressions per minute
      ii. adequate depth of at least 2 inches (5 cm), while avoiding excessive compression depths >2.4 inches (6 cm)

iii. allow full chest wall recoil (avoid leaning on chest between compressions)
iv. minimize interruptions (goal of compression fraction, defined as the time performing compressions divided by total time, as high as possible, with a target of at least 60%)

b. Ventilation guidelines:
i. Prior to advanced airway:
1. recommended compressions:ventilation ratio of 30:2 (via mouth-to-mouth/mouth-to-mask if lone rescuer, BVM if two or more trained rescuers present)
ii. If advanced airway (SGA or ETT): one ventilation every 6 seconds (10 ventilations per minute) with continuous chest compressions
1. ideal timing of advanced airway unclear, but it should NOT delay initial CPR and should not cause significant (>10 seconds) interruption in chest compressions
iii. When supplementary oxygen is available, use the maximal feasible inspired oxygen concentration during CPR

3. Use automatic external defibrillator (AED) as soon as ready and defibrillate if indicated (i.e., VF/pVT)
a. If **lone rescuer** and AED is **not nearby or easily accessible:**
i. begin cycle of 30 chest compressions and two breaths until help arrives, then have additional rescuer obtain AED
b. if **lone rescuer** and AED is **nearby or easily accessible:**
i. immediately obtain AED, attach defibrillator/monitor, then perform compressions/rescue breaths
c. If two or more trained rescuers are present:
i. one rescuer begins CPR while other rescuers obtain the AED and other emergency equipment
ii. defibrillate as soon as ready, if indicated
4. Obtain IV/intraosseous (IO) access, give epinephrine (1 mg IV/IO push every 3–5 minutes)
5. Every 2 minutes, check rhythm/pulse (aim for a <10 second pause in compressions) and consider additional medications/advanced airway/capnography depending on suspected etiology of arrest.

8. Explain the mechanism of blood flow during CPR.

Two basic models explain the mechanism of blood flow during CPR. In the cardiac pump model, the heart is squeezed between the sternum and the spine. Chest compressions mimic systole, and the atrioventricular valves close normally, ensuring unidirectional, antegrade flow. During the relaxation phase (diastole), intracardiac pressures fall, the valves open, and blood is drawn into the heart from the lungs and vena cava. In the thoracic pump model, the heart is considered a passive conduit. Chest compressions result in uniformly increased pressures throughout the thorax. Forward blood flow is achieved selectively in the arterial system, because the stiff-walled arteries resist collapse and retrograde flow is prevented in the great veins by one-way valves. Chest recoil results in increased negative intrathoracic pressures, which improve ventricular filling and coronary blood flow. These mechanisms have been substantiated in animal models, and both likely contribute to blood flow during CPR.

9. Is blood flow to the brain and heart adequate during CPR?

Even when performed by experts, CPR provides only approximately 30% of normal blood flow to the brain and 10% to 20% of normal blood flow to the heart. Blood flow to the heart occurs during the relaxation phase of CPR, whereas blood flow to the brain occurs during the compression phase of CPR. This is the foundation for the AHA's recommended CPR "duty cycle" of 50% (the proportion of the compression/decompression cycle spent in compression).

10. What is coronary perfusion pressure (CPP)? What is the association between CPP, CPR, and return of spontaneous circulation (ROSC)?

CPP is defined as the aortic pressure minus the right atrial pressure during diastole.

Better CPR produces better CPPs and higher CPPs are directly correlated with higher rates of ROSC. This emphasizes the importance of performing high-quality CPR with an ultimate emphasis on optimizing CPP.

11. What is capnography and how can it be used during resuscitation?

Capnography involves measurement of the partial pressure of carbon dioxide in exhaled gases (including quantitative end-tidal carbon dioxide [$EtCO_2$] values and display of the capnogram, a real-time CO_2 waveform). During periods of acutely decreased cardiac output, such as with cardiac arrest with performance of CPR, $EtCO_2$ levels may reflect acute changes in cardiac output. Animal studies have confirmed that $EtCO_2$ detected during CPR correlates with CPP, while clinical studies have demonstrated correlation between $EtCO_2$ levels and ROSC and survival after cardiac arrest. $EtCO_2$ levels <10 mmHg are strongly predictive of poor cardiac arrest outcomes, whereas elevated $EtCO_2$ levels suggest possible good outcomes. While no absolute value or relative change in $EtCO_2$ is perfectly predictive, use of capnography to guide chest compression quality and ROSC is useful.

12. Describe hands-off CPR.

Hands-off CPR refers to lifting the hands off the chest wall during decompression to maximize chest recoil. Incomplete chest wall recoil, commonly observed in human studies, has been shown to impede forward blood flow in animal models and is probably detrimental in humans

13. Discuss the role of vasopressors (epinephrine and vasopressin) during treatment of cardiac arrest.

The immediate goal of vasopressor pharmacologic therapy is to improve CPP, and thus myocardial blood flow, which is associated with ROSC. Epinephrine is an adrenergic agonist that increases systemic vascular resistance, thus improving CPP by augmenting the diastolic aortic:right atrial gradient. Epinephrine (1 mg IV/IO push every 3–5 minutes) is the only vasopressor currently included in the AHA Advanced Cardiac Life Support (ACLS) algorithm. If the rhythm is shockable, there is insufficient evidence regarding optimal timing of epinephrine in relation to defibrillation, but in nonshockable rhythms, epinephrine can be administered as soon as feasible after the onset of arrest.

