8th Edition

TINTINALLI'S EMERGENCY MEDICINE MANUAL

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Tintinalli's
Emergency Medicine Manual

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Preface

Prior to the spring of my third year of medical school, I hadn't heard of the specialty emergency medicine. I didn't know where in the medical center our "emergency room" (ER)[1] was and I didn't know that we had a combined emergency medicine (EM)/internal medicine (IM) residency program. Apparently, they didn't promote the program much among the medical students. One day, shortly before I was to begin my final year of medical school, an EM/IM resident enlightened me and convinced me to squeeze an EM elective into my upcoming schedule. Fast forward a few months, I began my EM rotation and was hooked. On September 21,1979, three weeks into my EM elective, emergency medicine (EM) was recognized as the 23rd American specialty. Yes, I'm that old and so is our specialty.

I prepared for my initial EM certification board exams using the first edition of *The Study Guide*. It was well written, easy to read, and much shorter than the current eighth edition of *Tintinalli's Emergency Medicine Manual*, which is derived from the eighth edition of *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. What a great honor it has been to work with Dr. Tintinalli and to contribute to both her namesake textbook and manual.

While a single editor compiled Tintinalli's first *Study Guide*, the eighth edition of *Tintinalli's Emergency Medicine Manual* includes contributors from across the globe, including several African nations where emergency medicine is an emerging specialty. The eighth edition includes "Palliative Care," which was certainly not on emergency medicine's radar in 1979, but is now recognized as a subspecialty of our discipline. We continue to publish the *Manual* in multiple languages for our readers around the world and hope that the *Manual* and its online version at *accessemergencymedicine.mhmedical.com* continues to serve the daily needs of medical students, residents, advanced practice providers, and practicing emergency physicians.

The co-editors Michael T. Fitch, Scott Joing, Vincent Wang, David M. Cline, O. John Ma, and I would like to thank all the authors for their excellent efforts in writing and updating chapters while also maintaining busy clinical schedules. Thanks, too, to the hardworking crew at McGraw Hill Education for their guidance in taking this project from draft to publication: Brian Belval, Christie Naglieri, Jessica Gonzalez, Juanita Thompson, and Poonam Bisht. Finally, I am grateful to have had such wonderful team of editors with whom to work. They made publishing this handbook a delight. Thanks Michael, Scott, Vincent, David, and John.

RKC dedicates this book to Marc, Matthew, Lissy, and Noah, as well as to emergency care providers around the world; MF dedicates this book to Missy, Mira, and Maya, and in memory of Dr. John Marx; SJ dedicates this book to wonderful Elizabeth, Micah, Owen, Britta, and Emmy along with the outstanding Hennepin County Medical Center EM faculty and residents; VW dedicates this book to Esther, Elijah, and Evaline; DMC dedicates this book to family: home, church, and professional; OJM dedicates this book to everyone dedicated to advancing quality of care and patient safety in emergency medicine.

[1]Prior to becoming known as the Emergency Department (ED), the area was known as the emergency room.

SECTION 1

Resuscitation Techniques

CHAPTER 1

Advanced Airway Support

Darren Braude

Airway assessment and management is one of the most critical interventions that emergency physicians perform. Intubation is not always necessary, however, and rushing into invasive airway management before initial resuscitation can be problematic.

RAPID AIRWAY ASSESSMENT

Perform a rapid clinical airway assessment which includes noting the patient's level of responsiveness, skin color, respiratory rate, and depth of respirations. Obtain oxygen saturation and capnography unless the patient is in impending or actual cardiac arrest. The goal is to determine if the patient is maintaining and protecting their airway and meeting critical oxygenation and ventilation goals. Nothing should be placed in the pharynx to assess gag reflex. Emergent and immediate decisions on airway management may proceed before obtaining blood gases and x-rays.

IMPENDING/ACTUAL CARDIAC ARREST

Open the airway and initiate low-volume ventilation unless following cardiocerebral resuscitation protocols. The primary focus of initial cardiopulmonary resuscitation is on establishing quality chest compressions and evaluating for a shockable rhythm. Once these priorities are addressed, the airway can be further managed with an extraglottic device or endotracheal intubation.

BASIC AIRWAY MANAGEMENT

Position the patient to open the airway, drain secretions and maximize oxygenation and ventilation, while maintaining cervical stabilization precautions if indicated. Place conscious patients in a sitting position, if possible, and unconscious patients on their side unless they require urgent invasive procedures. Patients who are unable to maintain an open airway should have one or two properly sized nasal trumpets placed if they are not anticoagulated or at risk for mid-face fractures; an oral airway may be used instead of, or in

addition to, the nasal airways if no gag reflex present. Provide supplemental oxygen if the room air saturation is below 94% with the goal of increasing saturation to above 94%; high flow oxygen should be avoided when possible.

NONINVASIVE POSITIVE PRESSURE VENTILATION

If ventilation is adequate but oxygenation is poor, consider immediate initiation of noninvasive ventilation. Noninvasive positive pressure ventilation (NIPPV) may be used as a temporizing measure while other treatments are initiated (e.g., nitrates in acute cardiogenic pulmonary edema), for preoxygenation prior to intubation in any medical condition, or as an alternative to invasive airway management in some cases, such as in patients with DNR or DNI status. NIPPV for emergency situations is commonly delivered via a full-face mask using either continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BPAP) using a ventilator, stand-alone reusable device, or a disposable device (CPAP only). CPAP provides the same amount of pressure support during inspiration and positive end-expiratory pressure (PEEP) during exhalation—usually 5 to 10 mmHg—while BPAP allows for increasing pressure support up to 15 mm Hg without overwhelming the patient with expiratory resistance, which may remain at 5 to 10 mm Hg. There are no studies showing a significant advantage to one system over another.

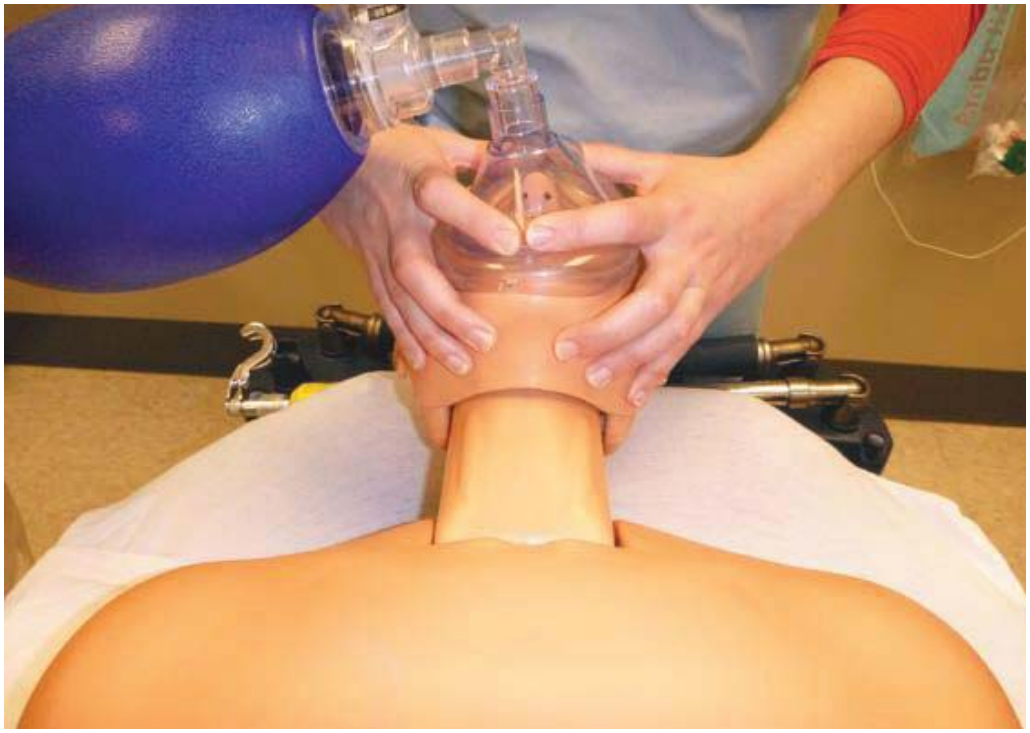
MASK VENTILATION

Begin mask ventilation for patients with poor respiratory effort. Patients should be placed in a sniffing or ramped position with airway adjuncts as previously discussed. Apply a properly fitted mask with one provider dedicated to maintaining a tight seal while a second provider or mechanical ventilator provides just enough volume to raise the chest. Two different hand grips are described to achieve a mask seal during two-person mask ventilation with the "T-E" preferred over the "E-C" in most cases (Fig. 1-1). If you are unable to achieve a tight mask seal consider placing an extraglottic device *if there is no gag reflex or other contraindication.* If good chest rise is noted but saturations remain poor despite supplemental oxygen, add PEEP.

