

Critical Care Medicine

Principles of Diagnosis and Management in the Adult

FIFTH EDITION

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CRITICAL CARE MEDICINE: PRINCIPLES OF DIAGNOSIS AND
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To our families
Gale, Nicholas, Jenny, and Lena
and
Christa, Bart, Michael, Kate, Walker,
Lauren, Reid, and Meg

Preface

Few fields in medicine have grown, evolved, and changed as rapidly as critical care medicine over the past 40 years. From its origins in the postoperative recovery room and the coronary care unit, the modern intensive care unit (ICU) represents the ultimate example of medicine's ability to supply the specialized personnel and technology necessary to sustain and restore seriously ill persons to productive lives. In more recent years critical care has extended beyond the walls of the ICU to the emergency department where partnerships between intensivists and emergency medicine physicians have been forged. Critically ill patients now may be held for significant time periods in the emergency department, the postoperative recovery room or special critical care holding areas while awaiting an ICU bed. While the field continues to evolve rapidly, sufficient principles, knowledge, and experience have accumulated in the past few decades to warrant the production of a textbook dedicated to adult critical care medicine. We chose to limit the subject matter of our book to the critical care of *adult* patients to allow the production of a comprehensive textbook in a single volume.

This book was envisioned to be multidisciplinary and multi-specialty, authored by acknowledged leaders in the field, and aimed primarily at practicing critical care physicians who spend the better part of their time caring for patients in an ICU. Thus the book would be appropriate for critical care internists as well as for surgical or anesthesia critical care specialists. The goal was to produce the acknowledged "best practice" standard in critical care medicine.

The first edition of the textbook was published in 1995, co-edited by Joe Parrillo and Roger Bone. The book sold exceedingly well for a first edition text. After the untimely death of Roger Bone in 1997, Phil Dellinger joined Joe Parrillo as the co-editor for the second, third, fourth, and now this fifth edition. As co-editors, we have labored to produce a highly readable text that can serve equally well for comprehensive review and as a reference source. We felt that it was important for usability and accessibility to keep the book to a single volume. This was a challenge, because critical care knowledge and technology have expanded significantly during the past decade. By placing emphasis on clear, concise writing and keeping the focus on critical care medicine for the adult, this goal was achieved.

Our view of critical care medicine is mirrored in the organization of the textbook. Modern critical care is a multidisciplinary specialty that includes much of the knowledge and technology contained in many disciplines represented by the classic organ-based subspecialties of medicine, as well as the specialties of surgery and anesthesiology. The book begins with a section consisting of chapters on the technology, procedures, and pharmacology that are essential to the practicing critical care physician. This section is followed by

sections devoted to the critical care aspects of cardiovascular, pulmonary, infectious, renal, metabolic, neurologic, gastrointestinal, and hematologic-oncologic diseases. Subsequent chapters are devoted to important social, ethical, and other issues such as psychiatric disorders, severity of illness scoring systems, and administrative issues in the ICU. This fifth edition has significant content additions and revisions, including new chapter authors for approximately a third of our chapters. Online videos are also available featuring a variety of content areas, including echocardiograms and bedside ultrasounds from a variety of exam sites. Further, a series of questions and answers with rationale for correct and incorrect answers are provided for each chapter to assist readers in board review and to reinforce the important content of each chapter.

Each chapter is designed to provide a comprehensive review of pertinent clinical, diagnostic, and management issues. This is primarily a clinical text, so the emphasis is on considerations important to the practicing critical care physician; also presented, however, are the scientific (physiologic, biochemical, and molecular biologic) data pertinent to the pathophysiology and management issues. We have aimed for a textbook length that is comprehensive but manageable. Substantial references (most now online) are provided for readers wishing to explore subjects in greater detail. We have identified key points and key references to highlight the most important issues within each chapter. Continued popular features of this fifth edition include a color-enhanced design and clinically useful management algorithms.

We have been fortunate to attract a truly exceptional group of authors to write the chapters for *Critical Care Medicine: Principles of Diagnosis and Management in the Adult*. For each chapter, we have chosen a seasoned clinician-scientist actively involved in critical care who is one of a handful of recognized experts on his or her chapter topic. We have continued the international flavor of our authorship. To provide uniformity in content and style, one or both of us have edited and revised each chapter.

We wish to thank the highly dedicated people who provided us with the assistance needed to complete a venture of this magnitude. Our thanks go to Ellen Lawlor, for her administrative assistance; and to the excellent editorial staff at Elsevier, including Anne Snyder, Nancy Duffy, and Rachel McMullen.

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1

Cardiac Arrest and Cardiopulmonary Resuscitation

BRIAN W. ROBERTS, TAPAN KAVI, AND STEPHEN TRZECIAK

CHAPTER OUTLINE

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- Rescue Breathing

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Epidemiology and General Principles

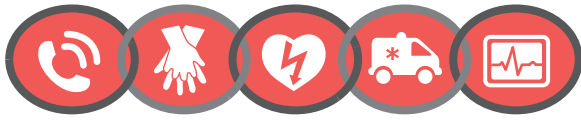
Sudden cardiac arrest is defined as the cessation of effective cardiac mechanical activity as confirmed by the absence of signs of circulation. Sudden cardiac arrest is the most common fatal manifestation of cardiovascular disease and a leading cause of death worldwide. The exact incidence of sudden cardiac arrest is unclear, but in the United States alone, it has been estimated to be as high as 450,000 persons annually.¹ Approximately 21% to 25% of sudden cardiac arrest events are due to pulseless ventricular arrhythmias (i.e., ventricular fibrillation [VF] or pulseless ventricular tachycardia [VT]), whereas the rest can be attributed to other cardiac rhythms (i.e., asystole or pulseless electrical activity [PEA]).^{2,3} Patients who suffer cardiac arrest due to VF or VT have a much higher chance

of surviving the event compared with patients who present with PEA/asystole.⁴ The prognosis is better in patients with ventricular arrhythmias because (1) ventricular arrhythmias are potentially treatable with defibrillation (i.e., “shockable” initial rhythm) to restore circulation, whereas the other initial rhythms are not, and (2) ventricular arrhythmias are typically a manifestation of a cardiac etiology of cardiac arrest (e.g., acute myocardial infarction), whereas the other initial rhythms are more likely to be related to a noncardiac etiology and perhaps an underlying condition that is less treatable. Clinical outcomes for cardiac arrest are poor. Approximately 11% of out-of-hospital cardiac arrest (OHCA)⁵ and 20% of in-hospital cardiac arrest (IHCA) patients survive to hospital discharge.⁶

The basic principles of resuscitation are an integral part of training for many health care providers (HCPs). Because timely interventions for cardiac arrest victims have the potential to be truly lifesaving, it is especially important for critical care practitioners to have a sound understanding of the evaluation and management of cardiac arrest. A number of critical actions (*chain of survival*) must occur in response to a cardiac arrest event. The chain of survival paradigm (Fig. 1.1) for the treatment of cardiac arrest remained unchanged in the 2015 American Heart Association (AHA) Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care from 2010 and is similar to other cardiac arrest guidelines. The chain of survival paradigm consists of five separate and distinct elements: immediate recognition that cardiac arrest has occurred and activation of the emergency response system; application of effective cardiopulmonary resuscitation (CPR); early defibrillation (if applicable); advanced cardiac life support; and initiation of postresuscitation care (e.g., targeted temperature management).⁷⁻⁹ When the chain of survival is implemented effectively survival for VF, OHCA can exceed 45%.³

Cardiopulmonary Resuscitation

For CPR to be effective in restoring spontaneous circulation, it must be applied immediately at the time of cardiac arrest. Therefore immediate recognition that a cardiac arrest has occurred and activation of the emergency response system are essential. Patients become unresponsive at the time of cardiac arrest. Agonal gasps may be observed in the early moments after a cardiac arrest event, although normal breathing ceases. Pulse checks (i.e., palpation of femoral or carotid arteries for detection of a pulse) are often



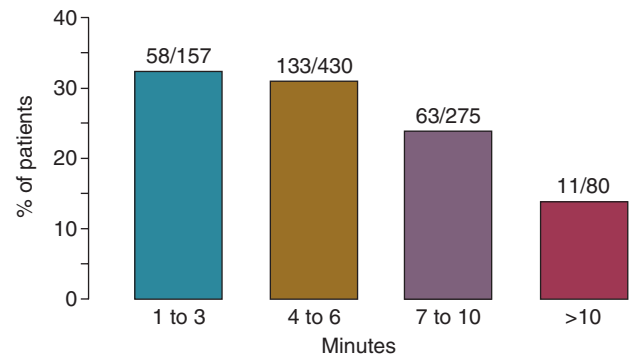
• **Fig. 1.1** The American Heart Association chain of survival paradigm.⁸⁹ This figure represents the critical actions needed to optimize the chances of survival from cardiac arrest. The links (from left to right) include (1) immediate recognition of cardiac arrest and activation of the emergency response system; (2) early and effective cardiopulmonary resuscitation; (3) defibrillation (if applicable); (4) advanced cardiac life support; and (5) post-cardiac arrest care (including target temperature management if appropriate). (From Travers AH, Rea TD, Bobrow BJ, et al. Part 4: CPR overview: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(18 suppl 3):S676–S684.)

unreliable, even when performed by experienced HCPs.¹⁰ Because delays in initiating CPR are associated with worse outcome, and prolonged attempts to detect a pulse may result in a delay in initiating CPR, prolonged pulse checks are to be avoided. CPR should be started immediately if the patient is unresponsive and either has agonal gasps or is not breathing.⁷

Chest Compressions

In CPR, chest compressions are used to circulate blood to the heart and brain until a pulse can be restored. High-quality chest compressions are vital for successful resuscitation. The mechanism by which chest compressions generate cardiac output is through an increase in intrathoracic pressure plus direct compression of the heart. As in 2010, the 2015 AHA guidelines recommend HCPs initiate the CPR sequence with chest compressions instead of rescue breaths (i.e., compressions-airway-breathing [C-A-B] vs. airway-breathing-compressions [A-B-C]) to minimize the time to chest compressions.⁷ With the patient lying in the supine position, the rescuer applies compressions to the lower half of the patient's sternum. The heel of one hand is placed over the lower half of the sternum and the heel of the other hand on top in an overlapping and parallel fashion. In addition to manual CPR, there has been an increase in the use of mechanical CPR devices. Studies have not demonstrated a difference in patient outcomes between manual and mechanical CPR.^{8,11}

The recommended compression depth in adults is at least 2 inches (5 cm) while avoiding chest compression depths greater than 2.4 inches (6 cm).^{7–9} The recommended rate of compression per the 2010 AHA guidelines was *at least* 100 compressions per minute.¹² The 2015 AHA guidelines now recommend a rate of 100 to 120 compressions per minute.⁷ These new AHA guidelines are in line with the 2010 and 2015 European Resuscitation Council guidelines for resuscitation, as well as the 2015 International Liaison Committee on Resuscitation recommendations.^{8,9,13} In addition, incomplete recoil of the chest impairs the cardiac output that is generated. Incomplete recoil occurs when HCPs lean on the chest wall between compressions; thus HCPs should avoid leaning to allow the chest wall to recoil completely between compressions. Owing to rescuer fatigue, the quality of chest compressions predictably decreases as the time providing chest compressions increases, and the persons providing chest compressions (even experienced HCPs) may not perceive fatigue or a decrease in the quality of their compressions.¹⁴ Therefore it is recommended



• **Fig. 1.2** Relationship between the time interval before attempted defibrillation and the proportion of patients discharged from the hospital alive after out-of-hospital cardiac arrest. (Modified from Weaver WD, Cobb LA, Hallstrom AP, et al. Factors influencing survival after out-of-hospital cardiac arrest. *J Am Coll Cardiol*. 1986;7:752–757.)

that rescuers performing chest compressions rotate every 2 minutes.^{7,9}

The quality of CPR is a critical determinant of surviving a cardiac arrest event.¹⁵ Minimization of interruptions in chest compressions is imperative. Interruptions in chest compressions during CPR have been quite common historically, and the “hands off” time has been shown to comprise a substantial amount of the total resuscitation time.¹⁵ The proportion of time chest compressions are performed during a cardiac arrest has been termed chest compression fraction. A higher chest compression fraction has been associated with increased survival in VF cardiac arrest,¹⁶ and increased return of spontaneous circulation (ROSC) in non-VF cardiac arrest.¹⁷ Guidelines recommend a chest compression fraction as high as possible, with a goal of at least 60%.^{7,9} A chest compression fraction of 80% is likely ideal and achievable in many settings.¹⁸ Potential reasons for decreased chest compression fraction include pulse checks, rhythm analysis, switching compressors, procedures (e.g., airway placement), and pauses before defibrillation (“preshock pause”). All of these potential reasons for interruptions should be minimized. Pauses related to rotating compressors, pulse checks, and delivering rescue breaths should take no longer than 10 seconds.^{7,9} Eliminating (or minimizing) preshock pauses has been associated with a higher likelihood of ROSC and improved clinical outcome.^{19,20}

Defibrillation

The next critically important action in the resuscitation of patients with cardiac arrest caused by pulseless ventricular arrhythmias (i.e., VF or pulseless VT) is rapid defibrillation. Delays in defibrillation are clearly deleterious, with a sharp decrease in survival as the time to defibrillation increases.²¹ With the advent of automatic external defibrillators and their dissemination into public places, both elements of effective CPR (effective chest compressions and rapid defibrillation) can be performed by lay rescuers in the field for patients with OHCA. Fig. 1.2 shows the importance of rapid defibrillation, with decreasing success of resuscitation with increasing time to defibrillation. Thus defibrillation should be performed as soon as possible. The amount of energy for defibrillation should be based on the specific manufacturer's guidance. To increase the chest compression fraction, chest compressions should resume immediately after defibrillation for adult cardiac arrest in any setting.^{7,9}

Rescue Breathing

Recommendations regarding ventilation during CPR depend on the rescuer (i.e., trained HCPs vs. layperson).^{7,9} For trained HCPs, the recommended ventilation strategy is a cycle of 30 chest compressions to 2 breaths until an endotracheal tube is placed, and then continuous chest compressions with 1 breath every 6 seconds after the endotracheal tube is placed. Excessive ventilations can be deleterious from a hemodynamic perspective owing to increased intrathoracic pressure and reduction in the cardiac output generated by CPR and thus should be avoided during resuscitation.²² Excessive ventilation could also potentially result in alkalemia. For OHCA it is reasonable for emergency medicine service providers to delay positive-pressure ventilation while providing up to 3 cycles of 200 continuous chest compressions with passive oxygenation.⁷

For laypersons who are attempting CPR in the field for a victim of OHCA, rescue breathing is no longer recommended. Rather, the recommended strategy is compression-only (or hands-only) CPR.^{7,9} The rationale is that compression-only CPR can increase the number of effective chest compressions that are delivered to the patient (i.e., minimizes interruptions for rescue breaths), and does not require mouth-to-mouth contact. Mouth-to-mouth contact is one of the perceived barriers to CPR in the field. By removing this element, the hope is that an increase in attempts at bystander CPR will result. Hands-only CPR has been found to be noninferior to conventional CPR including rescue breaths for victims of OHCA^{23–25}; thus hands-only CPR has become the preferred technique to teach lay rescuers. Fig. 1.3 displays the AHA algorithm for adult basic life support.