14. Why is vasopressin no longer included in AHA ACLS guidelines?

Vasopressin is a nonadrenergic agonist that acts directly on V_1 receptors and had been included in prior guidelines as an adjunct to epinephrine. Vasopressin was removed from the AHA ACLS algorithm in the interest of simplicity, as more recent evidence did not show an added benefit to administering either vasopressin alone or in combination with epinephrine, compared with epinephrine alone.

15. What are the appropriate routes of drug administration?

IV administration is the preferred route of drug therapy when treating cardiac arrest. A central venous catheter is ideal, although placement should not supersede other resuscitation efforts (e.g., chest compression). Use of a peripheral venous catheter results in a slightly delayed medication onset of action, although the peak drug effect is similar to that for the central route. An IO line may also be used and should take precedence over other approaches, including intramuscular or endotracheal routes. All drugs used for resuscitation can be given in conventional doses using IO access. Intracardiac administration should be reserved for cases of open cardiac massage.

16. I thought IO cannulation was only used as a last resort. What's the deal?

IO cannulation provides a quick, effective, and safe means to access a noncollapsible venous plexus, either in the proximal tibia, proximal humerus, or sternum. (The sternum should be avoided as an IO site in cardiac arrest, because it interferes with chest compressions.) It can be used in all age groups and allows for effective fluid resuscitation, drug delivery, and blood sampling for laboratory evaluation. In fact, the IO functions similar to that of a central line in terms of rapid access to the patient's central circulation.

17. What are the initial key treatment principles when managing patients with a shockable rhythm (VF/pVT)?

Adult patients treated by EMS with a shockable rhythm have a much higher probability of surviving to hospital discharge than patients with nonshockable presenting rhythms (according to CARES Registry 2017, 29% and 6%, respectively). Rapid identification and immediate defibrillation of VF/pVT as soon as the AED is available and ready (in addition to high-quality CPR) is the only rhythm-specific therapy proven to increase survival to hospital discharge and should not be delayed for other ACLS measures. While the AED is being retrieved and applied, high-quality uninterrupted CPR should be immediately initiated and resumed after rapid defibrillation. Recommended shock energy levels for defibrillation on biphasic monitors (preferred and most common) is based on the manufacturer recommendation (initial dose 120–200 J), or if unknown, use maximum available. If only monophasic defibrillator available, 360 J is recommended. If there are no signs of ROSC, continue down the ACLS algorithm, including additional shocks and consideration of antiarrhythmics.

18. What is the optimal placement of electrode pads used for defibrillation?

For ease of placement, the anterolateral pad position is recommended. However, for ventricular dysrhythmias, there is no efficacy difference between this position and the three others (anteroposterior, anterior–left infrascapular, or anterior–right infrascapular).

19. Should you administer one shock at a time or a sequence of shocks (also referred to as stacked shocking)?

No study has shown survival benefit with stacked shocks. If one shock fails to eliminate VF/pVT, the incremental benefit of another shock is low, and resumption of CPR is likely to confer greater value than another immediate shock.

20. Discuss the role of antiarrhythmics in shock refractory VF/pVT and the updated 2018 AHA focused guideline regarding addition of lidocaine to the choices of antiarrhythmic.

The primary objective of antiarrhythmics is to facilitate successful defibrillation and reduce risk of recurrent dysrhythmia. While early CPR and defibrillation are the only therapies associated with improved long-term survival in patients with VF/pVT, antidysrhythmics (2015 AHA guidelines included amiodarone) have been associated with increased rates of ROSC and survival to hospital admission. In 2018, the AHA updated the 2015 recommendations for antidysrhythmics for adult VF/pVT to include consideration of amiodarone (first dose, 300 mg IV/IO bolus; second dose, 150 mg IV/IO bolus) OR lidocaine (first dose, 1.0–1.5 mg/kg IV/IO; second dose, 0.5–0.75 mg/kg), class Ib antidysrhythmic, for VF/pVT that is unresponsive to defibrillation. The addition of lidocaine was based on the most recent randomized controlled trial (RCT) to date which found that lidocaine had comparable efficacy to amiodarone. The optimal sequence of administration of antidysrhythmic drugs during resuscitation or timing of drug administration in relation to shock delivery is still not known.

21. Should I routinely administer magnesium in adults with shock refractory VF/pVT?

No. The recent AHA guidelines recommend against the routine use of magnesium in patients with shock-refractory VF/pVT. However, magnesium should be considered for torsades de pointes.

22. What about esmolol for refractory VF? I've heard there may be compelling evidence to support its use in this situation.