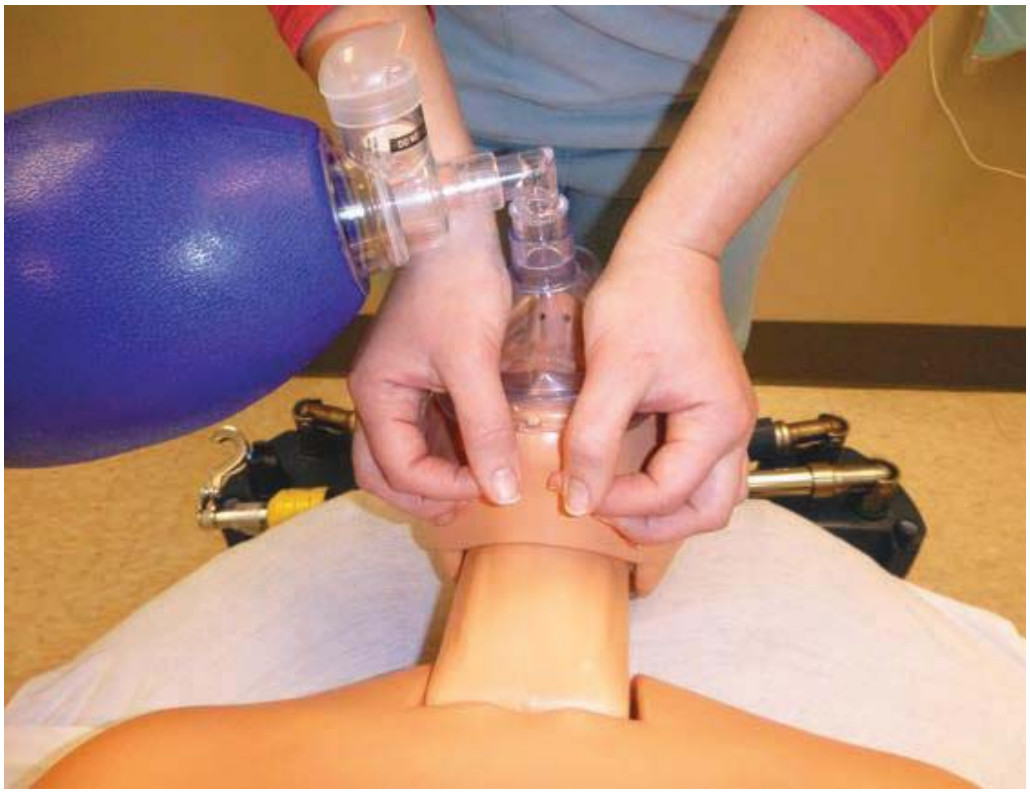
EXTRAGLOTTIC DEVICES

Extraglottic devices (EGDs) are placed blindly and fit into the following category: (1) supraglottic devices that include a mask that sits internally over the glottic opening or (2) retroglottic, dual-balloon devices that sit within the proximal esophagus and include distal and proximal balloons to direct the ventilation that occurs through holes between the two balloons into the airway. Supraglottic devices include, but are not limited to, the Ambu Auragain®, LMA Supreme®, LMA Protector®, LMA Fastrach, Intersurgical iGel®, and CookGas AirQ. Retroglottic devices include the Esophageal-Tracheal Combitube, the Rusch EasyTube, and the King Laryngeal Tube®. Many of these devices now include a channel for gastric decompression (theoretically lessens the risk of aspiration) and some facilitate blind or endoscopic intubation.

Extraglottic devices are most commonly used in the ED after a failed airway but may also be used primarily during cardiac arrest, for difficult mask



A



B

FIGURE 1-1. Mask ventilation: traditional "E-C" hand grip (A) and modified "T-E" hand grip (B).

ventilation or as part of rapid sequence airway procedures. It is critical to always have an appropriately sized EGD available during airway management to place the device in case difficulties are encountered but do not rely on an EGD to the exclusion of surgical airway when critical hypoxemia is encountered.

INTUBATION

Intubate patients in cardiac arrest after other critical resuscitation steps have been assured. Intubation is indicated for unconscious, nonarrested patients unless a rapidly correctable situation is suspected, such as an opioid overdose or simple postictal state. Consider intubation for conscious patients with refractory hypoxemia or a deteriorating clinical course. Rapid sequence intubation (RSI) technique should be used unless the patient's condition makes it unnecessary (i.e., cardiac arrest) or when it is contraindicated because of an anticipated difficult airway. RSI includes the simultaneous administration of an induction agent and a neuromuscular blocking agent to facilitate orotracheal intubation in the nonarrested/peri-arrested patient. Anticipated difficulty in mask ventilation, intubation, rescue with an extraglottic device and surgical airway placement are relative contraindications to RSI; awake techniques should be considered in these circumstances. Current evidence suggests that multiple intubation attempts are associated with adverse events. Thus, all efforts should be made to set up success on the first intubation attempt.

OROTRACHEAL INTUBATION

1. Prepare equipment, personnel, and drugs before attempting intubation. Assess airway difficulty and anticipate required airway rescue. Assemble and place suction, bag-valve-mask, and rescue devices within easy reach. Sufficient personnel should be present at the bedside to assist. Assign all the tasks in advance, including medication administration, cervical spine stabilization, external laryngeal manipulation, etc. *Use of a checklist is strongly encouraged.*
2. Ensure adequate ventilation and oxygenation and monitoring while preparing equipment. Preoxygenate with a non-rebreather oxygen mask at maximal oxygen flow rates, NIPPV, or mask ventilation if the patient is not ventilating adequately. Place a nasal cannula with up to 15 L/min of oxygen flow under the mask to provide for apneic oxygenation. *An inability to achieve an oxygen saturation of greater than 93% with these maneuvers places the patient at risk for critical desaturation after apnea is induced*; be prepared to perform controlled positive pressure mask ventilation.
3. Optimize physiology prior to intubation *if at all possible* to lessen the risk of peri-intubation complications. This may include administration of IV fluid boluses, inotropes, and/or vasopressors in addition to oxygenation as above.
4. Select, connect, and test the laryngoscope and blade. Video laryngoscopy (VL) is a good first choice if the operator is familiar with this technique. Direct laryngoscopy (DL) is a reasonable option if the operator has more experience with this technique. Select and test the endotracheal tube, commonly 7.5 mm in women and 8 mm in men. Use a stylet with a "straight-to-cuff" configuration for DL/nonhyperangulated VL blades; hyperangulated VL blades often come with proprietary stylets that include an optimal bend (Fig. 1-2).
5. Position the patient in the sniffing or ramped position to align the external ear canal and sternal notch (Fig. 1-3). If C-spine injury is suspected, maintain the head and neck in a neutral position with an assistant performing inline stabilization and a jaw thrust maneuver.

6. Evidence is mixed on whether pretreatment improves outcomes and is no longer routinely recommended. **Fentanyl,** 3 μg/kg, may be considered in normotensive patients with possible raised intracranial pressure, cardiac ischemia, or aortic dissection.

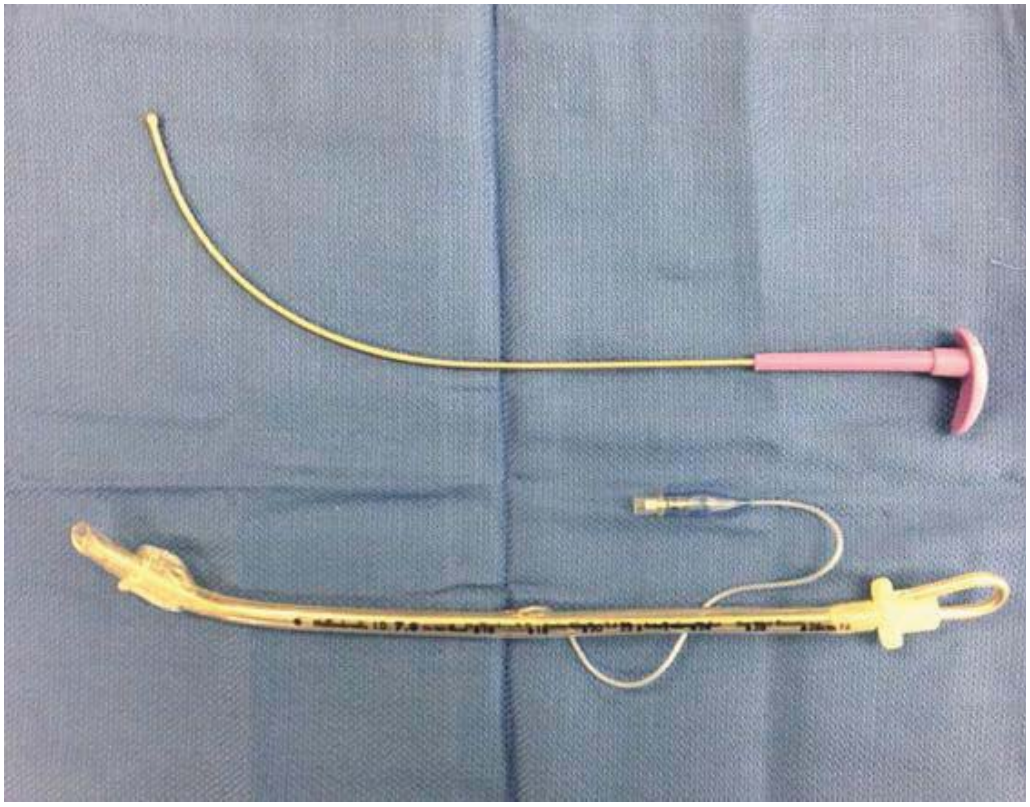


FIGURE 1-2. Top shows a stylet from Intubrite® intended for the hyperangulated video blade. Bottom demonstrates straight-to-cuff stylet shape for direct laryngoscopy.