Advanced Cardiac Life Support

There are several additional elements of resuscitation that are intended specifically for trained HCPs (e.g., advanced cardiac life support [ACLS]), and specifically this includes pharmacologic therapy. Fig. 1.4 displays the AHA algorithm for ACLS.²⁶

It is notable that the impact of recommended ACLS therapies on outcome from cardiac arrest remains a matter of debate. Some studies have shown that ACLS interventions did not improve clinical outcomes when compared to basic life support alone.²⁷

Vasopressors

The primary goal of pharmacologic interventions is to assist the achievement and maintenance of spontaneous circulation. The mainstay of pharmacologic interventions is vasopressor drugs. It is reasonable to administer epinephrine (1 mg) by intravenous (IV) or intraosseous (IO) route every 3 to 5 minutes during CPR until ROSC is achieved.^{8,26,28} If IV/IO access cannot be established, epinephrine could be administered via an endotracheal tube, but at a higher dose (2–2.5 mg). The optimal timing of epinephrine administration is currently unclear; however, observational studies have demonstrated that early administration of epinephrine is associated with improved outcomes in nonshockable rhythms.^{29,30}

Vasopressin as a substitute for epinephrine was removed from the 2015 AHA guidelines. One study found the use of intracardiac arrest vasopressin (20 IU/CPR cycle), epinephrine (1 mg/CPR cycle), and methylprednisolone (40 mg during first CPR cycle) followed by postcardiac arrest hydrocortisone (300 mg daily for 7 days maximum and gradual taper) increased the probability of successful ROSC and discharge with good neurologic outcome³¹;

however, the evidence for this practice is not currently strong enough to recommend its routine use.²⁶

Antiarrhythmic Drugs

Amiodarone is the preferred antiarrhythmic agent for refractory VF/VT cardiac arrest.⁸ Refractory VF/VT refers to VF or pulseless VT that persists or recurs after one or more defibrillation attempts. It is unlikely that antiarrhythmic drug therapy itself will convert refractory VT/VF; rather, the goal of antiarrhythmic drug therapy is to aid in reestablishing and maintaining an organized perfusing rhythm in conjunction with subsequent defibrillation. The recommended dose of amiodarone is 300 mg IV/IO for the first dose, 150 mg IV/IO for the second dose.^{26,28} Of note, antiarrhythmic drug therapy has not been demonstrated to improve survival or neurologic outcomes after VF/VT cardiac arrest.

Other Drug Therapies

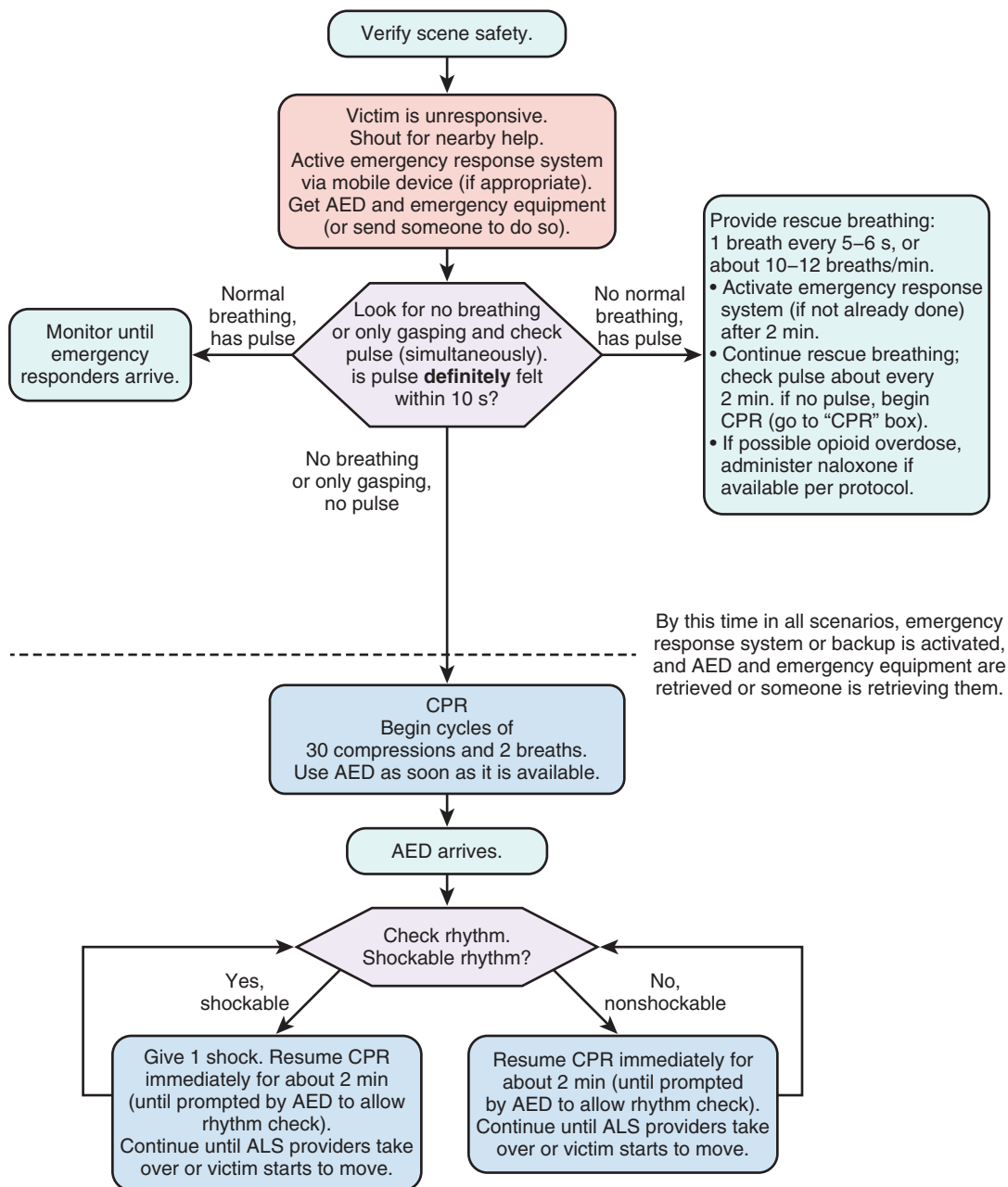
The use of atropine for PEA/asystole was removed from the ACLS guidelines in 2010. Along these lines, there is also insufficient evidence to recommend routine administration of sodium bicarbonate or calcium during CPR.^{28,32} In the setting of confirmed or suspected pulmonary embolism, limited evidence suggests that systemic thrombolysis administration during ongoing CPR is associated with ROSC and short-term survival,³³ with earlier administration associated with greater benefit.³⁴ However, in the setting of presumed cardiac etiology of cardiac arrest there is currently no evidence to support clinical benefit.³⁵ The decision to administer systemic thrombolytics during cardiac arrest may be considered in cases with a strong suspicion for pulmonary embolism, or cardiac etiology without immediate access to percutaneous coronary intervention.

End-Tidal Carbon Dioxide

End-tidal carbon dioxide (ETCO₂) may reflect cardiac output and pulmonary blood flow. ETCO₂ can be measured during CPR to monitor the quality of chest compressions in intubated patients. An ETCO₂ less than 10 mm Hg after 20 minutes of high-quality CPR has been demonstrated to be predictive of mortality,^{36,37} whereas an ETCO₂ greater than 20 mm Hg was associated with survival to hospital discharge.³⁶ Although predictive of outcome, a low ETCO₂ should not be used alone to discontinue CPR efforts. It is also important to realize a low ETCO₂ could be a result of bronchospasm, plugging or kinking of the endotracheal tube, hyperventilation, or an air leak in the airway.²⁶ A sudden, sustained increase in ETCO₂ (i.e., >40 mm Hg) during chest compressions can be an indication of ROSC.^{26,28,38} ETCO₂ monitoring in non-intubated patients may not accurately reflect the true ETCO₂ level and should not be used as an adjunct prognostication tool.

Extracorporeal CPR

Extracorporeal CPR (ECPR) refers to venoarterial extracorporeal membrane oxygenation and cardiopulmonary bypass during cardiac arrest. In theory ECPR could be used as a bridge to allow physicians time to treat reversible causes of cardiac arrest. To date there has not been a randomized control trial comparing ECPR to conventional CPR for prolonged resuscitations. The limited case reports and observational studies do not provide sufficient evidence to recommend the routine use of ECPR; however, it may be reasonable



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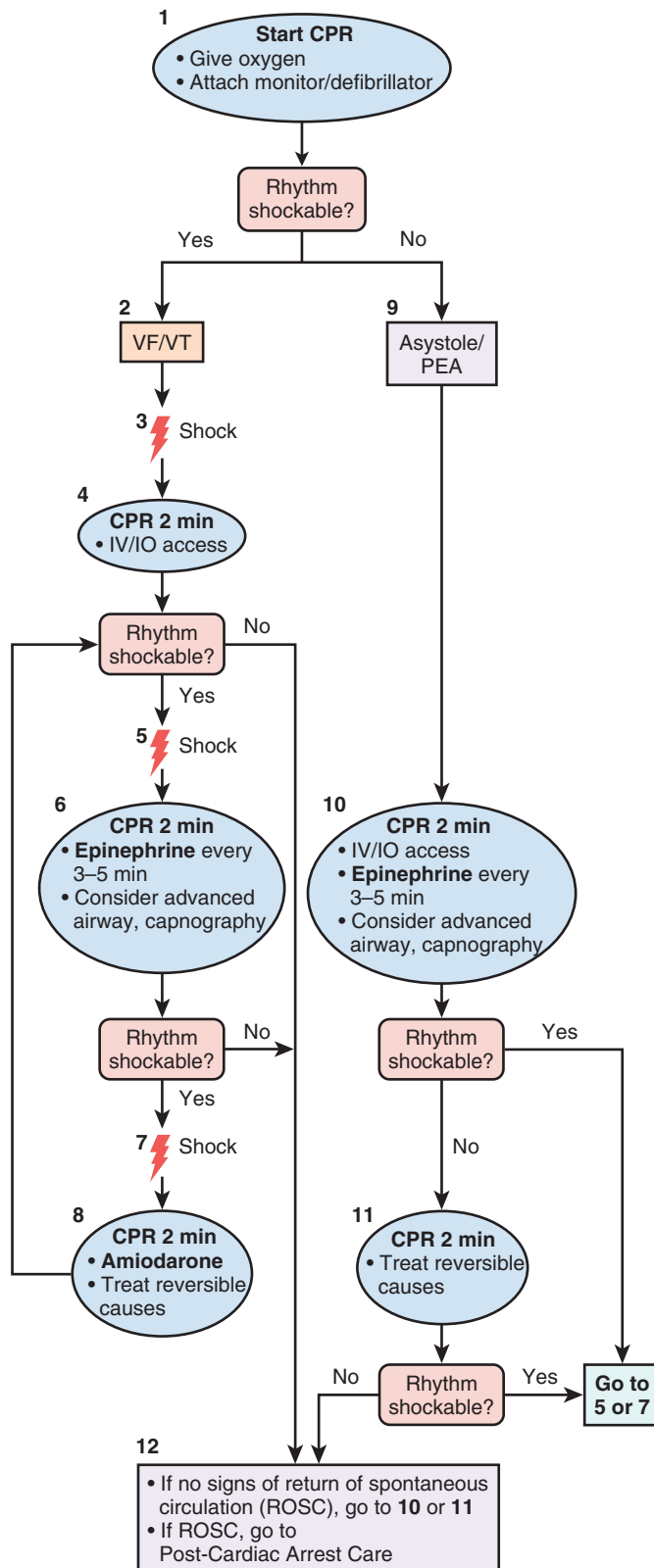
• **Fig. 1.3** American Heart Association adult Basic Life Support (BLS) algorithm. AED, Automated external defibrillator; ALS, advanced life support; CPR, cardiopulmonary resuscitation. (From Kleinman ME, Brennan EE, Goldberger ZD, et al. Part 5: adult basic life support and cardiopulmonary resuscitation quality: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(18 suppl 2):S414–S435.)

to consider ECPR for specific patients with a potentially reversible etiology of cardiac arrest.^{26,28}

Reversible Causes

When managing a cardiac arrest it is important to consider the potential etiologies of the arrest. In many situations it is not possible to maintain an organized perfusing rhythm until the underlying cause of the cardiac arrest is reversed. A frequently used mnemonic

for common potential reversible causes involves the “Hs and Ts.”²⁸ The “Hs” include hypoxia, hypovolemia (including hemorrhage), hyperkalemia, hypokalemia, hypoglycemia, hydrogen ions (i.e., metabolic acidosis), and hypothermia. The “Ts” include thrombosis (i.e., coronary occlusion and pulmonary embolism), tension pneumothorax, cardiac tamponade, and ingestion of therapeutic or toxic substances. Once the etiology of the cardiac arrest is identified (or strongly presumed), treatment should be aimed at reversing the specific cause.

**CPR Quality**

- Push hard (2 inches [5 cm]) and fast (100–120/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 min or sooner if fatigued
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
 - If $PETCO_2 < 10$ mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
 - If relaxation phase (diastolic) pressure < 20 mm Hg, attempt to improve CPR quality

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (e.g., initial dose of 120–200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered
- **Monophasic:** 360 J

Drug Therapy

- **Epinephrine IV/IO Dose:** 1 mg every 3–5 min
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus
Second dose: 150 mg

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 s (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in $PETCO_2$ (typically ≥ 40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

• **Fig. 1.4** American Heart Association Advanced Cardiac Life Support (ACLS) algorithm. CPR, Cardiopulmonary resuscitation; ET, endotracheal; IO, intraosseous; IV, intravenous; PEA, pulseless electrical activity; $PETCO_2$, partial pressure of end tidal carbon dioxide; pVT, pulseless ventricular tachycardia; ROSC, return of spontaneous circulation; VF, ventricular fibrillation; VT, ventricular tachycardia. (From Link MS, Berkow LC, Kudenchuk PJ, et al. Part 7: adult advanced cardiovascular life support: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(18 suppl 2):S444–S464.)

Postresuscitation Care

Even if ROSC is achieved with CPR and defibrillation, cardiac arrest victims are at extremely high risk of dying in the hospital, and many who survive sustain permanent crippling neurological sequelae. Approximately 50% to 60% of patients successfully resuscitated from OHCA do not survive. After ROSC global ischemia/reperfusion (I/R) injury results in potentially devastating neurologic disability. The primary cause of death among postresuscitation patients is brain injury. However, clinical trials have shown that targeted temperature management (TTM) after ROSC can improve outcomes. These landmark clinical trials have dramatically transformed the classical thinking about anoxic brain injury after cardiac arrest; this condition is, in fact, *treatable*. Early therapeutic interventions such as TTM initiated in the post-ROSC period can improve the trajectory of the long-term disease course. Accordingly, the postresuscitation care is now considered to be a crucial fifth link in the chain of survival paradigm (see Fig. 1.1).⁷⁻⁹

General Approach

Post-cardiac arrest syndrome is a heterogeneous disease process with varying precipitating factors and underlying etiologies. As such, post-cardiac arrest management must be tailored on a case-by-case basis for each specific patient. For patients resuscitated from cardiac arrest, admission to a critical care unit with the following capabilities should be considered³⁹:

- Critical care support to optimize cardiovascular indices and vital organ perfusion, and prevent repeat cardiac arrest (or provide rapid treatment of rearrest if it occurs)
- Interventional cardiac catheterization for possible percutaneous coronary intervention (PCI) if needed
- TTM for at least 24 hours in attempts to prevent permanent neurologic injury
- Systematic application of an evidence-based approach to neurologic prognostication to refrain from inappropriately early final determinations of poor neurologic prognosis (i.e., to prevent inappropriately early withdrawal of life support before the neurologic outcome can be known with certainty).

Hemodynamic Support

I/R triggers profound systemic inflammation. In clinical studies, ROSC has been associated with sharp increases in circulating cytokines and other markers of the inflammatory response. Accordingly, some investigators have referred to the post-cardiac arrest syndrome as a “sepsis-like” state. The clinical manifestations of the systemic inflammatory response may include marked hemodynamic derangements such as sustained arterial hypotension similar to septic shock. Hemodynamic instability occurs in approximately 50% of patients who survive to intensive care unit admission after ROSC, and thus the need for aggressive hemodynamic support (e.g., continuous infusion of vasoactive agents and perhaps advanced hemodynamic monitoring) should be anticipated.⁴⁰

In addition to a systemic inflammatory response, an equally important contributor to post-ROSC hemodynamic instability is myocardial stunning. Severe, but potentially reversible, global myocardial dysfunction is common after ROSC. The etiology is thought to be I/R injury, but treatment with defibrillation (if applied) could also contribute. Although the myocardial dysfunction occurs in the absence of an acute coronary event, myocardial ischemia may be an ongoing component of myocardial depression

if an acute coronary syndrome caused the cardiac arrest. Severe myocardial stunning may last for hours, but it often improves by the 24-hour mark after ROSC. An echocardiogram may be helpful in hemodynamic assessment after ROSC to determine if global myocardial depression is present, as this may affect decisions on vasoactive drug support (e.g., dobutamine) or mechanical augmentation (e.g., intraaortic balloon counterpulsation) until the myocardial function recovers. However, when needed, clinicians should be aware that β -adrenergic agents may increase the likelihood of dysrhythmia.