Esmolol is a Class II antidysrhythmic and has been hypothesized to abort refractory VF. Evidence for its use in this setting comes from two small observational studies. The first, a retrospective study published in 2014, originates from a single center in the United States and compares six patients who received esmolol (loading dose 500 mcg/kg, infusion 0–100 mcg/kg/min) after usual ACLS care (including at least three defibrillation attempts, 3 mg epinephrine, and 300 mg amiodarone) with 19 patients who received otherwise usual ACLS care but without esmolol. In the esmolol group, 50% ($n = 3$) were discharged from the hospital with good neurologic function as compared with only 11% ($n = 2$) in the nonesmolol group. The second study, a retrospective pre-post study performed in Korea and published in 2016, included a similar small number of patients with refractory VF and used the same dose of esmolol, again after usual ACLS care. Sustained ROSC was significantly more common in the esmolol group compared with the nonesmolol group (56% [$n = 9$] vs. 16% [$n = 4$]), and improved rates of survival with good neurologic function (19% [$n = 3$] vs. 8% [$n = 2$]). Based on this current limited evidence, esmolol may be considered in patients with refractory VF.

23. Should I routinely administer sodium bicarbonate during resuscitation?

Similarly, sodium bicarbonate is not recommended as routine therapy in the setting of cardiac arrest. A no- or low-flow state causes progressive respiratory and metabolic acidosis as a result of accumulation of carbon dioxide and lactate, respectively. Neither state can be corrected without adequate oxygenation, ventilation, and tissue perfusion. At present, no clinical data support the routine use of sodium bicarbonate, except in cases of hyperkalemia, tricyclic antidepressant overdose, or preexisting metabolic acidosis.

24. Should I routinely administer calcium during resuscitation?

Calcium is also not recommended as routine therapy in the setting of cardiac arrest. Although no data exist to support its routine use, it may be beneficial in the setting of hyperkalemia (most often seen in chronic renal failure/dialysis patients), hypocalcemia, or calcium channel blocker toxicity.

25. How should asystole be treated?

Identify and treat the underlying cause. Confirm the absence of cardiac activity in more than one electrocardiogram lead. Check for loose or disconnected cables and monitor leads. Finally, increase the amplitude to detect occult, fine VF.

26. Is defibrillation or electrical pacing useful for asystole?

Defibrillation is reserved for cases in which differentiation between asystole and fine VF is difficult. In these ambiguous situations, defibrillation should be employed after administration of epinephrine. Electrical pacing is occasionally attempted for asystole, but rarely effective in restoring pulses and is not recommended.

27. What should I do after ROSC?

Once ROSC is achieved, the vulnerable and highly tenuous postresuscitation period begins. This period is marked by a profound systemic inflammatory response syndrome resulting from whole-body ischemia and reperfusion. Patients often experience hemodynamic instability, multiple-organ dysfunction, and subsequent death (hours to days later). Prompt recognition and treatment of the inciting event and meticulous intensive care unit support are required to provide patients with the best probability for survival. In addition, early aggressive percutaneous coronary intervention and targeted temperature management (TTM) should be performed to improve survival and neurologic recovery, respectively.

28. What is TTM?

TTM refers to induced core hypothermia (32°C–36°C) and active temperature control (i.e., prevention of hyperthermia) for approximately 24 hours in comatose out-of-hospital cardiac arrest survivors. Two landmark studies published in the early 2000s showed this intervention to be highly efficacious for improving neurologic function, and a more recent study, published in 2013, showed similar results to the original studies, but no advantage of cooling to 33°C versus 36°C.

29. How much time do I have to reach the target temperature and should I perform this in the ED?

Although not known for sure, it is thought the sooner TTM is initiated the better. If possible, this intervention should be initiated in the ED.

30. What's the best approach to cooling for TTM?

Surface cooling and catheter-based cooling are the two approaches used for TTM. While both work to obtain hypothermic core temperatures, catheter-based cooling is probably superior in maintaining a constant core temperature while also preventing excessive hypothermia.

31. When may prehospital resuscitation efforts be terminated?

According to the most recent AHA ACLS guidelines, prehospital resuscitation can be discontinued in the out-of-hospital setting by EMS authorities when a valid no-CPR order is presented to the rescuers or when a patient is

deemed nonsurvivable after an adequate trial of basic life support and ACLS, including successful endotracheal intubation, achievement of IV/IO access, and administration of appropriate medications, and determination of a persistent asystolic or agonal rhythm, as well as when no reversible cause for the arrest is identified.

32. Can capnography be used *in isolation* to predict when to terminate a prolonged resuscitation?

No! While multiple prospective observational studies have found that survival is very unlikely in intubated patients if their $EtCO_2$ is $\leq$10 mmHg after 20 minutes of CPR, these studies are limited (observational and relatively small) and low $EtCO_2$ values in real clinical scenarios are difficult to interpret in isolation. Low $EtCO_2$ values may reflect inadequate cardiac output, but the low value could be confounded due to bronchospasm, obstruction of the endotracheal tube (due to mucus plugging, kinking or alveolar fluid), hyperventilation, and airway leak. Thus the 2015 AHA updated recommendations state:

> *In intubated patients, failure to achieve an $EtCO_2$ of greater than 10 mmHg by waveform capnography after 20 minutes of CPR may be considered as one component of a multimodal approach to decide when to end resuscitative efforts, but it* should not be used *in isolation.*