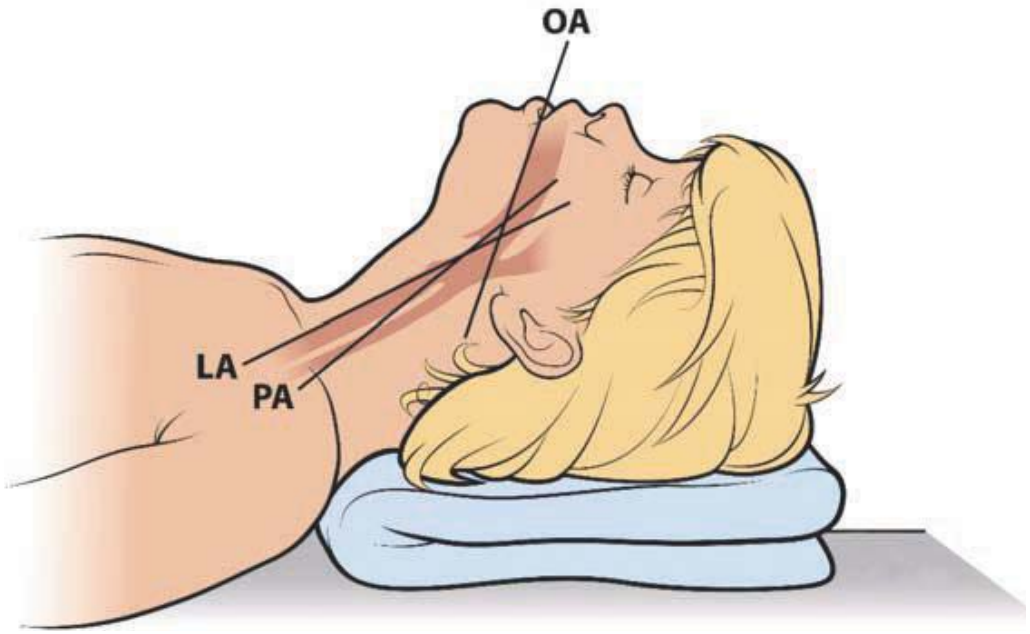


FIGURE 1-3. Sniffing position for optimal mask ventilation and intubation when cervical precautions not indicated. With permission from The Difficult Airway Course™ (www.theairwaysite.com).

7. Administer an intravenous induction agent via rapid push. **Etomidate,** 0.3 mg/kg, is an excellent choice in most circumstances. **Ketamine,** 1 to 2 mg/kg, has become a popular alternative and is generally safe, although cases of hypotension and hypertension have been reported. **Propofol,** 0.5 to 1.5 mg/kg, is another option in patients who are not at risk for hypotension.
8. The induction agent is immediately flushed with a paralytic agent. **Succinylcholine,** 1 to 2 mg/kg of total body weight, is commonly used unless there is risk of serious hyperkalemia (e.g., renal failure, neuromuscular disorders, subacute spinal cord injury, crush injury or burns). **Rocuronium,** 1 to 1.5 mg/kg of ideal body weight, is an increasingly common alternative.
9. Cricoid pressure is no longer recommended due to limited evidence of benefit and clear evidence of worsening laryngoscopic view.
10. Wait for paralysis to occur to diminish the risk of vomiting and aspiration. **Succinylcholine** usually takes effect in 30 to 45 seconds and **rocuruonium** in 60 seconds. Oxygenation should continue via nonrebreather or gentle mask ventilation during this interval.
11. Insert hyperangulated VL blades in the midline. Insert traditional curved blades (whether direct or video) on the right side of the mouth and sweep tongue to the left. Both blades are advanced into the valeculla to trigger to the hyoepiglottic ligament. *Do not over-insert hyperangulated blades*; keep the blade as shallow as possible with the airway visualized in the top half of the screen. Insert straight blades on the right side of the tongue and maintain this "paraglossal" position without sweeping the tongue and gently advance blade as far as it will go. Withdraw the blade slowly until the epiglottis drops into view and then lift it with the tip of the blade. Lift all the blades along the axis of the laryngoscope handle to avoid levering the blade on the teeth and causing dental trauma.
12. If only the epiglottis is visible, use an intubating stylet (aka Bougie) and/or perform external laryngeal manipulation of the thyroid cartilage with the operator's right hand on top of an assistant's hand (Fig. 1-4) to help bring the cords into view.
13. Once the vocal cords or posterior cartilages are visualized, gently pass the tube between the cords (or anterior to the posterior cartilages) until the balloon completely disappears and remove the stylet. When using a hyperangulated blade stylet, it helps to withdraw the stylet 2 to 3 cm once the tip of the tube just enters the airway, before advancing further. Advance tubes in adult females to approximately 21 cm at the corner of the mouth and in adult males to approximately 23 cm and then remove the stylet.
14. Confirm tracheal tube placement immediately with ETCO2. Confirm appropriate depth by listening for bilateral lung sounds and then secure tube. Obtain a portable chest x-ray to further evaluate tube depth and lung pathology. A chest x-ray should never be used to assess tracheal versus esophageal positioning.
15. *Abort the intubation attempt early if oxygen saturation is dropping* and begin immediate mask ventilation. Consider an additional attempt when saturations are maintained in the normal range with appropriate

FIGURE 1-4. External laryngeal manipulation with the intubator's right hand placed on top of assistant's hand which is holding the laryngeal cartilage. Another assistant is maintaining in-line cervical stabilization and providing a jaw thrust.

modification to the operator, laryngoscope and blade selection, patient positioning, use of bougie, etc. If unable to maintain saturations with mask ventilation, insert an EGD while preparing for a possible surgical airway. If saturations are maintained but intubation is unsuccessful within three attempts, or deemed unlikely to be successful at any point, place an EGD.

Surgical Airway

A surgical airway is performed either when intubation via the mouth or nose is not considered a reasonable clinical option or when intubation has failed and critical oxygen saturation cannot be maintained via other means.

A surgical airway is contraindicated in children younger than 10 years of age in whom transtracheal jet ventilation is the preferred subglottic technique. Although several surgical techniques have been described, the bougie-aided technique is described here. There are kits available for Seldinger-based and other "less invasive" techniques but these are not reviewed here.

1. Use sterile technique if possible.
2. Palpate the cricothyroid membrane and stabilize the larynx.
3. With a scalpel, make a vertical, 3- to 4-cm incision starting at the superior border of the thyroid cartilage. Incise caudally toward the suprasternal notch (Fig. 1-5).
4. Identify the cricothyroid membrane using blunt dissection if necessary and make a 2-cm horizontal incision. Immediately withdraw and secure the blade while inserting a gloved finger into the incision.
5. Place an adult bougie into the incision with the coude tip directed distally. The bougie should pass easily without resistance until hold-up is appreciated in the smaller airways, generally confirming tracheal placement.
6. Pass a 6.0-cuffed endotracheal tube or #4 cuffed tracheostomy tube over the bougie and into the airway (Fig. 1-6). If using an endotracheal tube stop advancing as soon the cuff is completely within the airway. Inflate cuff.
7. Confirm with capnography and easy chest rise with bilateral breath sounds.
8. Secure the tube.