Observational studies have demonstrated postresuscitation arterial hypotension to be associated with sharply lower survival,^{40,41} and a post-ROSC mean arterial blood pressure greater than 70 mm Hg to be associated with improved neurologic outcome at hospital discharge.⁴² However, it is currently unclear whether a specific blood pressure target or other hemodynamic goals are beneficial.⁴³ Expert opinion (and clinical intuition) suggests that hemodynamics and organ perfusion should be optimized, and rapidly reversing and preventing hypotensive after ROSC is recommended.^{44,45} Whether or not postresuscitation hypotension has a cause-and-effect relationship with worse neurologic injury or is simply a marker of the severity of the I/R injury that has occurred remains unclear.

Oxygenation and Ventilation

Given the ongoing I/R injury during the early period after ROSC, hypoxia should be avoided in all post-cardiac arrest patients. Exposure to hyperoxia (excessively high partial pressure of arterial oxygen [PaO₂]) has also been associated with poor clinical outcome among adult patients resuscitated from cardiac arrest and admitted to an intensive care unit.⁴⁶ These data corroborate the findings of numerous laboratory studies in animal models in which hyperoxia exposure after ROSC worsens brain histopathologic changes and neurologic function.⁴⁷⁻⁵¹ A paradox may exist regarding oxygen delivery to the injured brain, where inadequate oxygen delivery can exacerbate cerebral I/R injury, but excessive oxygen delivery can accelerate formation of oxygen free radicals and subsequent reperfusion injury. Although the results of observational studies of hyperoxia are mixed^{52,53} and no interventional studies have been performed, expert opinion advocates initially using the highest available oxygen concentration after ROSC to prevent hypoxia, and once the arterial oxygen saturation or PaO₂ can be measured, the fraction of inspired oxygen can be titrated down as much as possible while maintaining an arterial oxygen saturation of at least 94% to limit unnecessary exposure to an excessively high postresuscitation PaO₂.^{45,54}

Post-ROSC partial pressure of arterial carbon dioxide (PaCO₂) levels have been demonstrated to be associated with clinical outcomes. Specifically, hypocapnia is associated with worse neurologic outcome.⁵⁵⁻⁵⁷ It is possible that hypocapnia induces cerebral vasoconstriction and decreased cerebral blood flow, resulting in worsening cerebral ischemia. The effects of hypercapnia on clinical outcomes are currently unclear.⁵⁸ Current recommendations advocate maintaining normocapnia (i.e., PaCO₂ 35–45 mm Hg) after cardiac arrest.⁵⁴

Seizures

Seizures are not uncommon after anoxic brain injury. Routine seizure prophylaxis is not currently recommended in post-cardiac arrest syndrome. However, it is important to be vigilant in clinical assessment for any motor responses that could represent seizure

activity so that it can be treated promptly with anticonvulsant medications. Patients with status epilepticus may not have clinically detectable signs of seizure. Therefore continuous electroencephalography monitoring (if available) can be useful, especially if continuous administration of neuromuscular blocking agents becomes necessary for any reason.^{45,54}

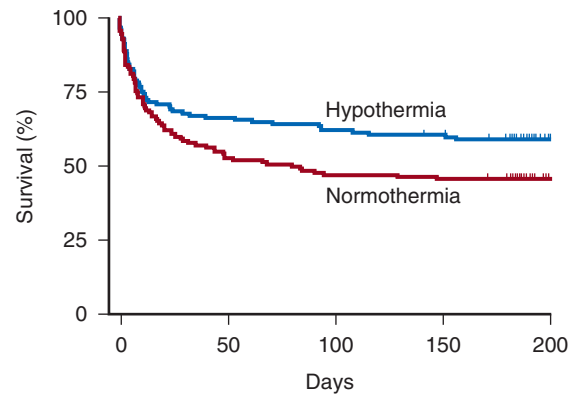
Cardiac Catheterization

Acute coronary syndrome is the most common etiology of sudden cardiac arrest. Clinicians should have a high clinical suspicion of acute myocardial ischemia as the inciting event for the cardiac arrest when no other obvious etiology of cardiac arrest is apparent. Although ST-segment myocardial infarction is an obvious indication for PCI, other subtler electrocardiogram abnormalities may indicate that an acute ischemic event caused the cardiac arrest. In patients with no obvious noncardiac etiology of cardiac arrest, a clinical suspicion of coronary ischemia, and an abnormal electrocardiogram after ROSC, consultation with an interventional cardiologist is encouraged and coronary angiography should be considered.⁵⁴ There is insufficient evidence to recommend PCI based on the presenting cardiac arrest rhythm (i.e., VF vs. non-VF). Recent studies and experience indicate that inducing TTM simultaneously with emergent percutaneous coronary intervention is feasible.

Targeted Temperature Management

TTM, previously referred to as therapeutic hypothermia or mild therapeutic hypothermia, is a treatment strategy of rapidly reducing the patient's body temperature after ROSC for the purposes of protection from neurologic injury. The body temperature is typically reduced to 32°C to 36°C for at least 24 hours. After ROSC the severity of the reperfusion injury can be mitigated, despite the fact that the initial ischemic injury has already occurred. Reperfusion injury refers to tissue and organ system injury that occurs when circulation is restored to tissues after a period of ischemia, and is characterized by inflammatory changes and oxidative damage that are in large part a consequence of oxidative stress. Neuronal cell death after I/R injury is not instantaneous, but rather a dynamic process. In animal models of cardiac arrest, brain histopathologic changes may not be found until 24 to 72 hours after cardiac arrest.⁵⁹ This indicates that a distinct therapeutic window of opportunity exists. In theory, TTM may protect the brain by attenuating or reversing all of the following pathophysiologic processes: disruption of cerebral energy metabolism, mitochondrial dysfunction, loss of calcium ion homeostasis, cellular excitotoxicity, oxygen free radical generation, and apoptosis.

The two original clinical trials of TTM targeting 32°C to 34°C were published in 2002.^{60,61} These trials showed improved outcomes with TTM targeting 32°C to 34°C for comatose survivors of witnessed OHCA with VF as the initial rhythm. The survival data for the largest of these clinical trials (i.e., the Hypothermia After Cardiac Arrest Study Group) appear in Fig. 1.5. For patients with OHCA and a non-VF initial rhythm or IHCA, there are no randomized clinical trials and observational data have conflicting results. In 2013, a randomized control trial of 939 unconscious adults with OHCA of presumed cardiac etiology found no difference between neurologic outcomes and survival at 6 months when temperature was controlled at 36°C versus 33°C (Table 1.1).⁶² It is currently unclear if certain subpopulations of post-cardiac arrest patients benefit from lower or higher temperatures.



No. at risk					
Hypothermia	137	92	86	83	11
Normothermia	138	74	66	64	9

• **Fig. 1.5** Cumulative survival in the normothermia and hypothermia groups from a randomized trial of therapeutic hypothermia in comatose survivors of out-of-hospital cardiac arrest due to ventricular fibrillation. Censored data are indicated by tick marks. (From Hypothermia After Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med* 2002;346(8):549–556.)

The current AHA guidelines for CPR and Emergency Cardiovascular Care recommend at least 24 hours of TTM targeting 32°C to 36°C for comatose survivors of OHCA caused by VF or pulseless VT, as well as non-VF/pulseless VT and IHCA.⁵⁴ These recommendations for non-VF/pulseless VT and IHCA are stronger than the 2010 AHA recommendations, the 2015 European Resuscitation Council and European Society of Intensive Care Medicine Guidelines for Post-resuscitation Care, and the 2015 International Liaison Committee on Resuscitation recommendations, which state that TTM may be considered for victims of IHCA and other arrest rhythms.^{8,45,63}

Appropriate selection of candidates for TTM is clearly important. If a patient does not follow verbal commands after ROSC is achieved, this indicates that the patient is at risk for brain injury and TTM should be strongly considered. If a patient is clearly following commands immediately after ROSC, then significant brain injury is less likely and it is probably reasonable to withhold TTM. There are multiple potential methods for inducing TTM, including specialized external or intravascular cooling devices for TTM, or a combination of conventional cooling methods such as ice packs, cooling blankets, and cold (4°C) intravenous saline infusion. Compared with the use of specialized cooling devices, overshoot (body temperature <31°C) is a not uncommon occurrence with use of ice packs, cooling blankets, and cold saline.⁶⁴ Regardless of which method is used, effective achievement of target temperature may be aided by the use of a uniform physician order set for TTM induction.⁶⁵ The current recommendation is to maintain TTM for at least 24 hours.^{8,45,54} Whether or not a longer duration of therapy could be beneficial is currently unknown.

Shivering with TTM induction is very common and should be anticipated regardless of which target temperature is chosen.⁶² Shivering can be detrimental to the patient by making goal temperature more difficult (or impossible) to achieve. Therefore immediate recognition and treatment of shivering is imperative. Adequate sedation and analgesics are an essential component of TTM, especially during the induction phase, and often the administration of additional sedative and/or opioid agents will be

TABLE 1.1 Neurologic Outcome and Mortality at 6 Months With Temperature Control in Comatose Survivors of Cardiac Arrest^a

Outcome	33°C Group	36°C Group	Hazard Ratio or Risk Ratio (95% CI) ^b	P Value
	No./total No. (%)	No./total No. (%)		
Primary outcome: deaths at end of trial	235/473 (50)	225/466 (48)	1.06 (0.89–1.28)	0.51
Secondary outcomes:				
Neurologic function at follow-up ^c				
CPC of 3–5	251/469 (54)	242/464 (52)	1.02 (0.88–1.16)	0.78
Modified Rankin scale score of 4–6	245/469 (52)	239/464 (52)	1.01 (0.89–1.14)	0.87
Deaths at 180 days	226/473 (48)	220/466 (47)	1.01 (0.87–1.15)	0.92

^aThe study shows the 6-month outcome and mortality for a randomized trial of temperature control at 33°C vs. 36°C in comatose survivors of out-of-hospital cardiac arrest with presumed cardiac etiology.

^bThe hazard ratio is shown for the primary outcome, and risk ratios are shown for the secondary outcomes.

^cThe neurologic follow-up was specified in the protocol to be performed at 180 days ± 2 weeks, but the time to follow-up was in some cases several weeks longer for logistic reasons. The Cerebral Performance Category (CPC) scale ranges from 1 to 5, with 1 representing good cerebral performance or minor disability, 2 moderate cerebral disability (function is sufficient for independent activities of daily life), 3 severe cerebral disability, 4 coma or vegetative state, and 5 brain death. Scores on the modified Rankin scale range from 0 to 6, with 0 representing no symptoms, 1 no clinically significant disability despite some symptoms, 2 slight disability (patient is able to look after own affairs without assistance), 3 moderate disability (patient requires some help but is able to walk unassisted), 4 moderately severe disability (patient is unable to attend to own bodily needs), 5 severe disability (patient is bedridden), and 6 death.

CI, Confidence interval.

From Nielsen N, Wetterslev J, Cronberg T, et al. Targeted temperature management at 33 degrees C versus 36 degrees C after cardiac arrest. *N Engl J Med*. 2013;369(23):2197-2206.

sufficient to ameliorate shivering. Magnesium sulfate can also be used to reduce the shivering threshold.⁶⁶ If shivering persists despite sedatives or opioid agents and magnesium, neuromuscular blocking agents may be required. If neuromuscular blocking agents are used, they are often necessary only in the induction phase of TTM when the temperature is dropping at a fast rate. Once the target temperature is achieved, patients often stop shivering. It is prudent to try to limit neuromuscular blocking agents to the induction phase of TTM, and they likely can be withheld thereafter. Unnecessarily prolonged neuromuscular blockade should be avoided because prolonged neuromuscular weakness may persist after discontinuation, and this could potentially make the patient's neurologic assessment at a later time point more challenging. When using neuromuscular blocking agents for the induction of TTM, their administration may be titrated to the resolution of shivering rather than complete paralysis, which may result in the administration of a much lower dose of neuromuscular blocker.

A number of potential complications are possible with TTM, including bradycardia, a “cold diuresis” resulting in hypovolemia and electrolyte derangements, hyperglycemia, coagulopathy, and perhaps increased risk of secondary infection. However, these potential complications are often not severe when they do occur, and in terms of risk-benefit determinations, the risk of anoxic brain injury usually greatly outweighs the risks of complications. Specifically, bradycardia is a normal response to hypothermia. It is typically not recommended to treat bradycardia during TTM as long as the blood pressure, lactate, mixed venous saturation, and urine output are sufficient, even with heart rates less than 40 beats/min.⁴⁵ In the TTM trial 33°C was associated with a lower heart rate, increased need for vasopressor support, and increased incidence of hypokalemia compared with 36°C.^{41,62} There was no difference in the rate of bleeding complications, infection, other cardiac arrhythmias (i.e., atrial fibrillation/flutter, tachycardia, bradycardia requiring pacing, and VT/VF), or other electrolyte derangements (i.e., hypomagnesemia, hypophosphatemia, and hypoglycemia).⁶²

Fever is not uncommon in the post-cardiac arrest population because of the intense proinflammatory response to ischemia/

reperfusion. The development of hyperthermia after completion of TTM (i.e., rebound hyperthermia) is associated with mortality and poor neurologic outcome.⁶⁷ Fever is clearly detrimental in brain-injured patients because it increases the cerebral metabolic rate; it should be treated aggressively after completion of TTM and in patients who do not receive TTM with antipyretic therapies and other techniques (e.g., cooling blankets).

Neurologic Prognostication

Neurologic prognostication after cardiac arrest is challenging, especially with the advent of TTM. Recovery of brain function—and correspondingly the findings on neurologic examination—are delayed with TTM. This delay is likely because of modulation of brain injury and slowed metabolism of sedative medications.⁶⁸ Neurologic prognostication immediately (e.g., first 24 hours) after resuscitation from cardiac arrest is especially unreliable. Among patients who are initially comatose after ROSC, 25% to 48% could potentially have a favorable outcome, especially if TTM is used.⁶² In general, the recommended approach is to wait a minimum of 72 hours after ROSC before neurologic prognostication.^{8,45,54} For neurologic examination findings to be used for neuroprognostication, and specifically for the determination of poor neurologic prognosis, the “false-positive rate” (FPR) (i.e., prognostication of a poor outcome that ultimately proves not to be poor), should approach zero. The reason it is imperative for a neurologic examination finding to show an FPR approaching zero is to prevent premature discontinuation of life support for patients who are potentially salvageable. Neurologic examination findings by themselves may not have an FPR of zero. Hence, a multimodal approach using a combination of clinical examination, neuroimaging, neurophysiologic tests, and biomarkers may be helpful for neurologic prognostication.⁶⁹

Clinical Examination

Motor responses to pain, brainstem reflexes, and myoclonus remain the standard of clinical assessment even after TTM.⁶⁹ Bilateral absence of pupillary reflexes at 72 hours after cardiac arrest is

highly predictive of poor neurologic outcome with or without TTM with an FPR of about 0.5%.^{70–73} The bilateral absence of corneal reflexes at 72 hours is also predictive of poor neurologic outcome, but with a higher FPR (5%) compared with pupillary reflexes.^{70,72} The utility of absent or extensor motor response to pain at 72 hours for the determination of poor prognosis is diminished with TTM.^{70,73} Similarly, the use of early post-anoxic myoclonus as a predictor of poor neurologic outcome after TTM is not sufficiently reliable, with recent reports of patients recovering well despite early myoclonus.^{74–76} Myoclonus accompanied by malignant electroencephalogram findings is a more reliable predictor of poor neurologic outcome.⁷⁷

Neuroimaging

Loss of gray-white matter differentiation on a computed tomography (CT) scan of the brain has been shown to be a reliable predictor of poor outcome, with an FPR of zero in multiple studies, but the sensitivity of this CT finding remains very low.^{78,79} Although it is a higher-resolution study, magnetic resonance imaging has not been shown to be more useful than CT with respect to prognostication. Abnormalities on diffusion and apparent diffusion coefficient sequence of brain magnetic resonance imaging have been evaluated across multiple studies with variable results regarding utility in neuroprognostication.⁶⁹ In general, data on the use of neuroimaging for neuroprognostication are few compared with clinical and neurophysiologic testing; thus clinical and neurophysiological testing are considered more reliable.⁷²

Neurophysiological Testing

EEG findings such as low voltage or suppressed background, burst suppression pattern, or discontinuous background are suggestive of poor outcome with FPR approaching zero.^{80,81} Absence of reactivity on EEG and epileptiform discharges after TTM have been shown to be predictive of a poor outcome with an FPR of 7% and 9%, respectively.⁸² Upper extremity somatosensory evoked potential, through stimulation of the median nerve and recording of response at the sensory cortex, is well studied and validated,

both with and without TTM. The absence of bilateral cortical responses (N20s) have been shown to be highly predictive of poor outcome (FPR of 0% in multiple studies), but the presence of cortical response does not predict good outcome.^{72,83}

Biomarkers

Serum neuron-specific enolase, a marker of neuronal damage, has been the most extensively studied biomarker as a prognostic tool after cardiac arrest. Before the advent of TTM, serum neuron-specific enolase concentration of more than 33 µg/mL was found to be highly predictive of poor outcome (FPR of 0%) when measured between 24 and 72 hours after cardiac arrest.⁸⁴ Now in the TTM era, the serum concentration cutoff value seems more difficult to determine because multiple studies have shown high FPR even with higher cutoffs.^{70,77,85–87}

New Studies

Long-latency somatosensory evoked potentials, a measurement of cerebral oxygenation and several new blood biomarkers, are among the upcoming promising modalities to improve prognostication after cardiac arrest. At this time, these modalities are considered investigational.