33. What are other reversible causes and immediate treatments of cardiopulmonary arrest?

- Hyperkalemia: calcium chloride (preferred over calcium gluconate), sodium bicarbonate, insulin and glucose, and nebulized albuterol.
- Anaphylaxis: intravascular volume expansion (using crystalloid) and epinephrine.
- Cardiac tamponade: pericardiocentesis or pericardiotomy.
- Tension pneumothorax: thoracic decompression.
- Hypovolemia: intravascular volume expansion using crystalloid solutions. In the setting of trauma, blood products should be given judiciously and concomitantly with crystalloid. Always consider using a level I infuser when large volumes are required over a short period of time.
- Torsades de pointes: defibrillation, magnesium sulfate, isoproterenol, or overdrive pacing.
- Toxic cardiopulmonary arrest:
  - Carbon monoxide poisoning occurs after prolonged exposure to smoke and inhalation of exhaust from incomplete combustion. High-flow and hyperbaric oxygen and management of acidosis are the cornerstones of treatment.
  - Cyanide poisoning occurs after intentional ingestion or after exposure to fire involving synthetic materials. The antidote for this includes hydroxycobalamin, which combines with cyanide to form cyanocobalamin (vitamin B_{12}). Sodium nitrite and sodium thiosulfate are considered second-line therapy for cyanide toxicity.
  - Tricyclic antidepressants act as type Ia antidysrhythmic agents and cause cardiac conduction slowing, ventricular dysrhythmias, hypotension, and seizures. Vigorous serum alkalinization with sodium bicarbonate and seizure control are required.
- Primary asphyxia: in addition to anaphylaxis, obstructive asphyxia may occur after foreign body aspiration, inflammatory conditions of the hypopharynx (e.g., epiglottitis or retropharyngeal abscess), or neck trauma. The latter results in edema or hematoma formation, subcutaneous emphysema, or laryngeal or tracheal disruption. Treatment includes establishment of a patent airway via endotracheal intubation or by cricothyrotomy and assisted ventilation with 100% oxygen.

34. What are the indications for open-chest cardiac massage?

The primary indication for open-chest cardiac massage is traumatic arrest. However, several other non–trauma-related indications include hypothermia, pulmonary embolism, cardiac tamponade, abdominal hemorrhage, third-trimester pregnancy, and patients with chest wall deformities that prevent adequate chest compressions.

35. What percentage of adult out-of-hospital cardiac arrest patients survive to hospital discharge?

Ten percent, according to national epidemiologic data from 2017.

KEY POINTS: MANAGEMENT OF CARDIAC ARREST

CPR and defibrillation are the most important components to the initial management of the cardiac arrest patient.

1. Treat VF with immediate defibrillation if the arrest is witnessed on monitor and AED ready; treat with CPR and then defibrillation if the arrest is unwitnessed or if need to obtain/apply AED.
2. If the arrest is caused by PEA, remember its common reversible causes (i.e., hypovolemia, hypoxia, cardiac tamponade, tension pneumothorax, hypothermia, massive pulmonary embolism, drug toxicity, electrolyte disturbances, acidemia, or myocardial infarction) and treat them appropriately.
3. If the arrest is the result of asystole, remember to exclude fine VF.
4. Quality of CPR should be continuously monitored.

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QUESTIONS

1. Cardiac arrest resulting from VF is:
   a. Best treated with immediate CPR followed by intravenous epinephrine
   b. Best treated with immediate defibrillation
   c. Associated with the highest mortality rate
   d. Best treated with magnesium sulfate
   The correct answer is *b.*
2. ROSC after cardiac arrest is:
   a. A rare event
   b. Most common when asystole is the initial rhythm
   c. Directly related to degree of CPP during CPR
   d. Associated with use of calcium
   The correct answer is *c.*
3. Asystole:
   a. Is associated with the highest survival rate
   b. May actually be fine VF
   c. Is treated with amiodarone and atropine
   d. Results from too aggressive CPR
   The correct answer is *b.*

CHAPTER 3

AIRWAY MANAGEMENT

Jeremy Collado, MD and W. Gannon Sungar, DO

1. Which ED patients need airway assessment?

Emergency physicians are masters of the airway and every patient in the ED should have their airway assessed.

2. What are the different mechanisms of respiratory failure?

Respiration consists of oxygenation and ventilation. Patients can experience respiratory failure by four main mechanisms:

- **Loss of airway protective reflexes** – often due to a nonrespiratory cause (e.g., trauma, toxicologic), resulting in collapse of the airway anatomy and loss of airway patency.
- **Hypoxemia** – failure of oxygenation, manifested by cyanosis and/or low readings on pulse oximetry.
- **Hypercapnia** – failure of ventilation, leading to elevated pCO_2 levels that results in acidosis and altered mental status.
- **Mixed** – failure of both oxygenation and ventilation.

3. How do I assess a patient's respiratory status?

If patients are able to speak clearly, they have an intact airway. Lacking this, signs of airway collapse include sonorous respirations, pooling of secretions, and inability to swallow. Assessment of oxygenation and ventilation is achieved by looking at the patient's skin color, work of breathing, respiratory rate, and mental status.