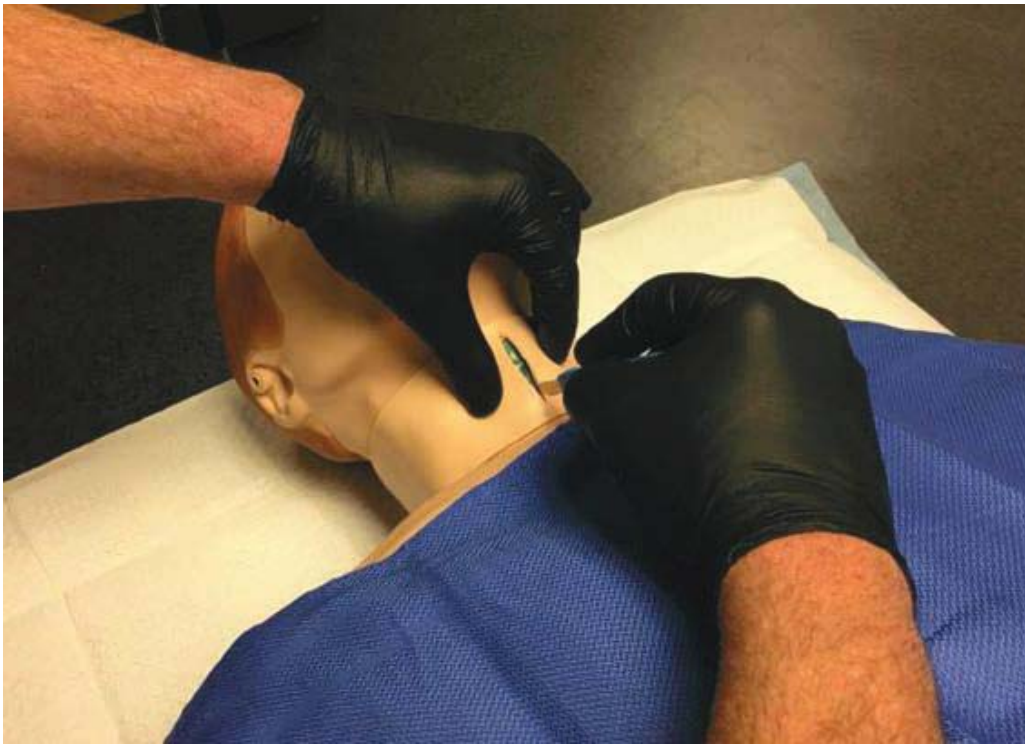


FIGURE 1-5. A 3- to 4-cm vertical midline incision overlying the cricothyroid membrane while the laryngeal cartilage is stabilized.

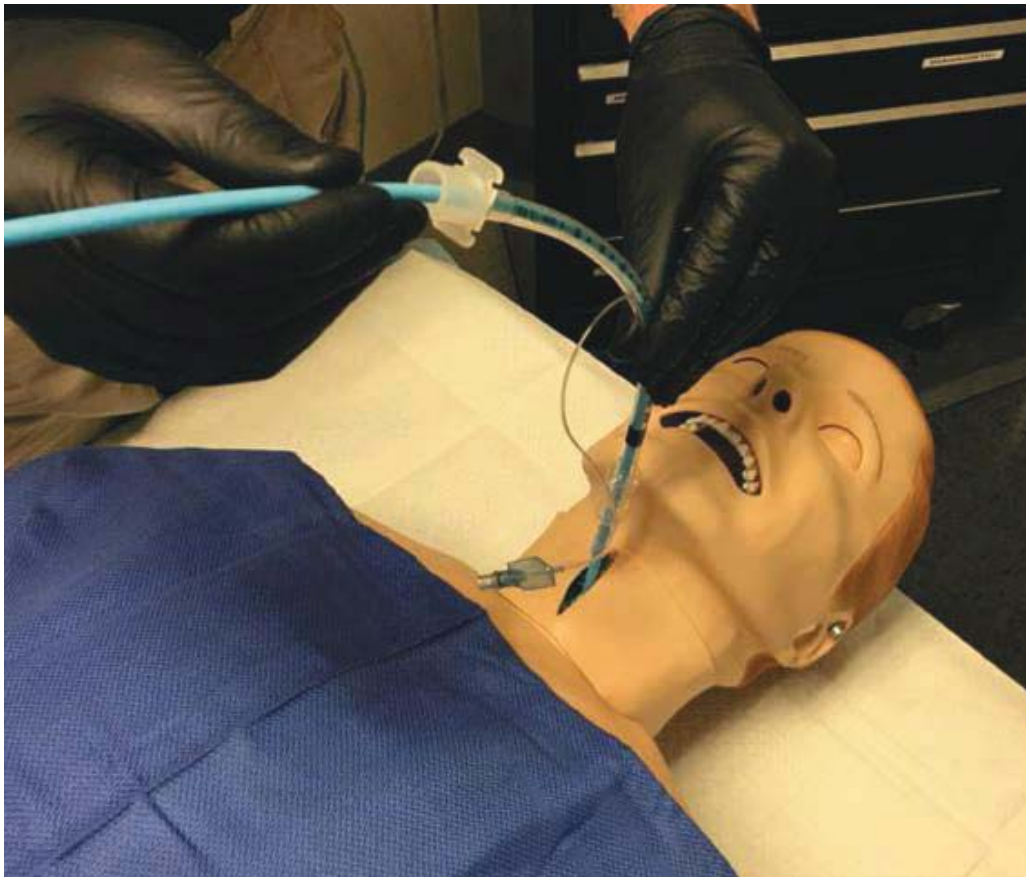


FIGURE 1-6. Passing a 6-0 endotracheal tube over a bougie that has been placed into the trachea through the cricothyroid membrane incision.

FURTHER READING

For further reading in *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th ed., see Chapter 28, "Noninvasive Airway Management," by Jestin N. Carlson and Henry E. Wang; Chapter 29, "Intubation and Mechanical Ventilation," by Robert J. Vissers and Daniel F. Danzl; and Chapter 30, "Surgical Airways," by Michael D. Smith and Donald M. Yealy.

CHAPTER 2

Management of Cardiac Rhythm Disturbances

James K. Takayesu

NONTACHYCARDIC IRREGULAR DYSRHYTHMIAS

Sinus Arrhythmia

Some variation in the sinoatrial (SA) node discharge rate is common; however, if the variation exceeds 120 milliseconds between the longest and shortest intervals, sinus arrhythmia is present. The electrocardiogram (ECG) characteristics of sinus arrhythmia are (*a*) normal sinus P waves and PR intervals, (*b*) 1:1 atrioventricular (AV) conduction, and (*c*) variation of at least 120 milliseconds between the shortest and longest P–P interval (Fig. 2-1). If two or more different P wave morphologies are present, atrial ectopy, wandering atrial pacemaker, or another competing nonsinus focus may be present. Sinus arrhythmias are affected primarily by respiration and are most commonly found in children and young adults, disappearing with advancing age. Occasional junctional escape beats may be present during very long P–P intervals. No treatment is required.

Premature Atrial Contractions

Premature atrial contractions (PACs) have the following ECG characteristics: (*a*) the ectopic P wave appears sooner (premature) than the next expected sinus beat; (*b*) the ectopic P wave has a different shape and direction; and (*c*) the ectopic P wave may or may not be conducted through the AV node (Fig. 2-2). Most PACs are conducted with typical QRS complexes, but some may be conducted aberrantly through the infranodal system, typically with a right bundle branch block pattern. When the PAC occurs during the absolute refractory period, it is not conducted. Since the sinus node is often depolarized and reset, the interval between normal P waves before and after the PAC will not be twice the existing P to P interval, creating a shorter pause than a fully compensatory pause (unlike that seen after most premature ventricular contractions). PACs are associated with stress, fatigue, alcohol use, tobacco, coffee, chronic obstructive pulmonary disease (COPD), digoxin toxicity, and coronary artery disease, and may occur after adenosine-converted paroxysmal supraventricular tachycardia (PSVT). Patients may complain of palpitations or an intermittent "sinking" or "fluttering" feeling in the chest. PACs are common in all ages, often in the

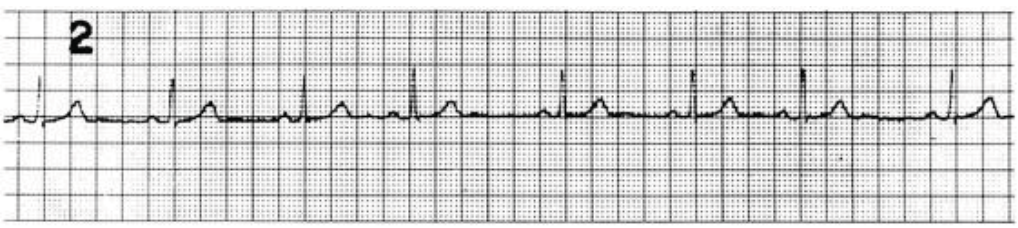


FIGURE 2-1. Sinus arrhythmia.

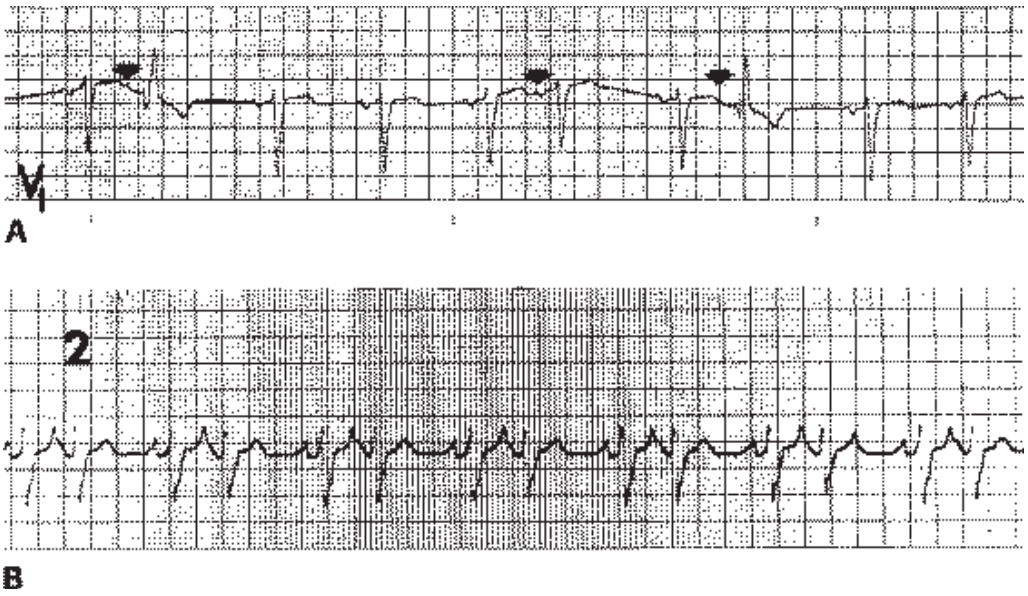


FIGURE 2-2. Premature atrial contractions (PACs). **A.** Ectopic P′ waves (arrows). **B.** Atrial bigeminy.

absence of significant heart disease, but can precipitate sustained atrial tachycardia, flutter, or fibrillation under certain circumstances.