One of the major limitations of all studies regarding neuroprognostication after cardiac arrest is the problem of self-fulfilling prophecy—that is, the presence of the neurologic finding or positive test influences clinician perception of severe anoxic injury and poor prognosis, leading to a withdrawal of care. Hence, instead of using a single modality to determine prognosis, a multimodal approach to neurologic prognosis seems most prudent. Waiting sufficient time (e.g., 72 hours or more in most cases) after cardiac arrest before making a definite prognosis, and paying particular importance to repeated neurologic examination without sedation, are the most important components of this multimodal approach. A multidisciplinary approach involving an intensivist, neurologist, and most importantly the patient's family members is the key to making decisions about goals of care based on neurologic prognosis and the minimally acceptable quality of life for the patient.

Key Points

- The quality of CPR (especially in the quality of and minimization of interruptions in chest compressions) is probably the single most important treatment-related determinant of outcome from cardiac arrest.
- Cellular damage from ischemia/reperfusion injury is a dynamic process, and a therapeutic window exists after resuscitation from cardiac arrest in which the effects can be attenuated.
- TTM is the first proven therapy to improve neurologic outcome after resuscitation from cardiac arrest, indicating that brain injury related to cardiac arrest is in fact a treatable condition.
- Neurologic prognostication can be very challenging in the early period after resuscitation from cardiac arrest, and in most cases attempts at prognostication should be withheld until at least the 72-hour mark from ROSC.

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Review Questions

1. The recommended chest compression rate during cardiopulmonary resuscitation is (compressions/min):
 - a. >100
 - b. >120
 - c. 100–120
 - d. 100–140

Answer: c. 100–120 compressions/min is the current recommended chest compression rate.^{7–9}

2. During cardiopulmonary resuscitation, once a definitive airway is placed (i.e., endotracheal tube) the recommended ventilation strategy is:
 - a. 1 breath every 3 seconds
 - b. 1 breath every 6 seconds
 - c. 2 breaths for every 30 chest compressions
 - d. 2 breaths for every 15 chest compressions

Answer: b. Once a definitive airway is placed the recommended ventilation strategy is 1 breath every 6 seconds.^{7–9}

3. The preferred antiarrhythmic agent for refractory ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest is:
 - a. Amiodarone
 - b. Lidocaine
 - c. Procainamide
 - d. β -Blocker

Answer: a. Amiodarone is the current recommended antiarrhythmic agent for refractory ventricular fibrillation/pulseless ventricular tachycardia.^{8,26,28}

4. During cardiopulmonary resuscitation, which of the following end-tidal carbon dioxide (ETCO₂) levels would be predictive of mortality?
 - a. 25 mm Hg immediately after return of spontaneous circulation
 - b. 25 mm Hg after 20 minutes of high-quality chest compressions
 - c. 9 mm Hg immediately after return of spontaneous circulation
 - d. 9 mm Hg after 20 minutes of high-quality chest compressions

Answer: d. An ETCO₂ less than 10 mm Hg following 20 minutes of high-quality chest compressions can be predictive of mortality. However, this criterion should not be used alone to decide to terminate resuscitation efforts.^{36,37}

5. After successful resuscitation from cardiac arrest the recommended temperature range and duration for targeted temperature management is:
 - a. 32°C–36°C for 12–24 hours
 - b. 34°C–36°C for 12–24 hours
 - c. 32°C–36°C for at least 24 hours
 - d. 34°C–36°C for at least 24 hours

Answer: c. The recommended temperature range and duration for targeted temperature management is 32°C–36°C for at least 24 hours.^{45,54,88}

6. After successful resuscitation from cardiac arrest a definite indication for emergent coronary angiography is:
 - a. Electrocardiogram (ECG) consistent with ST-segment elevation myocardial infarction (STEMI)
 - b. Ventricular fibrillation/pulseless ventricular tachycardia as the presenting cardiac arrest rhythm
 - c. Out-of-hospital cardiac arrest
 - d. Pulseless electrical activity as the presenting cardiac arrest rhythm

Answer: a. Only ECG findings consistent with STEMI are a definite indication for emergent coronary angiography. There is currently insufficient evidence to recommend routine coronary angiography based on initial cardiac arrest rhythm or location of cardiac arrest. For cardiac arrest patients without STEMI the decision to proceed with emergent coronary angiography should be made in conjunction with an interventional cardiologist.⁵⁴

7. The following is true regarding SSEPs when used for neuroprognostication after cardiac arrest:
 - a. The absence of bilateral cortical responses (N20s) has not been shown to be predictive of poor outcome, but the presence of cortical response predicts good outcome.
 - b. The absence of bilateral cortical responses (N20s) has been shown to be predictive of poor outcome, but the presence of cortical response does not predict good outcome.
 - c. The absence of bilateral cortical responses (N20s) has been shown to be predictive of poor outcome, and the presence of cortical response is predictive of good outcome.
 - d. Neither the absence nor the presence of bilateral cortical responses (N20s) has been shown to be predictive of outcome

Answer: b. The absence of bilateral cortical responses (N20s) has been shown to be highly predictive of poor outcome (false-positive rate of 0% in multiple studies), but the presence of cortical response does not predict good outcome.^{72,83}

2

Airway Management in the Critically Ill Adult

T.R. CRAIG AND G.G. LAVERY

CHAPTER OUTLINE

Structure and Function of the Normal Airway

- The Nose
- The Oral Cavity and Pharynx
- The Larynx
- The Tracheobronchial Tree
- Overview of Airway Function

Assessing Adequacy of the Airway

- Patency
- Protective Reflexes
- Inspired Oxygen Concentration
- Respiratory Drive

Management of the Airway

- Providing an Adequate Inspired Oxygen Concentration
- Establishing a Patent and Secure Airway
- Providing Ventilatory Support

Physiologic Sequelae and Complications of Tracheal Intubation

The Difficult Airway

- Recognizing the Potentially Difficult Airway
- The Airway Practitioner and the Clinical Setting
- Managing the Difficult Airway

Confirming Tube Position in the Trachea

Surgical Airway

- Cricothyrotomy
- Tracheostomy

Extubation in the Difficult Airway Patient (Decannulation)

Tube Displacement in the Critical Care Unit

- Endotracheal Tube
- Tracheostomy Tube

Human Factors

Common Problems in Airway Management

Appropriate management of the airway is the cornerstone of good resuscitation. It requires judgment (airway assessment), skill (airway maneuvers), and constant reassessment of the patient's condition. Although complex airway procedures have a high profile and are sometimes lifesaving, the timely use

of simple airway maneuvers is often very effective and may avoid the need for further intervention.

Structure and Function of the Normal Airway

Knowledge of the structure and function of the airway is required to manage the various conditions that may affect it. The airway begins at the nose and oral cavity and continues as the pharynx and larynx, which lead to the trachea (beginning at the lower edge of the cricoid cartilage) and finally the bronchial tree. The airway provides a pathway between the atmosphere and the lungs for gas/vapor; facilitates filtering, humidification, and heating of ambient air/gas before it reaches the lower airway; prevents nongaseous material from entering the lower airway; and allows phonation by controlling the flow of air through the larynx and oropharynx.¹⁻⁶

The Nose

Key to the respiratory function of the nose are the lateral walls, which on each side lie medial to the orbit, the ethmoid, and maxillary sinuses. Each lateral wall has three horizontal bony projections covered by highly vascular mucosa—the superior, middle, and inferior nasal conchae that greatly increase the surface area. The (nonolfactory) sensory innervation of the nasal mucosa is supplied by two divisions of the trigeminal nerve.

The Oral Cavity and Pharynx

The teeth form the lateral wall of the oral cavity, while the floor is the tongue—a mass of horizontal, vertical, and transverse muscle bundles attached to the mandible and the hyoid bone. The anterior two-thirds of the tongue has sensory innervation from the lingual nerve with taste sensation from the chordae tympani, whereas the posterior one-third has a sensory supply from the glossopharyngeal nerve. All intrinsic and extrinsic muscles of the tongue are supplied by the hypoglossal nerve, except the palatoglossus, which is supplied by the vagus nerve.

The adult pharynx is a midline structure, running anterior to the cervical prevertebral fascia, from the base of the skull to the level of the sixth cervical vertebra (approximately 14 cm), and continuing as the esophagus. It is a muscular tube with three portions: the nasopharynx, oropharynx, and laryngopharynx (or

hypopharynx). It contains three groups of lymphoid tissue: the adenoids, the pharyngeal tonsil (on the posterior wall), and the palatine (lingual) tonsils as well as the inner opening of the eustachian tube on each lateral wall. The vagus nerve supplies all but one of the pharyngeal muscles. Sensory supply is via branches of the glossopharyngeal and vagus nerves. The pharynx is a common pathway for the upper alimentary and respiratory tracts and is concerned with swallowing and phonation.

The Larynx

The larynx sits anteroinferior to the laryngopharynx, anterior to the fourth to the sixth cervical vertebrae, and posterior to the infrahyoid muscles, the deep cervical fascia, and the subcutaneous fat and skin that cover the front of the neck. Laterally lie the lobes of the thyroid gland and carotid sheath. The larynx acts as a sphincter at the upper end of the respiratory tract and is the organ of phonation. The skeleton of the larynx consists of the epiglottis, thyroid, cricoid, and paired arytenoid, cuneiform, and corniculate cartilages, together with the interconnecting ligaments, and has a volume of 4 mL. Two pairs of parallel horizontal folds project into the lumen of the larynx—the false vocal cords (lying superiorly) and the true vocal cords (inferiorly). The opening between the true cords is called the glottis. The larynx communicates above with the laryngopharynx and below with the trachea, which begins at the lower edge of the cricoid ring.

The superior aspect of the epiglottis is innervated by the glossopharyngeal nerve, whereas the inferior surface of the epiglottis and the larynx is innervated by the vagus, via its superior laryngeal nerve (SLN) and recurrent laryngeal nerve (RLN) branches. The external (motor) branch of the SLN supplies the cricothyroid muscle, and the internal branch is the sensory supply to the larynx down to the vocal cords. The RLN supplies all intrinsic laryngeal muscles and is the sensory supply to the larynx below the cords.

In health, the laryngeal abductor muscles contract early in inspiration, separating the vocal cords and facilitating airflow into the tracheobronchial tree. Movements of the thyroid and arytenoid cartilages alter the length and tension of the vocal cords, and sliding and rotational movements of the arytenoid cartilages can alter the shape of the glottic opening between the vocal cords. Fine control of the muscles producing these movements allows vocalization as air passes between the vocal cords in expiration. The sound volume is increased by resonance in the sinuses of the face and skull.

Injury to the SLN causes hoarseness secondary to a loss of tension in the ipsilateral vocal cord. Complete unilateral RLN palsy (loss of function) inactivates both ipsilateral adductor and abductor muscles, but the vocal cord remains adducted (at/near the midline) owing to action of the unopposed SLN-innervated cricothyroid muscle. In bilateral RLN palsy, both cords are in adduction. On inspiration, the adducted vocal cords then act like a Venturi device, generating a negative pressure that pulls the cords even more tightly together, producing inspiratory stridor—the characteristic sign of upper airway obstruction. Sudden complete adduction of the vocal cords caused by muscle spasm (laryngospasm) is a life-threatening form of airway obstruction that may be triggered by mechanical stimulation of the larynx or by cord irritation caused by aspiration of oral secretions, blood, or vomitus.

The Tracheobronchial Tree

The trachea is a midline fibrous tube, 2 cm in diameter, beginning at the level of the sixth cervical vertebra and extending 10 to 14 cm

to the level of the fourth thoracic vertebra before dividing into the right and left main bronchi (carina). The tracheal walls include 15 to 20 incomplete cartilaginous rings limited posteriorly by fibroelastic tissue and smooth muscle. The bronchial tree has a similar wall structure to the trachea.

In the neck the trachea lies anterior to the esophagus, with the RLN in the groove between the two. Anteriorly lie the cervical fascia, infrahyoid muscles, isthmus of the thyroid, and the jugular venous arch. Laterally lie the lobes of the thyroid gland and the carotid sheath. In the thorax, the trachea is traversed anteriorly by the brachiocephalic artery and vein (which may be damaged or eroded by the tracheostomy tube). To the left are the common carotid and subclavian arteries and the aortic arch. To the right are the vagus nerve, the azygos vein, and the pleura. The bifurcation of the trachea (carina) lies anterior to the esophagus and behind the pulmonary trunk.

The two main bronchi diverge from the carina, the right main bronchus being shorter, wider, and more vertical and running close to the pulmonary artery and the azygos vein. The left main bronchus passes under the arch of the aorta, anterior to the esophagus, thoracic duct, and descending aorta.⁷

Overview of Airway Function

In the nose, inspired gas is filtered, humidified, and warmed before entering the lungs. Resistance to gas flow through the nose is twice that of the mouth, explaining the need to mouth-breathe during exercise when the required gas flows are high. Warming and humidification continue in the pharynx and tracheobronchial tree. Between the trachea and the alveolar sacs, the airways divide 23 times, thus increasing the cross-sectional area for the gas exchange.

Particles in inspired air with diameters greater than 10 μm are trapped by hairs on the nasal mucosa. Particles less than 2 μm in diameter may reach the alveoli, where they are ingested by macrophages. Particles 2 to 10 μm in diameter fall on the mucus-covered bronchial walls (as airflow slows), initiating reflex bronchoconstriction and coughing. Such particles are moved upward away from the lungs at a rate of 16 mm/min by cilia that beat at a frequency of 1000 to 1500 cycles/min. Ciliated columnar epithelium lines the respiratory tract from the nose to the respiratory bronchioles (except at the vocal cords). Smoking and some inherited disorders (e.g., Kartagener syndrome) cause defective ciliary motility and a failure of this “mucus escalator.” This may allow more particles to reach the alveoli, thereby predisposing the patient to chronic pulmonary inflammation.⁸

The larynx prevents food and other foreign bodies from entering the trachea. Reflex closure of the glottic inlet occurs during swallowing and periods of increased intrathoracic (e.g., coughing, sneezing) or intraabdominal (e.g., vomiting, micturition) pressure. In unconscious patients, these reflexes are lost, so glottic closure may not occur, increasing the risk of pulmonary aspiration.