4. Does a lack of a gag reflex mean my patient can't protect their airway?

No, the lack of a gag reflex is an unreliable marker for airway collapse, as up to 25% of the population lacks a gag reflex at baseline. Also, the presence of a gag reflex does not imply ability to protect the airway.

5. What is a definitive airway?

A definitive airway is defined as a cuffed endotracheal (ET) tube inflated below the level of the vocal cords.

6. What is the most common cause of airway obstruction?

The tongue is the most common cause of airway obstruction, as it blocks the airway far more commonly than do foreign bodies or edema. With decreasing levels of consciousness, the supporting muscles in the floor of the mouth lose tone and the tongue falls posteriorly, obstructing the oropharynx.

7. How can I initially assist a patient in respiratory failure?

Patients with airway collapse may benefit from an airway maneuver or airway stenting, including:

- **Head tilt/chin lift** – lift the chin cephalad and anterior, creating slight extension of the head.
- **Jaw thrust** – lift anteriorly at the bilateral mandibular angles, moving the tongue off of the posterior oropharynx.
- **Artificial airway** – a nasopharyngeal airway (NPA) or an oropharyngeal airway (OPA) can be placed in the naris or mouth, respectively, to stent the tongue off of the posterior oropharynx and maintain upper airway patency.
- **Bag valve mask (BVM)** – after using one or more of the airway maneuvers above to establish airway patency, BVM can be used to support oxygenation and ventilation.

8. How do I predict patients who will be difficult to assist with a BVM?

The MOANS mnemonic can be a useful tool for predicting difficulty in using a BVM.

- M – Mask seal – things that could impair mask seal may include beard, vomitus, blood, facial trauma, anatomic abnormalities, or incorrect mask size.
- O – Obesity/obstruction, including oropharyngeal swelling or masses will also cause difficulty in using a BVM.
- A – Age $>$55 years old.
- N – No teeth – while edentulousness may make intubation easier, it makes using a BVM more difficult as the soft tissue collapses onto itself.
- S – Stiff lungs – pathology such as chronic obstructive pulmonary disease (COPD), asthma, chest trauma, etc., can impair easy movement of air in and out of the lungs.

9. What is rapid sequence intubation (RSI)?

RSI is a method of facilitating ET intubation by inducing unconsciousness and paralysis. Because patients requiring emergent airway management are at risk for aspiration, the airway must be secured as quickly as possible, ideally, after a period of preoxygenation followed by induction of unconsciousness, paralysis, and then intubation. Preferably, preoxygenation is performed without positive pressure ventilation to avoid insufflating the stomach.

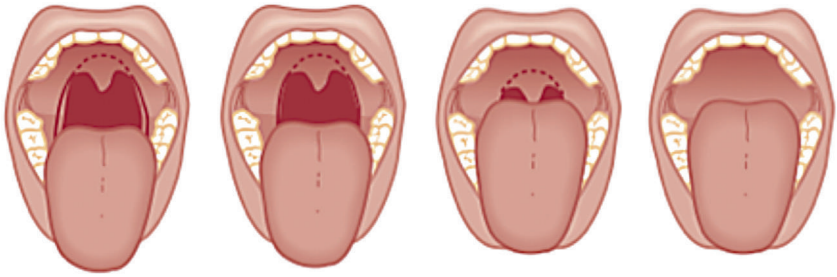

Fig. 3.1 Mallampati score. *(From Brown CA, Walls RM. Airway. In: Walls RM, Hockberger RS, Gausche-Hill M, eds. Emergency Medicine Concepts and Clinical Practice. 9th ed. Philadelphia: Elsevier; 2018:5.)*

10. How do I assess for a difficult intubation?

The LEMON mnemonic is a helpful reminder of factors associated with a difficult intubation:

- L – Look externally to assess for facial trauma, blood in the airway, etc.
- E – Evaluate using the 3-3-2 rule to predict difficult airway anatomy.
  - Patients who cannot open their mouth to fit three fingers between their central incisors may have limited mouth opening necessary for direct laryngoscopy.
  - A hyomental distance (hyoid to tip of chin) fewer than three finger breadths predicts a more difficult anterior larynx.
  - Fewer than two finger breadths from hyoid to the thyroid cartilage predicts a short neck and a cephalad larynx.
- M – The Mallampati score (Fig. 3.1) is a measure of baseline airway patency and is a good predictor of ease of laryngoscopy.
  - Class I – complete visualization of the uvula and the tonsillar pillars
  - Class II – visualization of the entire uvula
  - Class III – visualization of only the base of the uvula
  - Class IV – limited to visualization of the hard palate only
- O – Obstruction – evaluate for visualized foreign bodies and stridor.
- N – Neck mobility – decreased neck mobility due to kyphosis, c-collar, etc., may limit manipulation techniques that can aid in direct laryngoscopy.