Emergency Department Care and Disposition

1. Discontinue precipitating drugs (alcohol, tobacco, or coffee) or toxins.
2. Treat underlying disorders (stress or fatigue).

Premature Ventricular Contractions

Clinical Features

Premature ventricular contractions (PVCs) are due to impulses originating from single or multiple areas in the ventricles. The ECG characteristics of PVCs are as follows: (*a*) a premature and wide QRS complex; (*b*) no preceding P wave; (*c*) the ST segment and T wave of the PVC are directed opposite the preceding major QRS deflection; (*d*) most PVCs do not affect the sinus node, so there is usually a fully compensatory postectopic pause, or the PVC may be interpolated between two sinus beats; (*e*) many PVCs have a fixed coupling interval (within 40 milliseconds) from the preceding sinus beat; and (*f*) many PVCs are conducted into the atria, thus producing a retrograde P wave (Fig. 2-3). If three or more PVCs occur in a row, patients are considered to have nonsustained ventricular tachycardia.

PVCs are very common, occurring in most patients with ischemic heart disease and acute myocardial infarction (MI). Other common causes of PVCs include digoxin toxicity, congestive heart failure (CHF), hypokalemia, alkalosis, hypoxia, and sympathomimetic drugs. Pooled data and meta-analyses have found no reduction in mortality from suppressive or prophylactic treatment of PVCs. Ventricular parasystole occurs when the ectopic ventricular focus fires frequently enough to compete with the SA node and is associated with cardiac ischemia, electrolyte imbalance, and hypertensive or ischemic heart disease.

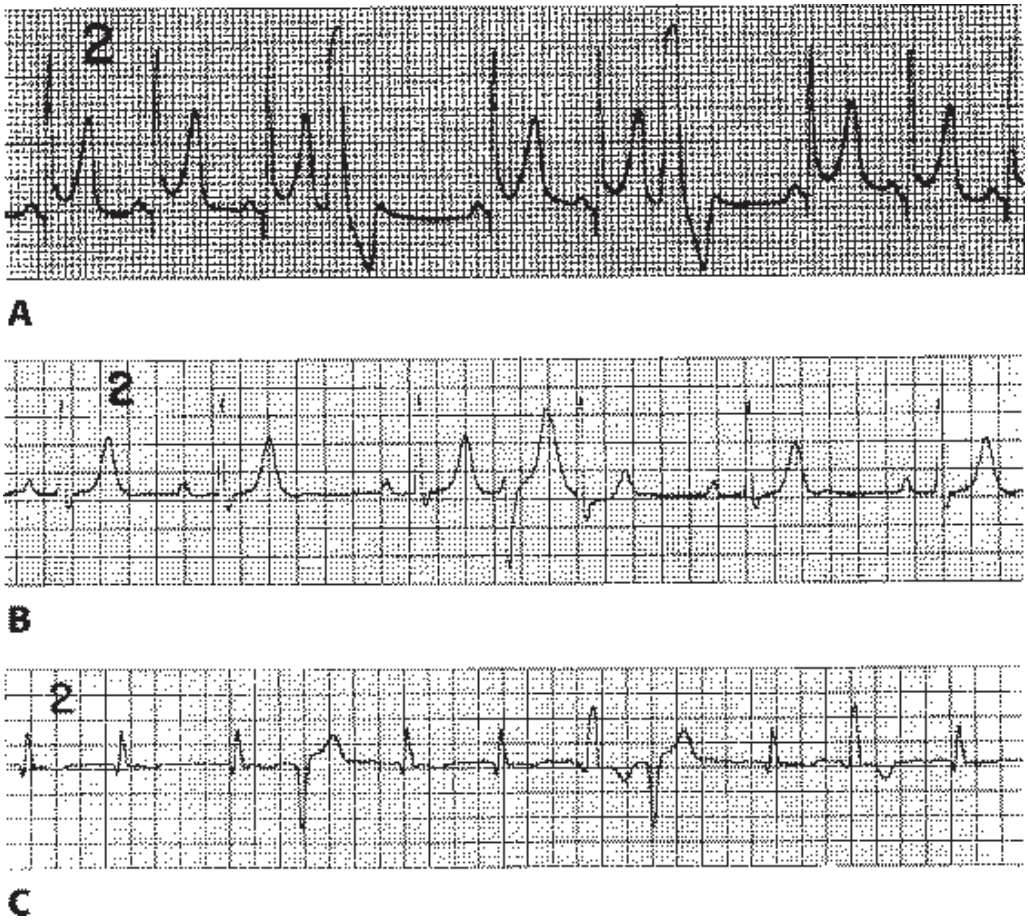


FIGURE 2-3. Premature ventricular contractions (PVCs). **A.** Unifocal PVC. **B.** Interpolated PVC. **C.** Multifocal PVCs.

Emergency Department Care and Disposition

1. Stable patients require no treatment.
2. Patients with three or more sequential PVCs should be managed as non-sustained VT.
3. Potential causes such as hypoxia, drug effect, or electrolyte disturbances should be treated.

BRADYDYSRHYTHMIAS

Sinus Bradycardia

Clinical Features

Sinus bradycardia occurs when the SA node rate becomes slower than 60 beats/min. The ECG characteristics of sinus bradycardia are (*a*) normal sinus P waves and PR intervals, (*b*) 1:1 AV conduction, and (*c*) atrial rate slower than 60 beats/min. Sinus bradycardia represents a suppression of the sinus node discharge rate, usually in response to three categories of stimuli: (*a*) physiologic (vagal tone), (*b*) pharmacologic (calcium channel blockers, β-blockers, or digoxin), and (*c*) pathologic (acute inferior myocardial infarction (MI), increased intracranial pressure, carotid sinus hypersensitivity, hypothyroidism, or sick sinus syndrome).

Emergency Department Care and Disposition

Sinus bradycardia usually does not require specific treatment unless the heart rate is slower than 50 beats/min and there is evidence of hypoperfusion.

1. **Transcutaneous cardiac pacing** is the only Class I treatment for unstable patients.
   a. Attach the patient to the monitor leads of the external pacing device.
   b. When placing transcutaneous pacing pads, place the anterior pad over the left lateral precordium and the posterior pad at the level of the heart in the right infrascapular area. Do not use multifunction pacing defibrillation pads unless the patient is unconscious as the pads cause a lot of discomfort.
   c. Slowly increase the pacing output from 0 mA to the lowest point where capture is observed, usually at 50 to 100 mA, but may be up to 200 mA. A widened QRS after each pacing spike denotes electrical capture.
   d. If needed, administer a sedative, such as lorazepam, 1 to 2 mg IV, or an opiate, such as morphine, 2 to 4 mg IV, for pain control.
2. **Atropine** is a Class IIa treatment for symptomatic bradycardia. The dose is 0.5 mg IV push, repeated every 3 to 5 minutes as needed up to a total of 3 mg IV. If given via endotracheal tube, increase the dose by 2 to 2.5 times over the IV dose. Slow administration or lower doses may cause paradoxical bradycardia. Atropine may not be effective in cardiac transplant patients since the heart is denervated and has no vagal stimulation.
3. **Epinephrine**, 2 to 10 μg/min IV, or **dopamine**, 3 to 10 μg/kg/min IV, may be used if external pacing is not available.
4. Permanent pacemaker placement is indicated in the patient with symptomatic recurrent or persistent sinus bradycardia due to sick sinus syndrome.
5. Glucagon 3 to 10 mg IV over 1 to 2 minutes, followed by an infusion of 1 to 5 mg/h may be used in β-blocker or calcium channel blocker toxicity.