Assessing Adequacy of the Airway

Adequacy of the airway should be considered in four aspects:

- *Patency.* Partial or complete obstruction compromises ventilation of the lungs and likewise gas exchange.
- *Protective reflexes.* These reflexes help maintain patency and prevent aspiration of material into the lower airways.
- *Inspired oxygen concentration.* Gas entering the pulmonary alveoli must have an appropriate oxygen concentration.

- *Respiratory drive.* A patent, secure airway is of little benefit without the movement of gas between the atmosphere and the pulmonary alveoli. This is achieved by the processes of inspiration and expiration.

Patency

Airway obstruction is most frequently due to reduced muscle tone, allowing the tongue to fall backward against the postpharyngeal wall, thereby blocking the airway. Loss of patency by this mechanism typically occurs in an obtunded or anesthetized patient lying supine. Other causes include the presence of blood, mucus, vomitus, or a foreign body in the lumen of the airway or edema, inflammation, swelling, or enlargement of the tissues lining or adjacent to the airway.

Upper airway obstruction has a characteristic presentation in the spontaneously breathing patient: noisy inspiration (stridor), poor expired airflow, intercostal retraction, increased respiratory distress, and paradoxical rocking movements of the thorax and abdomen.⁹ These resolve quickly if the obstruction is removed. In total airway obstruction, sounds of breathing are absent entirely, owing to complete lack of airflow through the larynx. Airway obstruction may occur in patients with an endotracheal tube (ET) or tracheostomy tube in situ as the result of mucous plugging or kinking of the tube or the patient's biting down on a tube placed orally. Spontaneously breathing patients with airway obstruction have the symptoms and signs described earlier, whereas patients supported by assisted (positive-pressure) breathing modes exhibit high inflation pressures, decreased tidal and minute volumes, increased end-tidal carbon dioxide (ETCO₂) levels, and decreased arterial oxygen saturation.

Protective Reflexes

The upper airway shares a common pathway with the upper gastrointestinal tract. Protective reflexes, which exist to safeguard airway patency and to prevent foreign material from entering the lower respiratory tract, involve the epiglottis, the vocal cords, and the sensory supply to the pharynx and larynx.¹⁰ Patients who can swallow normally have intact airway reflexes, and normal speech makes absence of such reflexes unlikely. Patients with a decreased level of consciousness (LOC) should be assumed to have inadequate protective reflexes.

Inspired Oxygen Concentration

Oxygen demand is elevated by the increased work of breathing associated with respiratory distress¹¹ and by the increased metabolic demands in critically ill or injured patients. Often, higher inspired oxygen concentrations are required to satisfy tissue oxygen demand and to prevent critical desaturations during maneuvers for managing the airway. A cuffed ET, connected to a supply of oxygen, is a sealed system in which the delivered oxygen concentration also is the inspired concentration. A patient wearing a face mask, however, inspires gas from the mask and surrounding ambient air. Because the patient will generate an initial inspiratory flow in the region of 30 to 60 L/min, and the fresh gas flow to a mask is on the order of 5 to 15 L/min, much of the tidal inspiration will be "ambient air" entrained from around the mask. The entrained air can dilute the concentration of oxygen inspired to less than 50%, even when 100% oxygen is delivered to the mask. This unwelcome reduction in inspired oxygen concentration can be mitigated by

- (1) using a mask with a reservoir bag, (2) ensuring that the mask is fitted firmly to the patient's face, (3) using a high rate of oxygen flow to the mask (15 L/min), and (4) supplying a higher oxygen concentration if not already using 100%.

Respiratory Drive

A patent, protected airway will not produce adequate oxygenation or excretion of carbon dioxide without adequate respiratory drive. Changing arterial carbon dioxide tension (PCO₂) by changing the hydrogen ion (H⁺) concentration in cerebrospinal fluid stimulates the respiratory center, which in turn controls minute volume and therefore arterial PCO₂ (negative feedback). This assumes that increased respiratory drive can produce an increase in minute ventilation (increased respiratory rate or tidal volume, or both), which may not occur if respiratory mechanics are disturbed. Brain injury and drugs such as opioids, sedatives, and alcohol are direct-acting respiratory center depressants.

Ventilation can be assessed qualitatively by looking, listening, and feeling. In a spontaneously breathing patient, listening to (and feeling) air movement while looking at the extent, nature, and frequency of thoracic movement provides an impression of ventilation. These parameters may be misleading, however. Objective assessment of minute ventilation requires PCO₂ measurement in arterial blood or monitoring of ETCO₂, which can be used as a real-time measure of the adequacy of minute ventilation. If respiratory drive or minute ventilation is inadequate, positive-pressure respiratory support may be required, and any underlying factors should be addressed if possible (e.g., depressant effect of sedatives or analgesics).

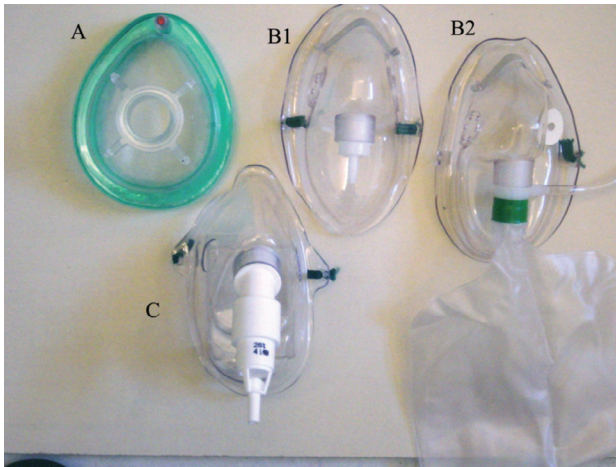
Management of the Airway

The aims of airway management are to provide an adequate inspired oxygen concentration; to establish a patent, secure airway; and to support ventilation if required.

Providing an Adequate Inspired Oxygen Concentration

Although oxygen can be administered via nasal cannula, this method does not ensure delivery of more than 30% to 40% oxygen (at most). Other disadvantages include lack of humidification of gases, patient discomfort with use of flow rates greater than 4 to 6 L/min, and predisposition to nasal mucosal irritation and potential bleeding. Therefore despite being more intrusive for patients, face masks are superior for oxygen administration. The three main types of face masks are shown in Fig. 2.1:

- The anesthesia-type face mask (mask A in Fig. 2.1) is a solid mask (with no vents) with a cushioned collar to provide a good seal. It is suitable for providing very high oxygen concentrations (approaching 100%) because entrainment is minimized and the anesthetic circuit normally includes a reservoir of gas. These masks become unacceptable for many *awake* patients within a few minutes because of the association with heat, moisture, and claustrophobia.
- The simple face mask has vents that allow dissipation of heat or humidity but that also entrain room air. These masks have no seal and are relatively loose-fitting. Such masks may have a reservoir bag (approximately 500 mL) sitting inferior to the mask (B2 in Fig. 2.1) or may not (B1 in Fig. 2.1). Using the latter, it is difficult to reliably deliver an inspired oxygen



• **Fig. 2.1** Face masks: anesthesia mask (A); simple face mask (B1); simple face mask with reservoir bag (B2); Venturi mask (C).

concentration in excess of 50% even with tight application and 100% oxygen flow to the mask. Under the same conditions, a simple mask with a reservoir bag can produce an inspired oxygen concentration of about 80%.

- The Venturi mask (C in Fig. 2.1) has vents that entrain a known proportion of ambient air when a set flow of 100% oxygen passes through a Venturi device. Thus the inspired oxygen concentration (usually 24% to 35%) is known.

Establishing a Patent and Secure Airway

Establishing a patent and secure airway can be achieved using simple airway maneuvers, further airway adjuncts, tracheal intubation, or a surgical airway.

Airway Maneuvers

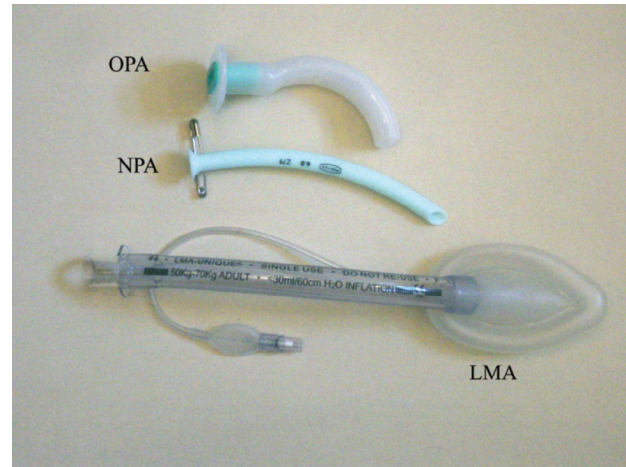
Simple airway maneuvers involve appropriate positioning, opening the airway, and keeping it open using artificial airways if needed.

Positioning for Airway Management

In the absence of any concerns about cervical spine stability (e.g., with trauma, rheumatoid arthritis, or severe osteoporosis), raising the patient's head slightly (5–10 cm) by means of a small pillow under the occiput can help in airway management. This adjustment extends the atlantooccipital joint and moves the oral, pharyngeal, and laryngeal axes into better alignment, providing the best straight line to the glottis (“sniffing” position).

Clearing the Airway

Acute airway obstruction in the obtunded patient is often due to the tongue or extraneous material—liquid (saliva, blood, gastric contents) or solid (teeth, broken dentures, food)—in the pharynx. In the supine position, secretions can be cleared under direct vision using a laryngoscope and a rigid suction catheter.¹² In some cases, a flexible suction catheter, introduced through the nose and nasopharynx, may be the best means of clearing the airway. A finger sweep of the pharynx may be used to detect and remove larger solid material in unconscious patients without an intact gag reflex. During all airway interventions, if cervical spine instability cannot be ruled out, relative movement of the cervical vertebrae must be prevented—most often by manual inline immobilization.^{12,13}



• **Fig. 2.2** Artificial airways: oropharyngeal airway (OPA); nasopharyngeal airway (NPA); laryngeal mask airway (LMA).

Triple Airway Maneuver

The triple airway maneuver often is beneficial in obtunded patients if it is not contraindicated by concerns about cervical spine instability. As indicated by its name, this maneuver has three components: head tilt (neck extension), jaw thrust (pulling the mandible forward), and mouth opening.¹⁴ The operator stands behind and above the patient's head. Then the maneuver is performed as follows:

- Extend the patient's neck with the operator's hands on both sides of the mandible.
- Elevate the mandible with the fingers of both hands, thereby lifting the base of the tongue away from the posterior pharyngeal wall.
- Open the mouth by pressing caudally on the anterior mandible with the thumbs or forefingers.

Artificial Airways

If the triple airway maneuver or any of its elements reduces airway obstruction, the benefit can be maintained for a prolonged period by introducing an artificial airway into the pharynx between the tongue and the posterior pharyngeal wall (Fig. 2.2).

The oropharyngeal airway (OPA) is the most commonly used artificial airway. Simple to insert, it is used temporarily to help facilitate oxygenation or ventilation before tracheal intubation. The OPA should be inserted with the convex side toward the tongue and then rotated through 180 degrees. Care must be taken to avoid pushing the tongue posteriorly, thereby worsening the obstruction. The nasopharyngeal airway has the same indications as for the OPA but significantly more contraindications¹⁵ (Box 2.1). It is better tolerated than the OPA, making it useful in semiconscious patients in whom the gag reflex is partially preserved. These artificial airways should be considered as a temporary adjunct—to be replaced with a more secure airway if the patient's condition fails to improve rapidly to the point at which an artificial airway no longer is needed. Such airways should not be used in association with *prolonged* positive-pressure ventilation.

Advanced Airway Adjuncts

Advanced airway adjuncts fill the gap between simple airway maneuvers and the insertion of a tracheal tube or surgical airway. These devices can be used to facilitate safe reliable airway management and manual ventilation in the prehospital or emergency

• BOX 2.1 Contraindications to Insertion of Oropharyngeal and Nasopharyngeal Airways

Contraindications to Oropharyngeal Airways

Inability to tolerate (gagging, vomiting)
 Airway swelling (burns, toxic gases, infection)
 Bleeding into the upper airway
 Absence of pharyngeal or laryngeal reflexes
 Impaired mouth opening (e.g., with trismus or temporomandibular joint dysfunction)

Contraindications to Nasopharyngeal Airways

Narrow nasal airway in young children
 Blocked or narrow nasal passages in adults
 Airway swelling (burns, toxic gases, infection)
 Bleeding into the upper airway
 Absence of pharyngeal or laryngeal reflexes
 Fractures of the midface or base of skull
 Clinical scenarios in which nasal hemorrhage would be disastrous

resuscitation setting, often without expert medical presence. The number of supraglottic airways (SGAs) available has increased dramatically in the past 2 decades. They may be used to maintain airway maintenance during low-risk surgery and have utility in airway management in obese patients, in airway rescue after failed intubation, and during an anticipated difficult airway.¹⁶

The laryngeal mask airway (LMA) is a small latex mask mounted on a hollow plastic tube.^{17,18} The airway is placed “blindly” in the lower pharynx overlying the glottis. The inflatable cuff helps wedge the mask in the hypopharynx, sitting obliquely over the laryngeal inlet. Although the LMA produces a seal that allows ventilation with gentle positive pressure, it does not definitively protect the airway from aspiration. Indications for use of the LMA in critical care are (1) as an alternative to other artificial airways, (2) the difficult airway, particularly the “cannot intubate–cannot ventilate” scenario, and (3) as a conduit for bronchoscopy. It is possible to pass a 6.0-mm ET through a standard LMA into the trachea, but the LMA must be left in situ. The intubating LMA, which was developed specifically to aid intubation with a tracheal tube, has a shorter steel tube with a wider bore, a tighter curve, and a distal silicone laryngeal cuff. A bar present near the laryngeal opening is designed to lift the epiglottis anteriorly. The intubating LMA allows the passage of a specially designed size 8.0 ET.

There is increasing evidence that many of the newer SGAs have better performance characteristics that may improve efficacy compared with the original LMA device. They are often easier to insert, with better oropharyngeal seal pressures, and may reduce the risk of aspiration.^{19,20}

Tracheal Intubation

If the foregoing interventions are not effective or are contraindicated, tracheal intubation is required. This modality provides (1) a secure, potentially long-term airway; (2) a safe route to deliver positive-pressure ventilation if required; and (3) significant protection against pulmonary aspiration.

Orotracheal intubation is the most widely used technique for clinicians practiced in direct laryngoscopy (indications and contraindications in [Box 2.2](#)). Normally, anesthesia with or without neuromuscular blockade is necessary for this procedure, which is summarized in [Box 2.3](#).

• BOX 2.2 Orotracheal Intubation: Indications and Relative Contraindications

Indications

Long-term correction or prevention of airway obstruction
 Securing the airway and protecting against pulmonary aspiration
 Facilitating positive-pressure ventilation
 Enabling bronchopulmonary toilet
 Optimizing access to pharynx, face, or neck at surgery

Contraindications (Relative)

Possibility of cervical spine instability
 Impaired mouth opening (e.g., trismus, temporomandibular joint dysfunction)
 Potential difficult airway
 Need for surgical immobilization of maxilla or mandible (wires, box frame)

• BOX 2.3 Procedure: Orotracheal Intubation

- Position patient and induce anesthesia ± neuromuscular blockade (if needed).
- Perform manual ventilation using triple airway maneuver and oropharyngeal airway.
- Hold laryngoscope handle (left hand) near the junction with blade.
- Insert the blade along the right side of the tongue—moving tongue to the left.
- Advance tip of the blade in the midline between tongue and epiglottis.
- Pull upward and along the line of the handle of the laryngoscope.
- Lift the epiglottis upward and visualize the vocal cords.
- Do *not* use the patient's teeth as a fulcrum when attempting to visualize the glottis.
- Stop advancing tube when cuff is 2 to 3 cm beyond the cords.
- Connect to a bag-valve system and pressurize it by squeezing bag.
- Inflate cuff until audible leak around tube stops.
- Check correct tube position (auscultation) and assess cuff pressure.
- Check end-tidal carbon dioxide trace.