11. What basic equipment is necessary for ET intubation?

- **Laryngoscope** – Direct and video (see later). There are two common direct laryngoscope blades, specifically:
  - **Macintosh** (curved blade), which is placed anterior to the epiglottis, into the vallecula, acting to lever the epiglottis off of the cords using the median glossoepiglottic fold.
  - **Miller** (straight blade), which is used to lift the underside of the epiglottis anteriorly, revealing the cords underneath. Miller blades are more commonly used in pediatric intubations where the epiglottis tends to be floppier.
- **Suction** – A Yankauer suction catheter should be available to help remove saliva, blood, or emesis from the airway, enhancing the view of the cords.
- **ET tube** – Adult males are generally able to accommodate a 7.5–9.0-mm ET tube, whereas women are generally intubated with a 7.0–8.0-mm tube.
- **Syringe** – A 10 mL syringe is needed to inflate the ET tube cuff.
- **Stylet** – A flexible, metal probe that is inserted into and used to shape the ET tube for intubation.
- **Gum elastic bougie** – A bougie is a semirigid introducer with a flexed tip that is frequently used for difficult intubations with incomplete visualization of the larynx. Lacking direct visualization, a bougie can be passed under the epiglottis and tracheal insertion can be confirmed as the flexed tip of the bougie bounces along the tracheal rings. An ET tube is then inserted into the trachea over the bougie. A bougie can also be used with clear, direct visualization of the airway anatomy. Although not standard of practice, recent data suggest an improvement in first-pass intubation success rate in patients with difficult airways using a bougie first approach.

12. What is a video laryngoscope and what types are there?

A video laryngoscope is a laryngoscope blade with a camera embedded at its tip that displays images on a screen most commonly placed at the patient's bedside. There are multiple companies that produce video laryngoscopes, many of which have several interchangeable blade shapes, from a traditional Macintosh blade to more

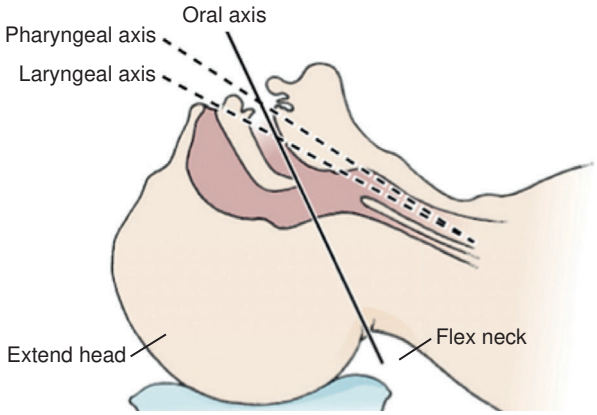

Fig. 3.2 Sniffing position. *(From Driver BE, Reardon RF. Tracheal intubation. In: Roberts JR, Hedges JR, eds. Clinical Procedures in Emergency Medicine. 7th ed. Philadelphia: Elsevier; 2019:71.)*

angulated blades. The systems with traditional blades often have the advantage of being able to be used both for direct as well as video laryngoscopy, depending on user preference and need.

13. What are the steps (7 P's) to RSI?

- **Preparation** – As with all procedures, preparation is the key to success. With every RSI attempt, equipment should be checked, medications should be planned in advance, and a back-up plan should be in place should the attempt fail.
- **Positioning** – The sniffing position (Fig. 3.2), with the neck flexed relative to the torso, the head extended, and the ear canal aligned horizontally with the sternal notch optimally aligns the oral, pharyngeal, and laryngeal axes for direct laryngoscopy. Sometimes pillows, blankets, soft ramps, and bed positioning are used to achieve the sniffing position, especially in obese patients.
- **Preoxygenation** – Adequate preoxygenation maximizes the time available to perform the intubation before desaturation occurs by replacing alveolar nitrogen with oxygen (nitrogen washout). Proper preoxygenation can be achieved with 3–5 minutes of normal ventilation with a nonrebreather mask or with eight vital capacity breaths at 100% fraction of inspired oxygen (FiO_2).
- **Pretreatment** – Laryngoscopy is a strong stimulus that can activate both the sympathetic and parasympathetic nervous systems. However, routine pretreatment with any medication other than a sedative and a paralytic has not been shown to improve outcomes. Atropine may be considered in children less than 12 months old to prevent bradycardia. Patients with head injuries may benefit from fentanyl and/or lidocaine to decrease the transient increase in intracranial pressure during intubation. Nebulized albuterol may help prevent laryngospasm in patients with reactive airway disease.
- **Paralyze** (and sedate with induction) – Induction medication should be immediately followed by administration of the paralytic (Table 3.1).
- **Pass the tube** – The tube should be visualized passing through the cords.
- **Postintubation management** – Immediately following intubation and confirmation of tube placement, the patient should be sedated, most commonly with propofol or a combination of an opioid and benzodiazepine.

14. What is passive apneic oxygenation?

Passive apneic oxygenation is a technique for preventing hypoxia during RSI, where a nasal cannula with high-flow oxygen is placed on the patient during the preoxygenation phase of RSI and left in place throughout the intubation attempt. Studies have shown that despite apnea during paralysis, passive oxygenation can greatly extend the time it takes for a patient to become critically hypoxic. Care should be taken with pediatric patients, as appropriate flow rates in order to prevent harm are based on size.

15. What medications are used for RSI?

Table 3.1 describes the pretreatment, induction, and paralytic medications frequently used for RSI.