Junctional Rhythms

Clinical Features

In patients with sinus bradycardia, SA node exit block, or AV block, junctional escape beats may occur, usually at a rate between 40 and 60 beats/min, depending on the level of the rescue pacemaker within the conduction system. Junctional escape beats may conduct retrogradely into the atria, but the QRS complex usually will mask any retrograde P wave (Fig. 2-4). When alternating rhythmically with the SA node, junctional escape beats

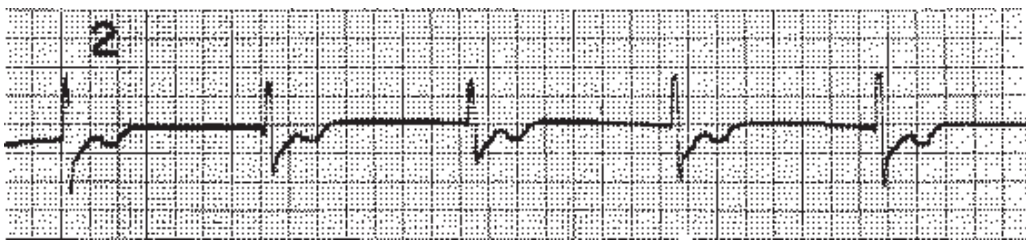


FIGURE 2-4. Junctional escape rhythm, rate 42.

may cause bigeminal or trigeminal rhythms. Sustained junctional escape rhythms may be seen with CHF, myocarditis, acute MI (especially inferior MI), hyperkalemia, or digoxin toxicity ("regularized Afib"). If the ventricular rate is too slow, myocardial or cerebral ischemia may develop. In cases of enhanced junctional automaticity, junctional rhythms may be accelerated (60 to 100 beats/min) or tachycardic (≥100 beats/min), thus overriding the SA node rate.

Emergency Department Care and Disposition

1. Isolated, infrequent junctional escape beats usually do not require specific treatment.
2. If sustained junctional escape rhythms are producing symptoms, treat the underlying cause.
3. In unstable patients, give **atropine** 0.5 mg IV every 5 minutes to a total of 3 mg. This will accelerate the SA node discharge rate and enhance AV nodal conduction.
4. Use transcutaneous or transvenous pacing in unstable patients not responsive to atropine.
5. Manage patients with digoxin toxicity as discussed for SVT.

Idioventricular Rhythm

Clinical Features

The ECG characteristics of idioventricular rhythm (IVR) are (*a*) wide and regular QRS complexes; (*b*) a rate between 40 and 100 beats/min, often close to the preceding sinus rate; (*c*) mostly runs of short duration (3 to 30 beats/min); and (*d*) an AIVR often beginning with a fusion beat (Fig. 2-5). This condition is found most commonly with an acute MI or in the setting of reperfusion after successful thrombolysis.

Emergency Department Care and Disposition

Treatment is not necessary unless the patient is unstable or pulseless. On occasion, especially after reperfusion therapy, the IVR may be the only functioning pacemaker, and suppression with lidocaine can lead to cardiac asystole.

If the patient is hypotensive or in arrest, treatment includes identifying contributing mechanical factors (e.g., aggressive volume resuscitation) and α-adrenergic agents.

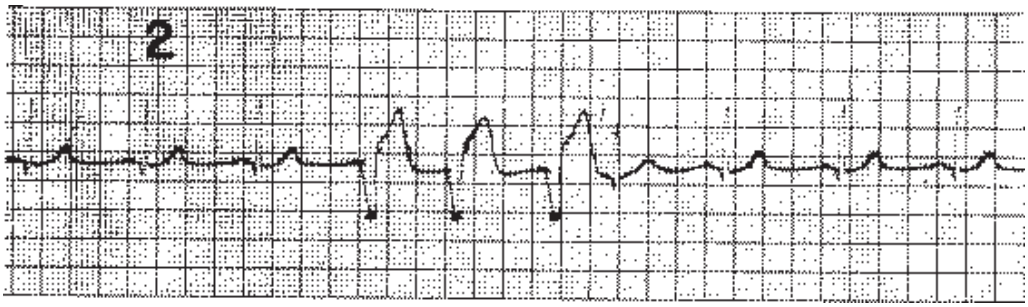


FIGURE 2-5. Accelerated idioventricular rhythms (AIVRs).

Sick Sinus Syndrome

Clinical Features

Otherwise known as tachy-brady syndrome, sick sinus syndrome consists of a variety of abnormalities in impulse generation and conduction, leading to various supraventricular tachycardic rhythms as well as bradycardia due to sinus arrest and SA block. It can be seen in myocardial ischemia, myocarditis, rheumatologic disease, cardiomyopathies, or metastatic disease. Conditions that increase vagal tone such acute abdominal pain, thyrotoxicosis, and hypo- or hyperkalemia exacerbate this condition.

Emergency Department Care and Disposition

Treatment should be based on the presenting rhythm depending on the heart rate and patient instability. Temporary pacing may be needed and admission for permanent pacemaker placement is frequently indicated.

ATRIOVENTRICULAR BLOCKS

First-Degree Atrioventricular (AV) Block

First-degree AV block is characterized by a delay in AV conduction, manifested by a prolonged PR interval (>200 milliseconds). It can be found in normal hearts and in association with increased vagal tone, digoxin toxicity, inferior MI, amyloid, and myocarditis. First-degree AV block needs no treatment. Second-degree AV block is characterized by intermittent AV nodal conduction: some atrial impulses reach the ventricles, whereas others are blocked, thereby causing "grouped beating." These blocks can be subdivided into nodal blocks which are typically reversible and infranodal blocks which are due to irreversible conduction system disease. Third-degree AV block is characterized by complete interruption in AV conduction with resulting AV dissociation.

Second-Degree Mobitz I (Wenckebach) AV Block

Clinical Features

Mobitz I AV block is a nodal block causing a progressive prolongation of conduction through the AV node until the atrial impulse is completely blocked. Usually, only one atrial impulse is blocked at a time. After the dropped beat, the AV conduction returns to normal and the cycle usually repeats itself with the same conduction ratio (fixed ratio) or a different conduction ratio (variable ratio). Although the PR intervals progressively lengthen before the dropped beat, the increments by which they lengthen *decrease* with successive beats causing a progressive *shortening* of each successive R–R interval before the dropped beat (Fig. 2-6). This block is

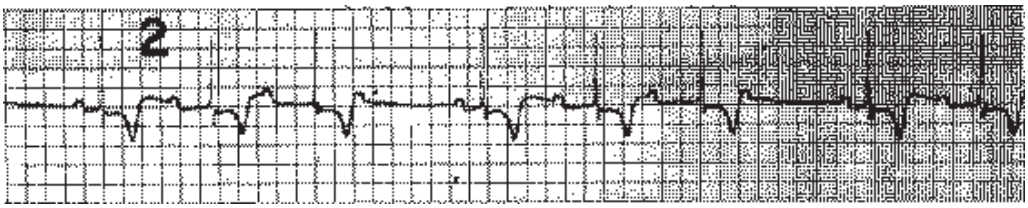


FIGURE 2-6. Second-degree Mobitz I (Wenckebach) AV block 4:3 AV conduction.

often transient and usually associated with an acute inferior MI, digoxin toxicity, or myocarditis or can be seen after cardiac surgery. Because the blockade occurs at the level of the AV node itself rather than at the infranodal conducting system, this is usually a stable rhythm.

Emergency Department Care and Disposition

1. Specific treatment is not necessary unless slow ventricular rates produce signs of hypoperfusion.
2. In cases associated with acute inferior MI, provide adequate volume resuscitation before initiating further interventions.
3. Administer **atropine** 0.5 mg IV repeated every 5 minutes. Titrate to the desired heart rate or until the total dose reaches 3 mg.
4. Although rarely needed, transcutaneous pacing may be used.

Second-Degree Mobitz II AV Block

Clinical Features

Mobitz II AV block is typically due to infranodal disease, causing a constant PR interval with intermittent nonconducted atrial beats (Fig. 2-7). One or more beats may be nonconducted at a single time. This block indicates significant damage or dysfunction of the infranodal conduction system; therefore, the QRS complexes are usually wide coming from the low His–Purkinje bundle or the ventricles. Type II blocks are more dangerous than type I blocks because they are usually permanent and may progress suddenly to complete heart block, especially in the setting of an acute anterior MI, and almost always require permanent cardiac pacemaker placement. When second-degree AV block occurs with a fixed conduction ratio of 2:1, it is not possible to differentiate between a Mobitz type I (Wenckebach) and Mobitz type II block.