TABLE 2.1 Drugs Used to Facilitate Tracheal Intubation

Drug	Dose (Intravenous)
Induction Agent	
Propofol	1–2.5 mg/kg
Opioids	
Fentanyl	1.0–1.5 µg/kg
Morphine	0.15 mg/kg
Nondepolarizing Agents	
Atracurium	0.4–0.5 mg/kg
Vecuronium	0.1 mg/kg
Rocuronium	0.45–0.6 mg/kg
Depolarizing Agent	
Succinylcholine (suxamethonium)	1.0–1.5 mg/kg

Tracheal intubation requires lack of patient awareness (as in the unconscious state or with general anesthesia) and the abolition of protective laryngeal and pharyngeal reflexes. The drugs commonly used to achieve these states are shown in [Table 2.1](#). Anesthesia is achieved using an intravenous induction agent, although intravenous

sedatives (e.g., midazolam) theoretically may be used. Opioids often are used in conjunction with induction agents because they may reduce the cardiovascular sequelae of laryngoscopy and intubation (tachycardia and hypertension) and may contribute to the patient's unconsciousness.

Abolition of protective laryngeal and pharyngeal reflexes may be achieved by inducing a deep level of unconsciousness using one or more of the aforementioned agents, followed by inhalation of high concentrations of a volatile anesthetic agent (e.g., sevoflurane, isoflurane). This technique sometimes is used in the anticipated difficult airway scenario to obtain conditions suitable for tracheal intubation in a patient who is still breathing spontaneously.

More often, a muscle relaxant is used to abolish the protective reflexes, abduct the vocal cords, and facilitate tracheal intubation. In the elective situation, nondepolarizing neuromuscular blocking agents are used. These drugs have the disadvantage of requiring several minutes to exert their effect, during which the patient must receive ventilation via a mask, thus allowing the possibility of gastric dilation and pulmonary aspiration. In patients at high risk of the latter (e.g., nonfasting patients), a depolarizing muscle relaxant (succinylcholine) can be used because it produces suitable conditions for intubation within 15 to 20 seconds, and mask ventilation is not required. Succinylcholine has several side effects—among them hyperkalemia, muscle pains, and (rarely) malignant hyperpyrexia. Alternatively, the ability to antagonize the effects of rocuronium rapidly with sugammadex may be an advantage. If rapid antagonism of rocuronium with sugammadex is part of the failed intubation plan, then the appropriate dose (16 mg/kg) must be readily available.

Nasotracheal intubation shares the problems and contraindications associated with the NPA. The technique usually is used when there are relative contraindications to the oral route (e.g., anatomic abnormalities, cervical spine instability). Nasotracheal intubation may be achieved under direct vision or (if spontaneously breathing) with the use of a blind technique, either with the patient under general anesthesia or in the awake or lightly sedated patient with appropriate local anesthesia (Box 2.4). If orotracheal or nasotracheal intubation is required but cannot be achieved, then a surgical airway is required (see later text).

With a need for isolation of one lung from another, a double-lumen tube (having one cuffed tracheal lumen and one cuffed bronchial lumen fused longitudinally) can be used.²¹ The main indications are (1) to facilitate some pulmonary or thoracic surgical procedures; (2) to isolate a lung containing contaminated fluid (e.g., in lung abscess) or blood, thereby preventing contralateral spread; and (3) to enable differential or independent lung ventilation. Intermittent lung ventilation allows each lung to be treated separately—for example, to deliver positive-pressure ventilation with high positive end-expiratory pressure to one lung while applying low levels to the other. Such a strategy may be advantageous in cases of pulmonary air leak (bronchopleural fistula, bronchial tear, or severe lung trauma) or in severe unilateral lung disease requiring ventilatory support.^{21,22}

Providing Ventilatory Support

If a patient has no (or inadequate) spontaneous ventilation, then a means of generating gas flow to the lower respiratory tract must be provided. Negative pressure, mimicking the actions of the respiratory muscles, occasionally is used in some patients who require long-term ventilation. In acute care, however, ventilation is achieved using positive pressure, which requires an unobstructed airway; in the nonintubated patient, this is best achieved by proper

• BOX 2.4 Procedure: Nasotracheal Intubation (Blind and Under Direct Vision)

Preparation and Assessment of the Patient

1. Use a nasal decongestant such as phenylephrine to reduce bleeding.
2. Provide local anesthesia to the nasal mucosa.
3. Examine each nostril for patency and deformity.
4. Choose the most patent nostril, and use an appropriate-size ET.
5. After induction of anesthesia, position the head and neck as for oral intubation.

Blind Nasotracheal Intubation

- Keep patient breathing spontaneously.
- Insert well-lubricated ET into the nostril (concavity forward, bevel lateral).
- While passing ET along nasal floor, listen for audible breathing through the tube.
- Advance ET, rotating as needed to maintain clear breath sounds.
- ET will pass through cords, and patient may cough.
- Technique takes time, so it is not suitable for a patient experiencing desaturation.
- Do not force passage of ET, because this could cause bleeding.

Nasotracheal Intubation (Direct Vision)

- Patient may be apneic with or without relaxants.
- Gently advance ET through the nose.
- When ET tip is in oropharynx, perform laryngoscopy.
- Visualize ET in pharynx and advance toward glottis.
- Advance ET through cords into trachea, under direct vision if possible.
- Use Magill forceps if required to guide tip while advancing ET.
- Try to avoid damaging cuff if using forceps to help passage through cords.

ET, Endotracheal tube.

positioning, the triple airway maneuver, and use of an OPA or NPA. In a patient without an ET in place, particularly if some degree of airway obstruction exists, positive-pressure ventilation often causes gastric distention and (potentially) regurgitation and pulmonary aspiration.

Bag-Valve-Mask Ventilation

Ventilation with a mask requires an (almost) airtight fit between mask and face. This is best achieved by firmly pressing the mask against the patient's face using the thumb and index finger (C-grip) while pulling the mandible upward toward the mask with the other three fingers. The other hand is used to squeeze the reservoir bag, generating positive pressure. Excessive pressure from the C-grip on the mask may lead to backward movement of the mandible with subsequent airway obstruction, or a tilt of the mask with leakage of gas. If a proper seal is difficult to attain, placing a hand on each side of the mask and mandible is advised, with a second person manually compressing the reservoir bag (four-handed ventilation). Bag-valve-mask systems have a self-reinflating bag, which springs back after compression, thereby drawing gas in through a port with a one-way valve. It is important to have a large reservoir bag with a continuous flow of oxygen attached to this port to achieve a high inspired oxygen concentration.^{23,24} Bag-valve-mask ventilation usually is a short-term measure in urgent situations or is used in preparation for tracheal intubation.

Prolonged Ventilation Using a Sealed Tube in the Trachea

Ventilation of the lungs with a bag-valve-mask arrangement is difficult and potentially dangerous if required for more than a few

minutes or if the patient needs to be transported. In these instances, ventilation through a sealed tube in the trachea is indicated. Orotracheal or nasotracheal intubation, surgical cricothyrotomy, and tracheostomy all achieve the same result: a cuffed tube in the trachea, allowing the use of positive-pressure ventilation and protecting the lungs from aspiration.

Apneic Oxygenation

Apneic oxygenation is achieved using a narrow catheter that sits in the trachea and carries a flow of 100% oxygen. The catheter may be passed into the trachea via an ET or under direct vision through the larynx. This apparatus can be set up as a low-flow open system (gas flow rate of 5 to 8 L/min) or as a high-pressure (jet ventilation) system²⁵ and can be used to maintain oxygenation with a difficult airway either at intubation or at extubation (see later text).

Physiologic Sequelae and Complications of Tracheal Intubation

Laryngoscopy is a noxious stimulus that, in an awake or lightly sedated patient, would provoke coughing, retching, or vomiting and laryngospasm. In clinical practice, however, laryngoscopy and tracheal intubation usually are performed after induction of anesthesia, and in emergency situations, the patient often is hypoxic and hypercarbic, with increased sympathetic nervous system activity. Thus the physiologic effects of laryngoscopy and tracheal intubation tend to be masked.

Laryngoscopy and intubation cause an increase in circulating catecholamines and increased sympathetic nervous system activity, leading to hypertension and tachycardia. This represents an increase in myocardial work and myocardial oxygen demand, which may provoke cardiac dysrhythmias and myocardial hypoxia or ischemia. Laryngoscopy increases cerebral blood flow and intracranial pressure—particularly in patients who are hypoxic or hypercarbic at the time of intubation.²⁶ This rise in intracranial pressure will be exaggerated if cerebral venous drainage is impeded by violent coughing, bucking, or breath-holding.

Coughing and laryngospasm occur frequently in patients undergoing laryngoscopy and intubation when muscle relaxation and anesthesia are inadequate. Increased bronchial smooth muscle tone, which increases airway resistance, may occur as a reflex response to laryngoscopy or may be due to the physical presence of the ET in the trachea; in its most severe form, termed bronchospasm, this increased tone causes audible wheeze and ventilatory difficulty.

The cross-sectional area of the ET is less than that of the airway and therefore causes increased resistance to gas flow. This difference usually is unimportant with positive-pressure ventilation but causes a significant increase in work of breathing in spontaneously breathing patients. Resistance is directly related to $1/r^4$ (where r is the radius of the ET) and will be minimized by use of a large-bore ET.

Laryngoscopy and intubation may cause bruising, abrasion, laceration, bleeding, or displacement or dislocation of the structures in and near the airway (e.g., lips, teeth or dental prostheses, tongue, epiglottis, vocal cords, laryngeal cartilages). Dislodged structures such as teeth or dentures may be aspirated, blocking the airway more distally. Less common complications include perforation of the airway with the potential for the development of a retropharyngeal abscess or mediastinitis. Over time, erosions resulting from pressure and ischemia may develop on the lips or

tongue (or external nares and anterior nose in patients with a nasotracheal tube) and in the larynx or upper trachea. These lesions result in a breach of the mucosa with the potential for secondary infection. In the case of the lips and tongue, such lesions are (temporarily) disfiguring and painful and may inhibit attempts to talk or swallow.

The mucosa of the upper trachea (subglottic area) is subjected to the pressure of the cuff of the ET. This pressure reduces perfusion of the tracheal mucosa and, combined with the mechanical movement of the tube (from patient head movements, nursing procedures, or rhythmic flexion with action of the ventilator), tends to cause mucosal damage and increase the risk of superficial infection. These processes may lead to ulceration of the tracheal mucosa, fibrous scarring, contraction, and ultimately stenosis, which can be a life-limiting or life-threatening problem.

Any tube in the trachea has a significant effect on the mechanisms protecting the airway from aspiration and infection. The mucus escalator may be inhibited by mucosal injury and by the lack of warm humidified airflow over the respiratory epithelium.²⁷ The disruption of normal swallowing results in the pooling of saliva and other debris in the pharynx and larynx above the upper surface of the tube's inflatable cuff, which may become the source of respiratory infection if the secretions become colonized with microorganisms, or may pass beyond the cuff into the lower airways—that is, pulmonary aspiration (silent or overt).^{28,29} The former may occur as a result of (1) colonization of the gastric secretions and the regurgitation of this material up the esophagus to the pharynx or (2) transmission of microorganisms from the health care environment to the pharynx via medical equipment or the hands of hospital staff or visitors (cross-infection).^{29–32}

The presence of a tube traversing the larynx and sealing the trachea makes phonation impossible. The implications of this limitation for patients and their families often are ignored. If patients cannot tell caregivers about pain, nausea, or other concerns, they may become frustrated, agitated, or violent. This may result in the excessive use of sedative or psychoactive drugs, which prolong the time ventilation is used and the stay in the intensive care unit (ICU), with the risk of infection increased accordingly.³³ The inability to communicate may therefore be a real threat to patient survival. Potential solutions involve the use of letter and picture boards, “speaking valves” (with tracheostomy), laryngeal microphones, or computer-based communication packages. The involvement and innovations of disciplines such as the speech and language center may be advantageous.

The Difficult Airway

The difficult airway has been defined as “the clinical situation in which a conventionally trained anesthetist experiences difficulty with mask ventilation of the upper airway, tracheal intubation, or both.”³⁴ It has been a commonly documented cause of adverse events, including airway injury, hypoxic brain injury, and death under anesthesia.^{35–41} The frequency of difficulty with mask ventilation has been estimated to be between 1.4% and 7.8%,^{42–44} whereas tracheal intubation using direct laryngoscopy is difficult in 1.5% to 8.5% and impossible in up to 0.5% of general anesthetics.^{40,45} The incidence of failed intubation is approximately 1:2000 in the nonobstetric population and 1:300 in the obstetric population.⁴⁶ In the critical care unit, up to 20% of all critical incidents are airway related,^{47–49} and such incidents may occur at intubation, at extubation, or during the course of treatment (as with the acutely displaced or obstructed ET or tracheostomy tube).

Recognizing the Potentially Difficult Airway

Many conditions are associated with airway difficulty (Table 2.2), including anatomic abnormalities, which may be associated with an unusual appearance, thereby alerting the examiner. The goal is to identify the potentially difficult airway and develop a plan to secure it. Factors including age older than 55 years, body mass index greater than 26 kg/m², presence of a beard, lack of teeth, and a history of snoring have been identified as independent variables predicting difficulty with mask ventilation—in turn associated with difficult tracheal intubation.^{43,50}

Mallampati⁵¹ developed a grading system (subsequently modified⁴⁶) that predicted ease of tracheal intubation at direct laryngoscopy.

The predictive value of the Mallampati system has been shown to be limited^{52,53} because many factors that have no influence on the Mallampati classification—mobility of head and neck, mandibular or maxillary development, dentition, compliance of neck structures, and body shape—can influence laryngeal view.^{35,48,54,55} A study of a complex system including some of these factors found the rate of difficult intubation to be 1.5%, but with a false-positive rate of 12%.⁵⁶ A risk index based on the Mallampati classification, a history of difficult intubation, and five other variables lacked sufficient sensitivity and specificity.⁵⁷ Airway management should be based on the fact that the difficult airway cannot be reliably predicted.^{58,59} This is a particularly important consideration in the critical care environment.

TABLE 2.2 Conditions Associated With Difficult Airway

Causative Factors	Associated Conditions/Disorders
Abnormal anatomy/development	Small mouth or large tongue Dental abnormality Prognathia Obesity Advanced pregnancy Acromegaly Syndromic child ^a
Inability to open mouth	Masseter muscle spasm (dental abscess) Temporomandibular joint dysfunction Facial burns Postradiotherapy fibrosis Scleroderma
Cervical immobility/abnormality	Short neck/obesity Poor cervical mobility (e.g., ankylosing spondylitis) Previous cervical spine surgery Presence of cervical collar Postradiotherapy fibrosis
Pharyngeal or laryngeal abnormality	High or anterior larynx Deep vallecula: inability to reach base of epiglottis with blade of scope Anatomic abnormality of epiglottis or hypopharynx (e.g., tumor) Subglottic stenosis
Injury	Traumatic debris Obstructing foreign bodies Basilar skull fracture Bleeding into airway or adjacent swelling/hematoma Fractured maxilla/mandible Cervical spine instability (confirmed or potential) Laryngeal fracture or disruption
Infections	Epiglottitis Abscess Croup, bronchiolitis Laryngeal papillomatosis Tetanus/trismus
Connective tissue/inflammatory disorders	Rheumatoid arthritis: temporomandibular joint or cervical spine involvement, cricoarytenoid arthritis Ankylosing spondylitis Scleroderma Sarcoidosis
Endocrine disorders	Goiter: airway compression or deviation Hypothyroidism, acromegaly: large tongue

^aFor example, Klippel-Feil syndrome (limited neck extension); Beckwith-Weidemann syndrome (restricted TM joint mobility and large tongue); Pierre Robin Syndrome (limited submandibular space)

The Obstructed Airway

Although the most common reason for an obstructed airway in the intubated patient is posterior displacement of the tongue in association with a depressed LOC, it is the less common causes that provide the greatest challenges. It is important to quickly elucidate the level at which the obstruction occurs and, if possible, the nature of the obstructing lesion: infection or edema (epiglottitis, pharyngeal or tonsillar abscess, mediastinal abscess), neoplasm (primary malignant or benign tumor, metastatic spread, direct extension from nearby structures), thyroid enlargement, vascular lesions, trauma, or foreign body or impacted food.