16. What are the contraindications to using succinylcholine?

Because succinylcholine causes muscle depolarization and release of intracellular potassium, potentially life-threatening hyperkalemia can occur in certain high-risk populations. Succinylcholine is contraindicated in patients at risk for baseline hyperkalemia, including end-stage renal disease, severe acidosis, patients with major burns or crush injuries in the past 3–5 days (NOT ACUTELY), and any condition causing upregulation of acetylcholine receptors at the neuromuscular junction, including neuromuscular disease, stroke, and spinal injury.

Table 3.1 Common Medications for Rapid Sequence Intubation

	Pretreatment			*Induction*				*Paralytic*	
	ATROPINE	**LIDOCAINE**	**FENTANYL**	**ETOMIDATE**	**KETAMINE**	**MIDAZOLAM**	**PROPOFOL**	**SUCCINYL-CHOLINE**	**ROCURONIUM**
Class	Anticholinergic	Amino amide	Opioid analgesic	Imidazole derivative	PCP derivative	Benzodiazepine	GABA agonist	Depolarizing agent	Nondepolarizing agent
Dose	0.02 mg/kg	1.5–2 mg/kg	2–5 mcg/kg	0.3 mg/kg	1–2 mg/kg	0.1–0.3 mg/kg	1.5–3 mg/kg	1.5 mg/kg	1.2 mg/kg
Administration	3–5 min before intubation			Immediately before paralytic				Onset: 45–60 sec Duration: 5–9 min	Onset: 45–60 sec Duration: 20–75 min
Effect/benefit	Blunts bradycardic response to increased vagal tone from laryngoscopy Decreases bronchorrhea	Blunts elevation in ICP associated with laryngoscopy	Decreases sympathetic response to intubation (ICP, tachycardia, hypertension)	Hemodynamically neutral Decreases ICP	Bronchodilator (good for RAD) Preserves respiratory drive (awake intubation) Nystagmus May elevate ICP/IOP	Anticonvulsant effect Decreases ICP	Decreases ICP Decreased airway resistance Rapid on/off	Fasciculations Hyperkalemia Increased ICP, IOP	Nondepolarizing so no fasciculations or risk of hyperkalemia May increase HR, BP, CO
Notes	Not routinely recommended except before a second dose of succinylcholine in young children Max dose 1 mg	No longer routinely used for patients with suspected head trauma	Use in patients at risk for decompensation with sympathetic surge (aortic dissection, ICH, etc.)	Proposed adrenal suppression, but not clinically relevant with single dose used for RSI	Adverse effects: • Bronchorrhea • Laryngospasm • Cardiac depressant • Tachycardia • Hypertension • Emergence phenomenon	Adverse effects: • Negative inotrope • Hypotension	Adverse effects: • Hypotension • Pain on infusion • Anaphylaxis with soy/egg allergy	Life-threatening hyperkalemia in burns, crush injury, neuromuscular disease, and acidosis Malignant hyperthermia	Prolonged duration may delay neurologic examination in trauma patients

BP, Blood pressure; *CO,* cardiac output; *GABA,* γ-aminobutyric acid; *HR,* heart rate; *ICH,* intracranial hemorrhage; *ICP,* intracranial pressure; *IOP,* intraocular pressure; *PCP,* phencyclidine; *RAD,* reactive airway disease; *RSI,* rapid sequence intubation.

17. How deep do I advance an ET tube?

A traditional rule is that an ET tube should be placed at a depth equal to three times the tube size in centimeters (e.g., an 8.0-mm tube should be placed 24 cm at the teeth). Due to airway anatomy, deeper tube placement will usually end up in the right mainstem bronchus.

18. How do I confirm ET tube placement?

ET tube placement is best confirmed by direct visualization of the tube passing through the vocal cords. Signs of correct tracheal tube placement include condensation in the tube, bilateral breath sounds with bagging, absence of breath sounds over the epigastrium, and color change on colorimetric capnometry. Continuous waveform end-tidal CO_2 (capnography) is the most reliable method for confirming and monitoring correct ET tube placement. If continuous waveform is not available, a colorimetric end-tidal CO_2 detector should be used. Chest x-ray is valuable for confirming appropriate depth of tube placement, but should not be relied upon to confirm ET placement due to the time it takes to complete.

19. What are the contraindications to RSI?

RSI is contraindicated in any patient where securing of the airway is predicted to be difficult. Anticipation of a difficult airway based on anatomic features (foreign body, allergic reaction, airway infections, malignancies) or traumatic anatomic distortion (massive facial trauma, facial burns) is a relative contraindication to RSI. Difficult airway must be considered in the context of the environment, skill of the operator, and available equipment.

20. What are the steps to awake fiberoptic intubation?

Awake fiberoptic intubation is an excellent option for patients with some respiratory effort in whom RSI is contraindicated, most commonly due to airway obstruction. Patients can be placed in the upright position, the oropharynx is anesthetized with nebulized or topical anesthetic spray, and patients are given moderate sedation, often with ketamine, maintaining their respiratory drive. A flexible fiberoptic scope already threaded through an ET tube is then maneuvered into the oropharynx via a nasal or oral approach and passed through the cords under direct visualization, at which point the ET tube is advanced.