Emergency Department Care and Disposition

1. **Atropine** 0.5 to 1 mg IV bolus repeated every 5 minutes as needed up to 3 mg total dose is first-line treatment for symptomatic patients but

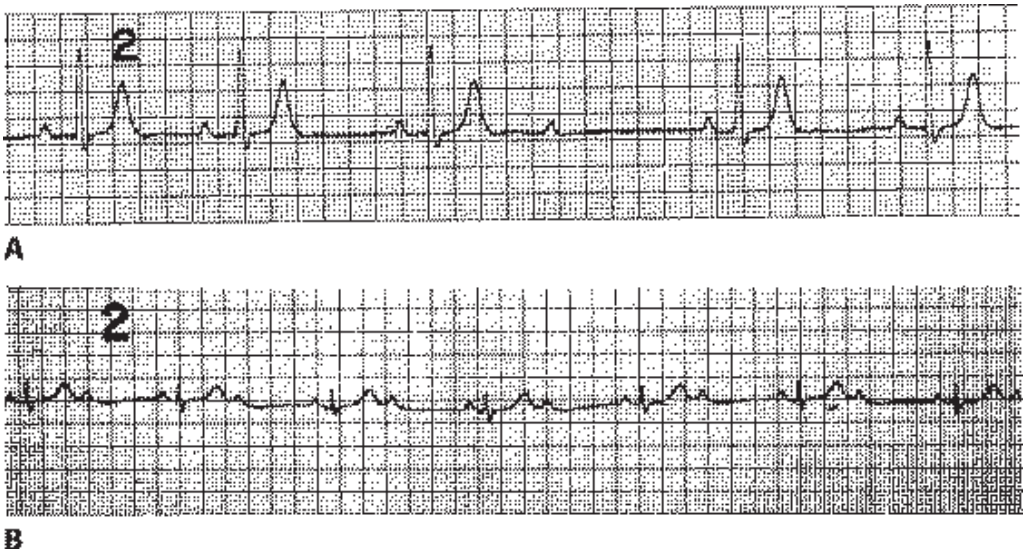


FIGURE 2-7. A. Second-degree Mobitz II AV block. **B.** Second-degree AV block with 2:1 AV conduction.

may be ineffective. All patients should have transcutaneous pacing pads positioned and ready for use in the case of further deterioration into complete heart block.
2. Initiate transcutaneous cardiac pacing (see section on sinus bradycardia) in patients unresponsive to atropine.
3. If transcutaneous pacing is unsuccessful, initiate transvenous pacing (0.2 to 20 mA at 40 to 140 beats/min via a semi-floating or balloon-tipped pacing catheter).

Third-Degree (Complete) AV Block

Clinical Features

In third-degree AV block, there is no AV conduction. The ventricles are paced by an escape pacemaker from the AV node or the infranodal conduction system at a rate slower than the atrial rate (Fig. 2-8). When third-degree AV block occurs at the AV node, a junctional escape pacemaker takes over with a ventricular rate of 40 to 60 beats/min, and because the rhythm originates from above the bifurcation of the His bundle, the QRS complexes are narrow. Nodal third-degree AV block may develop in up to 8% of acute inferior MIs and it is usually transient, although it may last for several days.

When third-degree AV block occurs at the infranodal level, the ventricles are driven by a ventricular escape rhythm at a rate slower than 40 beats/min. Third-degree AV block located in the bundle branch or the Purkinje system invariably has an escape rhythm with a wide QRS complex. Like Mobitz type II block, this indicates structural damage to the infranodal conduction system and can be seen in acute anterior MIs. The ventricular escape pacemaker is usually inadequate to maintain cardiac output and is unstable with periods of ventricular asystole.

Emergency Department Care and Disposition

1. Perform transcutaneous cardiac pacing in unstable patients until a transvenous pacemaker can be placed.
2. In stable patients, apply transcutaneous pacing pads. Treat the same as second-degree Mobitz II AV block.

FASCICULAR BLOCKS

Conduction blocks may arise in one or more of the three infranodal conduction pathways. Blockage of either of the left fascicles does not prolong the QRS duration, but will change the QRS axis. Left anterior fascicular block (LAFB) causes left axis deviation with qR complex seen in aVR, while the

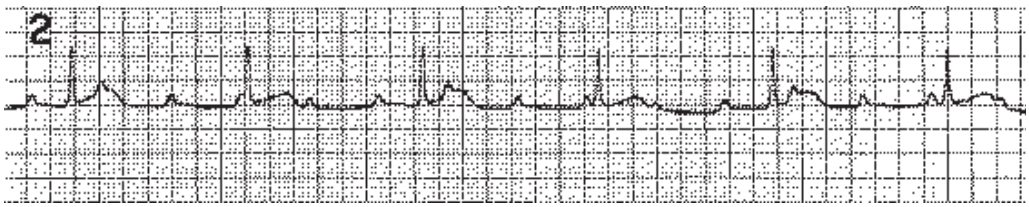


FIGURE 2-8. Third-degree AV block.

much less common left posterior fascicular block (LPFB) causes right axis deviation. Right bundle branch block (RBBB) will prolong the QRS duration (>120 milliseconds) and cause an RSR', or "rabbit ears," in the early precordial leads (V1–2). Bifascicular block denotes a combination of any two of these fascicles, the most notable of which is left bundle branch block (LAFB + LPFB). Trifascicular block denotes the presence of first degree AV block in the presence of a bifascicular block and is indicative of significant conduction system disease that includes the AV node, thus increasing the risk of Mobitz II or third-degree AV block and the potential need for permanent pacemaker placement.

NARROW COMPLEX TACHYCARDIAS

Sinus Tachycardia

Clinical Features

The ECG characteristics of sinus tachycardia are (*a*) normal sinus P waves and PR intervals and (*b*) an atrial rate usually between 100 and 160 beats/min. Sinus tachycardia is in response to one of three categories of stimuli: (*a*) physiologic (pain or exertion), (*b*) pharmacologic (sympathomimetics, caffeine, or bronchodilators), or (*c*) pathologic (fever, hypoxia, anemia, hypovolemia, pulmonary embolism, or hyperthyroidism). In many of these conditions, the increased heart rate is an effort to increase cardiac output to match increased circulatory needs.

Emergency Department Care and Disposition

Since sinus tachycardia is a compensatory rhythm, the focus should be on diagnosing and treating the underlying condition.

Atrial Flutter

Clinical Features

Atrial flutter is a rhythm that originates from a small area within the atria. ECG characteristics of atrial flutter are (*a*) a regular atrial rate between 250 and 350 beats/min; (*b*) "saw tooth" flutter waves directed superiorly and most visible in leads II, III, and aV_F; and (*c*) AV block, usually 2:1, but occasionally greater or irregular (Fig. 2-9). One-to-one conduction may occur if a bypass tract is present. Carotid sinus massage or Valsalva maneuvers are useful techniques to slow the ventricular response by increasing the degree of AV block, which can unmask flutter waves in uncertain cases.

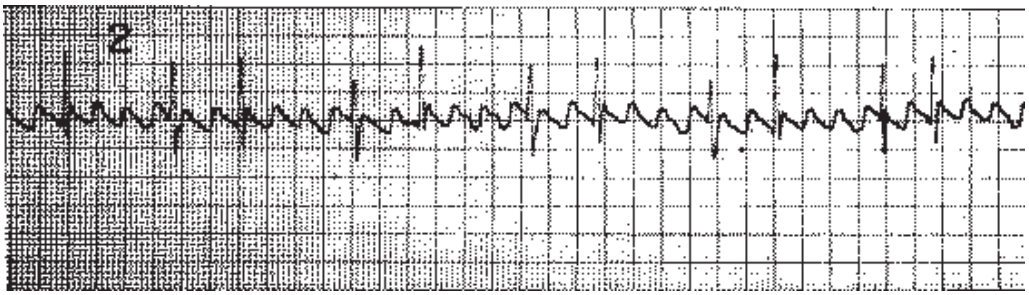


FIGURE 2-9. Atrial flutter.

Atrial flutter is seen most commonly in patients with ischemic heart disease as well as CHF, acute MI, pulmonary embolus, myocarditis, blunt chest trauma, and digoxin toxicity. Atrial flutter may be a transitional arrhythmia between sinus rhythm and atrial fibrillation. Consider anticoagulation in patients with an unclear time of onset or duration longer than 48 hours before conversion to sinus rhythm due to increased risk of atrial thrombus and embolization.

Emergency Department Care

The treatment is the same as atrial fibrillation and is discussed below.

Atrial Fibrillation

Clinical Features

Atrial fibrillation (Afib) occurs when there are multiple, small areas of atrial myocardium continuously discharging in a disorganized fashion. This results in loss of effective atrial contraction and decreases left ventricular end-diastolic volume, which may precipitate CHF in patients with impaired cardiac function. The ECG characteristics of Afib are (*a*) fibrillatory waves of atrial activity, best seen in leads V_1, V_2, V_3, and aV_F; and (*b*) an irregular ventricular response, usually between 170 and 180 beats/min in patients with a healthy AV node (Fig. 2-10).

Afib may be paroxysmal (lasting for less than 7 days), persistent (lasting for more than 7 days), or chronic (continuous). Afib can be idiopathic (lone Afib) or may be found in association with longstanding hypertension, ischemic heart disease, rheumatic heart disease, alcohol use ("holiday heart"), COPD, and thyrotoxicosis. Patients with LV dysfunction who depend on atrial contraction may suffer acute CHF with Afib onset. Rates of greater than 300 beats/min with a wide QRS complex are concerning for a preexcitation syndrome such as Wolff–Parkinson–White (WPW) (Fig. 2-11).