Airway lesions above the level of the vocal cords are considered to lie in the upper airway and commonly manifest with stridor.⁶⁰ If breathing is labored and associated with difficulty breathing at night, rather than just noisy breathing, then the narrowing probably is more than 50%. Patients with these lesions usually fall into one of two groups: (1) those who can be intubated, usually under inhalational induction, with an otorhinolaryngologist or other qualified surgeon/physician immediately available to perform rigid bronchoscopy or tracheostomy if required, or (2) those who require a tracheostomy performed while under local anesthesia. In patients with midtracheal obstruction, computed tomography imaging usually is necessary to discover the exact level and nature of the obstruction and to allow planning of airway management for nonemergency clinical presentations.⁶⁰ Tracheostomy often is not beneficial because the tube may not be long enough to bypass the obstruction. In such instances, fiberoptic intubation often may be useful. Lower tracheal obstruction often is due to space-occupying lesions in the mediastinum and necessitates multidisciplinary planning involving otorhinolaryngology, cardiothoracic surgery, anesthesia, and critical care team members.

Trauma and the Airway

Airway management in the trauma victim provides additional challenges because the victim often has other life-threatening conditions and preparation time for management of the difficult airway is limited. Approximately 15% of severely injured patients have maxillofacial involvement, and 5% to 10% of patients with blunt trauma have an associated cervical spine injury (often associated with head injury).⁶¹

Problems encountered in trauma patients include presence in the airway of debris or foreign bodies (e.g., teeth), vomitus, or regurgitated gastric contents; airway edema; tongue swelling; blood and bleeding; and fractures (maxilla and mandible). Patients must be assumed to have a full stomach (requiring bimanual cricoid pressure and a rapid-sequence induction for intubation), and many will have pulmonary aspiration before the airway is secured. An important consideration in most cases is the need to avoid movement of the cervical spine at laryngoscopy or intubation.^{12,13} Direct injury to the larynx is rare but may result in laryngeal disruption, producing progressive hoarseness and subcutaneous emphysema. Tracheal intubation, if attempted, requires great care and skill because it may cause further laryngeal disruption. With Le Fort fractures, airway obstruction or compromised respiration requiring immediate airway control is present in 25% of cases.⁶² Postoperative bleeding after operations to the neck (thyroid gland, carotid, larynx) may compress or displace the airway, leading to difficulty in intubation.

The Airway Practitioner and the Clinical Setting

Although airway difficulties often are due to anatomic factors as discussed, it is important to recognize that the inability to perform

• BOX 2.5 Common Errors Compromising Successful Intubation

Poor patient positioning
 Failure to ensure appropriate assistance
 Faulty light source in laryngoscope or no alternative scope
 Failure to use a longer blade in appropriate patients
 Use of inappropriate tracheal tube (size or shape)
 Lack of immediate availability of airway adjuncts

an airway maneuver also may be due to a practitioner's inexperience or lack of skill.^{63–68} Expert opinion and clinical evidence also identify lack of skilled assistance as a factor in airway-related adverse events.^{69–72} As might be expected, inexperience and lack of suitable help may contribute to failure in optimizing the conditions for laryngoscopy (Box 2.5). Airway and ventilatory management performed in the prehospital setting or in the hospital but outside an operating room (OR) carries a higher frequency of adverse events and a higher mortality rate compared with those performed using anesthesia in an OR.^{73–77} In the critical care unit, all invasive airway maneuvers are potentially difficult.⁷⁸ Positioning is more difficult on an ICU bed than on an OR table. The airway structures may be edematous after previous laryngoscopy or presence of an ET. Neck immobility or the need to avoid movement in a potentially unstable cervical spine may be other contributing factors.^{79–81} Poor gas exchange in ICU patients reduces the effectiveness of preoxygenation and increases the risk of significant hypoxia before the airway is secured.⁸² Cardiovascular instability may produce hypotension or hypoperfusion, or it may lead to misleading oximetry readings (including failure to record any value at all), a further confounding factor for the attending staff.^{83,84}

Managing the Difficult Airway

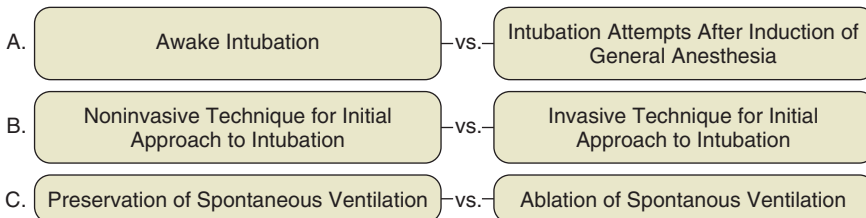
Management of the difficult airway can be considered in the framework of three possible clinical scenarios with progressively increasing risks for the patient: (1) the anticipated difficult airway; (2) the unanticipated difficult airway; and (3) the difficult airway resulting in a can't intubate–can't ventilate situation.

Requirements for clinicians involved in airway management include the following:

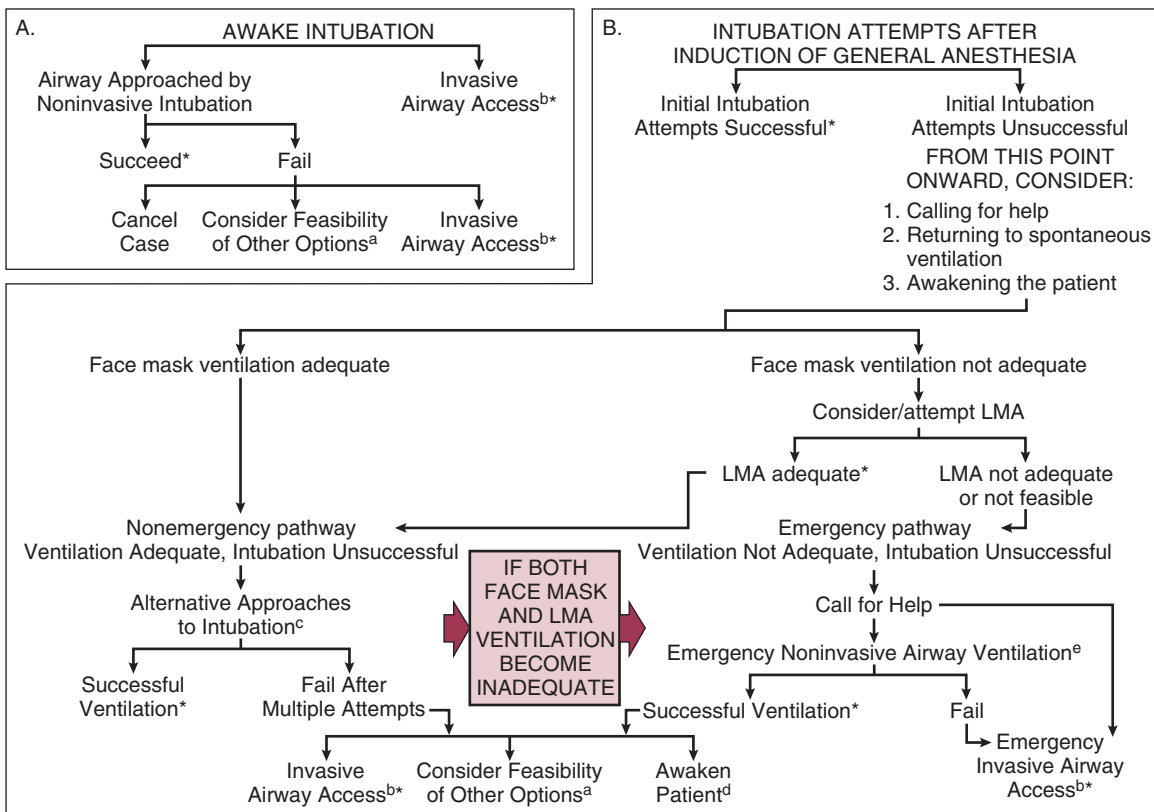
- Expertise in recognition and assessment of the potentially difficult airway. This involves the use of the assessment techniques noted previously and a “sixth sense.”⁵⁸
- The ability to formulate a plan (with alternatives).³⁴
- Familiarity with algorithm(s) that outline a sequence of actions designed to maintain oxygenation, ventilation, and patient safety. The American Society of Anesthesiologists guidelines³⁴ and the composite plan from the Difficult Airway Society (DAS)⁸⁵ are shown in Figs. 2.3 through 2.5. Fig. 2.4 summarizes four airway plans (A–D), available from the DAS website (www.das.uk.com).
- The skills and experience to use a number of airway adjuncts, particularly those relevant to the unanticipated difficult airway. Basic preparation for management of a difficult airway includes (1) the availability of the correct equipment to hand the clinician, (2) informing the patient with a known or suspected difficult airway, (3) assigning an appropriately skilled individual to provide assistance during the procedure, (4) preanesthetic preoxygenation

DIFFICULT AIRWAY ALGORITHM

1. Assess the likelihood and clinical impact of basic management problems:
 - Difficult ventilation
 - Difficult intubation
 - Difficulty with patient cooperation or consent
 - Difficult tracheostomy
2. Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management.
3. Consider the relative merits and feasibility of basic management choices:



4. Develop primary and alternative strategies:



* Confirm ventilation, tracheal intubation, or LMA placement with exhaled CO₂.

^a Other options include (but are not limited to) surgery utilizing face mask or LMA anesthesia, local anesthesia infiltration, and regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore these options may be of limited value if this step in the algorithm has been reached via the Emergency Pathway.

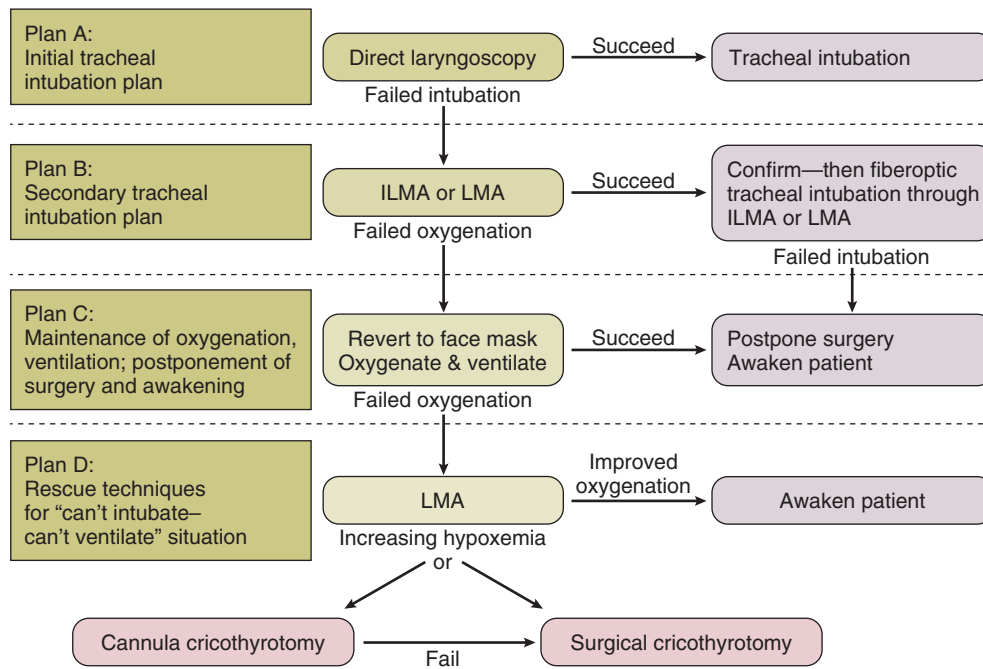
^b Invasive airway access includes surgical or percutaneous tracheostomy and cricothyrotomy.

^c Alternative noninvasive approaches to difficult intubation include (but are not limited to) use of different laryngoscope blades, LMA as an intubation conduit (with or without fiberoptic guidance), fiberoptic intubation, intubating stylet or tube changer, light wand, retrograde intubation, and blind oral or nasal intubation.

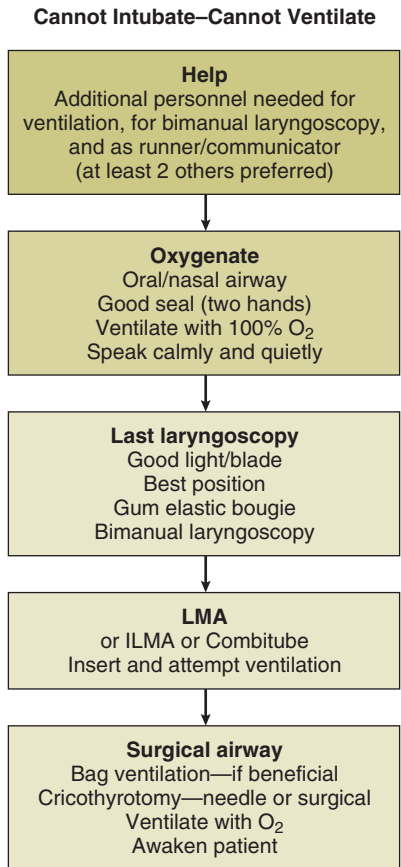
^d Consider reparation of the patient for awake intubation or canceling surgery.

^e Options for emergency noninvasive airway ventilation include (but are not limited to) rigid bronchoscope, esophageal-tracheal Combitube ventilation, and transtracheal jet ventilation.

• **Fig. 2.3** Algorithm for managing the difficult airway. CO₂, Carbon dioxide; LMA, laryngeal mask airway. (Adapted from Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology*. 2003;98:1269–1277.)



• **Fig. 2.4** A four-component algorithm for managing the difficult airway. *ILMA*, Intubating laryngeal mask airway; *LMA*, laryngeal mask airway. (From Henderson JJ, Popat MT, Latto IP, et al. Difficult Airway Society guidelines for management of the unanticipated difficult intubation. *Anaesthesia*. 2004;59:675–694.)



• **Fig. 2.5** Flow chart for the cannot intubate–cannot ventilate scenario. *ILMA*, Intubating laryngeal mask airway; *LMA*, laryngeal mask airway.

by mask, and (5) administration of supplemental oxygen throughout the process.

The Anticipated Difficult Airway

The anticipated difficult airway is the “least lethal” of the three scenarios—with time to consider strategy, optimize patient status, and obtain appropriate adjuncts and personnel. The key questions are as follows:

1. Should the patient be kept awake or be anesthetized for intubation?
2. Which technique should be used for intubation?

Awake Intubation

Awake intubation is more time-consuming, requires experienced personnel, is less pleasant for the patient (compared with intubation under anesthesia), and may have to be abandoned as a result of the patient’s inability or unwillingness to cooperate. Because spontaneous breathing and pharyngeal/laryngeal muscle tone are maintained, however, it is significantly safer. The techniques available are fiberoptic and retrograde intubation. It also may be used in patients judged to be at risk for a difficult airway, whereupon an initial direct laryngoscopic view allows intubation.

Fiberoptic Intubation. Fiberoptic intubation is a technique in which a flexible endoscope with a tracheal tube loaded along its length is passed through the glottis. The tracheal tube is then pushed off the endoscope and into the trachea, and the endoscope is withdrawn. An informed patient, trained assistance, and adequate preparation time make fiberoptic intubation less stressful. The nasotracheal route is used most often and requires the use of nasal vasoconstrictors. Nebulized local anesthetic is delivered to the airway via face mask. Sedation may be given, but ideally the patient should

remain breathing spontaneously and responsive to verbal commands. The procedure often is time-consuming and tends to be used in elective situations⁸⁶ (Box 2.6).