21. What is delayed sequence intubation (DSI)?

DSI can be used in patients with hypoxia refractory to traditional preoxygenation techniques. DSI is the use of ketamine for procedural sedation with the procedure being preoxygenation. Patients are given 1–2 mg/kg of ketamine and then bag valve mask ventilated or placed on a noninvasive ventilator until their oxygenation can be maximized, at which point standard RSI is performed.

22. What is an extraglottic airway device (EAD)?

An EAD is a device that is placed without direct visualization above or posterior to the larynx, typically blocking off the esophagus, allowing ventilation and oxygenation. There are various types with differing pros and cons, including prevention of aspiration, ability to intubate through the device, and poor seal, to name a few. These are good rescue devices for patients who are difficult to bag valve mask or as a temporizing method after failed ET intubation.

23. What is a laryngeal mask airway (LMA)?

An LMA is a type of EAD that has an oval mask with a cuffed rim that is inserted into the oropharynx. It is intended to create a seal directly over the larynx, allowing ventilation and oxygenation. An intubating LMA is an LMA with a rigid channel through which an ET tube can be directed through the cords. LMAs are considered the rescue device of choice in a cannot-intubate/cannot-ventilate scenario.

24. What is a King airway? What is a Combitube?

Both the Combitube and the King airway (Fig. 3.3) are types of EADs that are most frequently used by prehospital providers.

- **King airway** – The King LT is a single-lumen tube with a small distal balloon and a larger proximal balloon that is inserted blindly into the mouth with the intention of placing the tip in the esophagus. The two balloons are then inflated, blocking the esophagus and oropharynx, and isolating the supraglottic space. Ventilation is achieved through a side port between the two balloons.
- **Combitube** – The Combitube is a dual-lumen tube that is blindly inserted into either the esophagus (95% of the time) or the trachea (remaining 5%). If the tip is placed in the esophagus, the oropharyngeal and esophageal balloons can be inflated, and the patient can be ventilated through a side port of the longer lumen, similar to the King LT. If the tip of the tube is placed in the trachea, the patient can be ventilated through the distal tip using the shorter lumen.

25. What are the indications for a surgical airway?

Cricothyrotomy is the surgical airway of choice in the ED for patients with a failed airway. A failed airway is failure to intubate, ventilate, and oxygenate by other means. A cricothyrotomy is performed by making a vertical

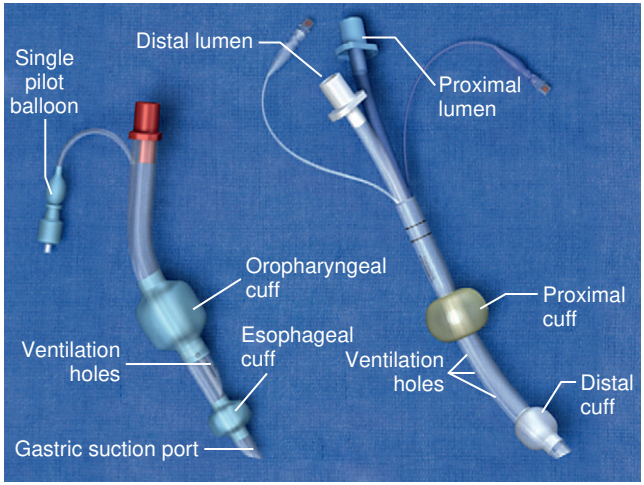

Fig. 3.3 King LT *(left)* and Combitube *(right). (From: Driver BE, Reardon RF. Basic airway management and decision making. In: Roberts JR, Hedges JR, eds. Clinical Procedures in Emergency Medicine. 7th ed. Philadelphia: Elsevier; 2019:59.)*

Table 3.2 Laryngoscope Blade Sizes for Pediatric Intubations

AGE	LARYNGOSCOPE BLADE SIZE
Premature infant	0
Full-term infant	1
Older children	2
Adults	3–4

incision over the cricothyroid membrane, palpating down to and making a horizontal incision through the membrane and inserting an ET tube.

26. What factors make pediatric airway interventions more difficult?

Direct laryngoscopy can be more challenging in the pediatric patient due to anatomic differences, including a relatively large occiput causing neck flexion, a more superior and anterior larynx, a relatively larger tongue, and an epiglottis that is shorter and more difficult to manipulate. Additionally, pediatric patients have higher relative oxygen consumption and lower residual capacity and, therefore, become hypoxic much more quickly than adult patients.

27. How do I know what equipment size to use for pediatric airway interventions?

Laryngoscope sizes can be seen in Table 3.2. Cuffed ET tubes are now recommended for pediatrics. Both cuffed and uncuffed ET tube size can be estimated using tools such as a Broselow-Luten tape, the age-based Handtevy system, or with the following formula:

$$\text{Cuffed ET tube size} = (\text{Age}/4) + 3.5$$

28. What is the surgical airway option for pediatric patients?

Because of anatomic differences in children, including a smaller or absent cricothyroid membrane and an immature larynx, surgical cricothyrotomy is contraindicated in children less than 5–12 years (cutoff age is controversial). The surgical airway of choice in pediatric patients is transtracheal jet ventilation, in which a large-bore needle is inserted through the cricothyroid membrane and high-flow oxygen is delivered. This is only a temporizing measure, as hypoventilation with this device quickly leads to hypercarbia.