Retrograde Intubation. For retrograde intubation,⁸⁷ local anesthesia is provided and the cricothyroid membrane is punctured by a needle through which a wire or catheter is passed upward through the vocal cords. The wire is visualized in the pharynx, brought out through the mouth, is used to guide the ET through the vocal cords, and then is withdrawn. This technique also can be used to guide a fiberoptic scope through the vocal cords. Owing to time constraints, it is not suitable for emergency airway access and is contraindicated in the presence of an expanding neck hematoma or coagulopathy.

Intubation Under Anesthesia

It may be decided, despite the safety advantage of awake intubation, to anesthetize the patient before attempted intubation. Preparation of the patient, equipment, and staff is paramount (Box 2.7). Adjuncts such as those described later should be available, either to improve the chances of intubation or to provide a safe alternative airway if intubation cannot be achieved.

Unanticipated Airway Difficulty

The DAS guidelines⁸⁵ focus on the unanticipated difficult airway, an unpredictable clinical problem. More focus is placed on the need to be prepared and accountable and to optimize conditions and reduce patient morbidity in this situation. Those involved in

• BOX 2.6 Indications for Fiberoptic Intubation

Anticipated difficult intubation
Avoidance of dental damage in high-risk patient
Direct laryngeal trauma
Other need for awake intubation

• BOX 2.7 Checklist for Anticipated Difficult Intubation of Patient Under General Anesthesia

- Prepare and assess the patient.
- Prepare and test the equipment.
- Ensure skilled assistance with knowledge of BURP/bimanual laryngoscopy.
- Have the following available:
 - A range of laryngoscopes including specialized blades and handles
 - Videolaryngoscope
 - A range of tracheal tubes lubricated and cuffs tested for patency (*women*: 7.0–7.5 mm in internal diameter; *men*: 7.5–9.0 mm in internal diameter)
 - Gum-elastic bougie or tracheal tube stylet
 - Supraglottic airways (LMA, ILMA)
 - Flexible fiberoptic intubation equipment
 - Equipment suitable for emergency invasive airway access
 - An exhaled carbon dioxide monitor
- Check battery and bulb function.
- Check functioning of suction devices.
- Use optimal patient position.
- Preoxygenation with 100% oxygen for 3 to 5 minutes if possible

BURP, Backward, upward, and rightward pressure.

the airway management of critically ill individuals must be familiar with alternative airway devices and techniques, including when to perform emergency invasive airway access. The unanticipated difficult airway allows only a short period to solve the problem if significant hypoxemia, hypercarbia, and hemodynamic instability are to be avoided. The patient usually is anesthetized, may be apneic, and may have received muscle relaxants, and previous initial attempt(s) at intubation may have been unsuccessful. If appropriate equipment, assistance, and experience are not immediately at hand, little time is available to obtain them. Nevertheless, it is essential to maintain oxygenation and avoid hypercarbia if possible—commonly by mask ventilation with 100% oxygen. The four-handed technique often is used. Restoration of ventilation is the priority by either an SGA, an invasive intervention, or by waking the patient if possible. The new DAS guidelines favor the use of second-generation SGAs as they have been designed to provide a better seal and reduce the risk of aspiration.

If the practitioner is inexperienced, if the patient has had no (or a relatively short-acting) muscle relaxant, and if ventilation is not a problem, it may be appropriate to let the patient recover consciousness. An awake intubation can then be planned either after a short period of recovery or on another occasion. With an experienced practitioner, it may be appropriate to continue using techniques to improve the chances of visualizing and intubating the larynx. As discussed next, various adjuncts may be useful in this situation and also in the anticipated difficult airway when it has been decided to intubate with the patient under anesthesia.

The U.K. Royal College of Anaesthetists Fourth National Audit Project (NAP4) provided significant insight into contemporary airway management by identifying several deficiencies that increase the risk of adverse outcomes relating to the airway in critically ill patients.⁸⁸ Failure to plan (ahead) appeared to be a common causative factor in a large number of poor airway outcomes. This was sometimes due to (1) poor assessment (no recognition that there was a potential problem needing a plan), (2) failure to plan even when potential problems were recognized, and (3) failure to have alternative plan(s) (in the event that “plan A” was not successful). Many poor outcomes are the result of repeated use of an approach that has already (sometimes repeatedly) failed. NAP4 revealed a number of scenarios or patient-related factors that seemed to predispose to poor airway outcomes. These predictors are summarized in Box 2.8.

Strategies to Improve First-Attempt Success at Intubation in Critical Illness

Airway management in critically ill patients is high risk owing to both anatomic and physiologic characteristics that increase risk of complications, including aspiration, hypoxemia, hemodynamic compromise, hypoxic brain injury, and death.^{89,90}

• BOX 2.8 Predictors for Poor Airway Outcomes

Use of a supraglottic airway (only) in patients with a recognized airway difficulty

Cases in which awake fiberoptic intubation was indicated, but not used
Failure to use capnography or failure to interpret capnography trace correctly
Airway maneuvers in obese patients, in the emergency department, or in the intensive care unit

Use of emergency cannula cricothyrotomy (60% failure rate)

Displacement of a tracheostomy tube

Events related to emergence, recovery, and extubation

Preoxygenation

The process of preoxygenation replaces the nitrogen-rich air in the alveoli with oxygen. Optimization of this may be suboptimal in critically ill patients who may have physiologic derangements, including high oxygen requirements, shunt physiology, and lack of cooperation, all of which may increase the possibility of hypoxemia. In addition to oxygen supplementation, 20-degree elevation of the head can improve preoxygenation and increase apnea time.⁹¹ Noninvasive ventilation has also been used to improve preoxygenation before intubation in patients with obesity and shunt physiology.⁹² A high-flow nasal cannula can provide humidified high-flow oxygen up to 70 L/min. It is being used increasingly in preoxygenation of patients and also in extubated patients.⁹³

Bimanual Laryngoscopy

Application of pressure on the cricoid area or the upper anterior tracheal wall, or both, by the laryngoscopist (a technique sometimes termed bimanual laryngoscopy) may improve laryngeal view.^{94,95} When the view is optimized by the laryngoscopist, an assistant maintains the pressure and thus the position of the larynx, freeing the hand of the laryngoscopist to perform the intubation. The use of “blind” cricoid pressure, or BURP (backward, upward, and rightward pressure), by an assistant may impair laryngeal visualization.^{94,96,97} The use of cricoid pressure in the difficult intubation scenario has not been extensively studied and opinions remain divided on its effectiveness.

Stylet (“Introducer”) and Gum Elastic Bougie

The *stylet* is a smooth, malleable metal or plastic rod that is placed inside an ET to adjust the curvature—typically into a J- or hockey-stick shape to allow the tip of the ET to be directed through a poorly visualized or unseen glottis. The stylet must not project beyond the end of the ET to avoid potential laceration or perforation of the airway.

The *gum elastic bougie* is a blunt-ended, malleable rod that during direct laryngoscopy may be passed through the poorly or nonvisualized larynx by putting a J-shaped bend at the tip and passing it blind in the midline upward beyond the base of the epiglottis. Then, keeping the laryngoscope in the same position in the pharynx, the ET can be “railroaded” over the bougie, which is then withdrawn. For many critical care practitioners, it is the first-choice adjunct in the difficult intubation situation.^{95,98}

Different Laryngoscope or Blade

More than 50 types of curved and straight laryngoscope blades are available; the most commonly used is the curved Macintosh blade. Using specific blades in certain circumstances has been both encouraged and discouraged. In patients with a large lower jaw or “deep pharynx,” the view at laryngoscopy is often improved significantly by using a size 4 Macintosh blade (rather than the more common adult size 3). This ensures the tip of the blade can reach the base of the vallecula to lift the epiglottis. Other blades, such as the McCoy, may be advantageous in specific situations.

Lighted Stylet

A lighted stylet (light wand) is a malleable fiberoptic light source that can be passed along the lumen of an ET to facilitate blind intubation by transillumination. It allows the tracheal lumen to be distinguished from the (more posterior) esophagus on the basis of the greater intensity of light visible through anterior soft tissues of the neck as the ET passes beyond the vocal cords.⁹⁹ In elective anesthesia, the intubation time and failure rate with light

wand–assisted intubation were similar to those with direct laryngoscopy,¹⁰⁰ and in a large North American survey, the light wand was the preferred alternative airway device in the difficult intubation scenario.¹⁰¹ A potential disadvantage is the need for low ambient light, which may not be desirable (or easily achieved) in a critical care setting.

Video Laryngoscopy

Guidelines for unanticipated difficult intubation emphasize the importance of the first attempt at laryngoscopy with the aim of plan A to maximize the likelihood of success at the first attempt or, failing that, to limit the duration and number of attempts at laryngoscopy. The role of video laryngoscopes in difficult intubation has been recognized by the DAS 2015 guidelines⁸⁵ and it is recommended that all anesthesiologists be trained in and have immediate access to them. Videolaryngoscopes provide a better view of the larynx than with a standard Macintosh blade (direct laryngoscopy).¹⁰² The improved laryngeal view is a result of two factors: for videolaryngoscopes with Macintosh-shaped blades, a camera on the distal end of the blade provides an increased field of view compared with direct laryngoscopy, whereas for videolaryngoscopes with extra curved blades, there is an increased capacity to “see around the corner” and gain a view normally beyond the standard Macintosh blade. They are proposed to have advantages as being less traumatic to the airways and having a higher rate of successful intubation when used as a rescue device if direct laryngoscopy fails.¹⁰³ They may be particularly useful in patients with cervical instability, either by providing a better glottic view or by a reduction in upper cervical movement during intubation.¹⁰⁴ However, an improved laryngeal view does not always equate to a successful intubation. Intubation time can also be prolonged with the videolaryngoscope, especially in inexperienced hands.¹⁰⁵

Videolaryngoscopes may have training advantages—the trainer can observe the screen while the trainee performs a procedure and can help optimize blade position and advise the trainee in real time.

Fiberoptic Intubation

The fiberoptic bronchoscope can be used in the unanticipated difficult airway if it is readily available and the operator is skilled.^{40,106,107} With an anesthetized patient, the technique may be more difficult. Loss of muscle tone tends to allow the epiglottis and tongue to fall back against the pharyngeal wall. This can be counteracted by lifting the mandible.

Cannot Intubate–Cannot Ventilate

A cannot intubate–cannot ventilate scenario is an uncommon but life-threatening situation best managed by adherence to an appropriate algorithm.^{34,85} Fig. 2.5 presents a flow sheet summarizing the appropriate actions.¹⁰⁸

All personnel involved will be pressured (and motivated) by the potential for severe injury to the patient. Efficient teamwork is more likely in an environment that is relatively calm. Although it may be difficult, shouting, impatience, anger, and panic should be avoided in such situations, whereas effective communication may be lifesaving (see “Human Factors”).

Confirming Tube Position in the Trachea

A critical factor in the difficult airway scenario, potentially leading to death or brain injury, is failure to recognize misplacement of the ET. Attempted intubation of the trachea may result in esophageal

intubation. This alone is not life-threatening unless it goes unrecognized.⁷⁸ Thus confirmation of ET placement in the trachea is essential.

Visualizing the ET as it passes between the vocal cords into the trachea is the definitive means of assessing correct tube positioning. This may not always be possible, however, owing to poor visualization. In addition, the laryngoscopist may be reluctant to accept that the ET is not in the trachea. Several clinical observations support the presence of the ET in the trachea.

Chest wall movement with positive-pressure ventilation (manual or mechanical) is usual but may be absent in patients with chronic obstructive pulmonary disease, obesity, or decreased compliance (e.g., in severe bronchospasm). Although condensation of water vapor in the ET suggests that the expired gas is from the lungs, this also may occur with esophageal intubation. The *absence of water vapor* usually is indicative of esophageal intubation. *Auscultation of breath sounds* (in both axillae) supports correct tube positioning but is not absolute confirmation.¹⁰⁹ Apparent inequality of breath sounds heard in the axillae may suggest intubation of a bronchus by an ET that has passed beyond the carina. Of note, after emergency intubation and clinical confirmation of the ET in the trachea, 15% of ETs may still be inappropriately close to the carina.¹¹⁰

The use of capnography to detect ETCO₂ is the most reliable objective method of confirming tube position.¹¹¹ False-positive results may be obtained initially when exhaled gases enter the esophagus during mask ventilation¹¹² or (rarely) when the patient is generating carbon dioxide in the gastrointestinal tract (as with recent ingestion of carbonated beverages or bicarbonate-based antacids).¹¹³ A false-negative result (ET in trachea but no carbon dioxide gas detected) may be obtained when pulmonary blood flow is minimal, as in cardiac arrest.¹¹⁴ Visualizing the trachea or carina through a *fiberoptic bronchoscope*, which may be readily available in critical care, also will confirm correct placement of the ET.

Surgical Airway

The indication for a surgical airway is inability to intubate the trachea in a patient who requires it, and the techniques available are cricothyrotomy and tracheostomy.

Cricothyrotomy

Cricothyrotomy may be performed as a percutaneous (needle) or open surgical procedure (Box 2.9). The indication for both these techniques is the cannot intubate–cannot ventilate situation. Although needle cricothyrotomy is an emergency airway procedure, the technique is similar to that for “mini-tracheostomy,” which is performed electively. Unlike the other surgical airway techniques, a needle cricothyrotomy does *not* create a definitive airway. It will not allow excretion of carbon dioxide but will produce satisfactory oxygenation for 30 to 40 minutes. It can be viewed as a form of apneic ventilation (see later discussion). There are several methods of connecting the intravenous cannula to a gas delivery circuit with the facility to ventilate, using equipment and connections readily available in the hospital. The appropriate method thus should be thought out in advance with equipment available on the difficult airway trolley or bag. New commercial kits that come preassembled also are available.

A surgical cricothyrotomy allows a cuffed tube to be inserted through the cricothyroid membrane into the lower larynx or upper

• BOX 2.9 Procedure: Needle and Surgical Cricothyrotomy

The cricothyroid membrane is diamond-shaped and lies between the thyroid and the cricoid cartilages. Inject subdermal lidocaine and epinephrine (adrenaline) for local anesthesia.

Needle Cricothyrotomy

- Identify the cricothyroid membrane and the midline.
- Insert a 14-gauge intravenous cannula and syringe through the skin and membrane.
- Continuously apply negative pressure until air enters the syringe.
- Stop at this point and push the cannula off the needle into the trachea.
- The insertion of the cannula into the trachea allows apneic (low-pressure) ventilation or jet (high-pressure) ventilation.

Surgical Cricothyrotomy

- Make a 1.5-cm skin incision over the cricothyroid membrane.
- Incise the superficial fascia and subcutaneous fat.
- Divide the cricothyroid membrane (short blade, blunt forceps, or the handle of a scalpel often is used).
- Insert (size 6.0) cuffed tracheostomy tube through membrane between the thyroid and cricoid cartilages.

• BOX 2.10 Tracheostomy: Indications and Contraindications

Indications for Tracheostomy

Inability to maintain a patent airway
 Suspected cervical spine instability (percutaneous technique only)
 Prevention of damage to vocal cords and (possibly) subglottic stenosis
 Abnormal anatomy (percutaneous only)
 Upper airway obstruction
 Requirement for tracheobronchial toilet with suctioning
 Part of larger surgical procedure (e.g., laryngectomy)

Contraindications to Tracheostomy

Local inflammation
 Bleeding disorder (relative)
 Arterial bleeding in neck/upper thorax

trachea. This allows positive-pressure ventilation for considerable periods and also protects against pulmonary aspiration.

Tracheostomy

A tracheostomy is an opening in the trachea—usually between the second and third tracheal rings or one space higher—that may be created surgically or made percutaneously.^{115,116} The indications for and contraindications to tracheostomy are summarized in Box 2.10. Compared with long-term orotracheal or nasotracheal intubation, tracheostomy often contributes to a patient who is less agitated, requires less sedation, and who may be weaned from ventilation more easily.^{33,117} This increased ability to wean is sometimes attributed to reduced anatomic dead space. The potential reduction in sedation after tracheostomy, however, is a much greater advantage to weaning than the small reduction in dead space. The benefits and complications of tracheostomy are listed in Box 2.11. Percutaneous tracheostomy is normally carried out by medical staff in the ICU (Box 2.12).