Patients with Afib who are not anticoagulated have a yearly embolic event rate as high as 5% and a lifetime risk greater than 25%. Conversion from atrial fibrillation of 12 hours duration or less to sinus rhythm carries a 0.3% risk of arterial embolism compared to a risk of 1% for durations of 12 to 48 hours. Patients with heart failure and diabetes mellitus are particularly at risk with embolic rates as high as 9.8%. Anticoagulation for 3 weeks is required before cardioversion in patients with atrial fibrillation for longer than 48 hours duration and in those patients with an uncertain time of onset who are not on anticoagulation therapy.

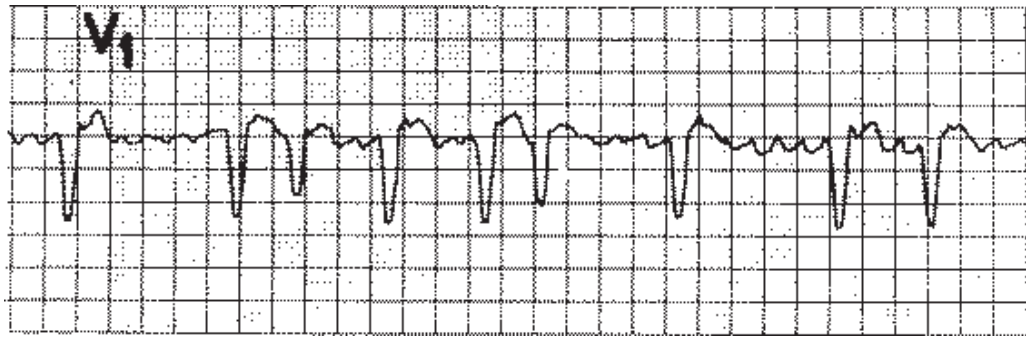


FIGURE 2-10. Atrial fibrillation.

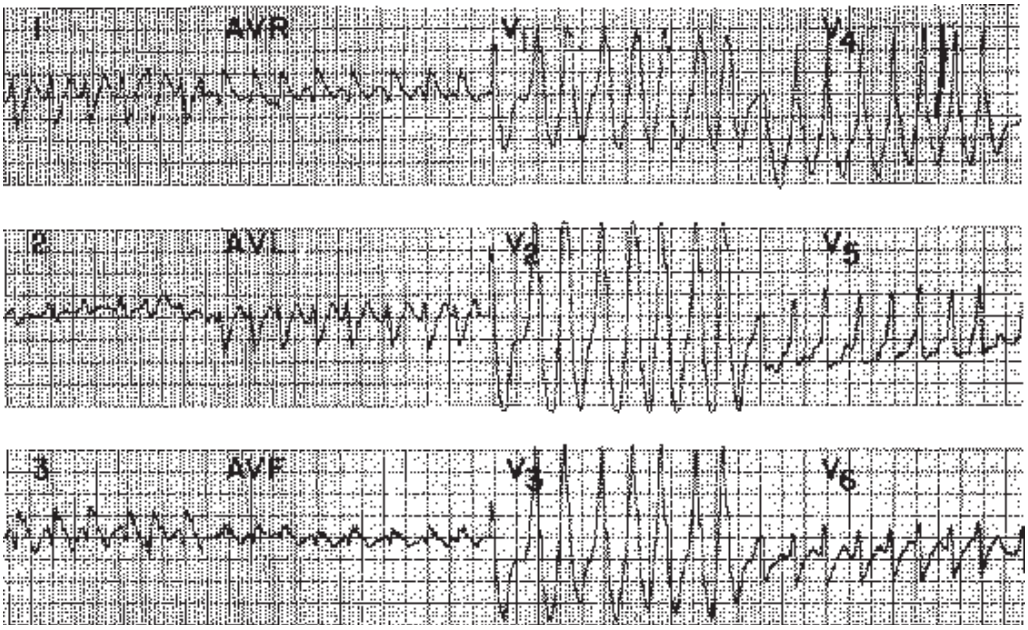


FIGURE 2-11. Atrial fibrillation in Wolff–Parkinson–White syndrome.

Emergency Department Care and Disposition

1. Treat unstable patients with synchronized cardioversion (150 to 200 J).
2. Stable patients with Afib for longer than 48 hours should be anticoagulated before cardioversion. Consider a transesophageal echocardiogram to rule out atrial thrombus before cardioversion.
3. Control rate with diltiazem. Administer 15 to 20 mg (or 0.25 mg/kg) IV over 2 minutes followed by a continuous IV infusion, 5 to 10 mg/h, to maintain rate control. Give a second dose of 25 mg (0.35 mg/kg) in 15 minutes if the first dose fails to control the rate. Alternative rate control agents for patients with normal cardiac function include **verapamil** 2.5 to 5 mg IV or **metoprolol** 5 to 10 mg IV. Treat patients with preexcitation syndromes (e.g., WPW) with **procainamide** 15 to 17 mg/kg IV over 30 minutes followed by an infusion of at 1 to 4 mg/min up to 50 mg/kg or until 50% QRS widening is noted. Avoid β-adrenergic or calcium channel blockers (i.e., verapamil) due to the risk of causing degeneration to VF.
4. In patients with impaired cardiac function (EF <40%), use **amiodarone** 5 mg/kg IV over 30 minutes, followed by 1200 mg over 24 hours (contraindicated in patients with iodine or shellfish allergy; increased risk of rhabdomyolysis if coadministered with simvastatin).
5. Patients with Afib with a clear duration less than 48 hours may be considered for chemical or electrical cardioversion in the emergency department. **Ibutilide** has the highest success rate and is dosed at 0.01 mg/kg IV up to 1 mg, infused over 10 minutes. Median time to conversion is 20 to 30 minutes. Ibutilide should not be administered to patients with known structural heart disease, hypokalemia, prolonged QTc intervals, hypomagnesemia, or CHF because of the possibility of provoking torsades de pointes. Monitor for 4 to 6 hours after giving ibutilide. Patients with impaired cardiac function may be cardioverted with amiodarone or electrically.

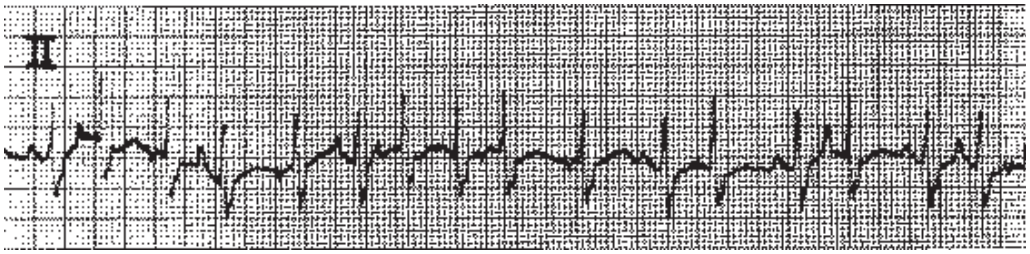


FIGURE 2-12. Multifocal atrial tachycardia (MFAT).

Multifocal Atrial Tachycardia

Clinical Features

Multifocal atrial tachycardia (MAT) is defined as at least three different sites of atrial ectopy. The ECG characteristics of MAT are (*a*) three or more differently shaped P waves; (*b*) changing PP, PR, and RR intervals; and (*c*) atrial rhythm usually between 100 and 180 beats/min (Fig. 2-12). Because the rhythm is irregularly irregular, MAT can be confused with atrial flutter or atrial fibrillation (AFib). MAT is found most often in elderly patients with decompensated COPD, but it also may be found in patients with CHF, sepsis, methylxanthine toxicity, or digoxin toxicity.

Emergency Department Care and Disposition

1. Treat the underlying disorder.
2. Specific antiarrhythmic treatment is not indicated.
3. **Magnesium sulfate** 2 g IV over 60 seconds followed by a constant infusion of 1 to 2 g/h may decrease ectopy and convert MAT to sinus rhythm in some patients.
4. Replete potassium levels to greater than 4 mEq/L to increase myocardial membrane stability.

Paroxysmal Supraventricular Tachycardia

Clinical Features

Supraventricular tachycardia (SVT) is a regular, rapid rhythm that arises from impulse reentry or an ectopic pacemaker above the bifurcation of the His bundle. The reentrant variety is the most common (Fig. 2-13). Patients often present with acute, symptomatic episodes termed paroxysmal supraventricular tachycardia (PSVT). Atrioventricular nodal reentrant tachycardia (AVnRT) can occur in a normal heart or in association with rheumatic heart disease, acute pericarditis, MI, mitral valve prolapse, or preexcitation syndromes. In patients with atrioventricular bypass tracts (AVRT), reentry can occur in either direction, usually (80% to 90% of patients) in a direction that goes down the AV node and up the bypass tract producing a narrow QRS complex (orthodromic conduction). In the remaining 10% to 20% of patients, reentry occurs in the reverse direction (antidromic conduction). Ectopic SVT usually originates in the atria, with an atrial rate of 100 to 250 beats/min and may be seen in patients with acute MI, chronic lung disease, pneumonia, alcohol intoxication, or digoxin toxicity. There is a high incidence of tachyarrhythmias in patients with preexcitation syndromes including PSVT (40% to 80%), atrial fibrillation (10% to 20%), and atrial flutter (about 5%).

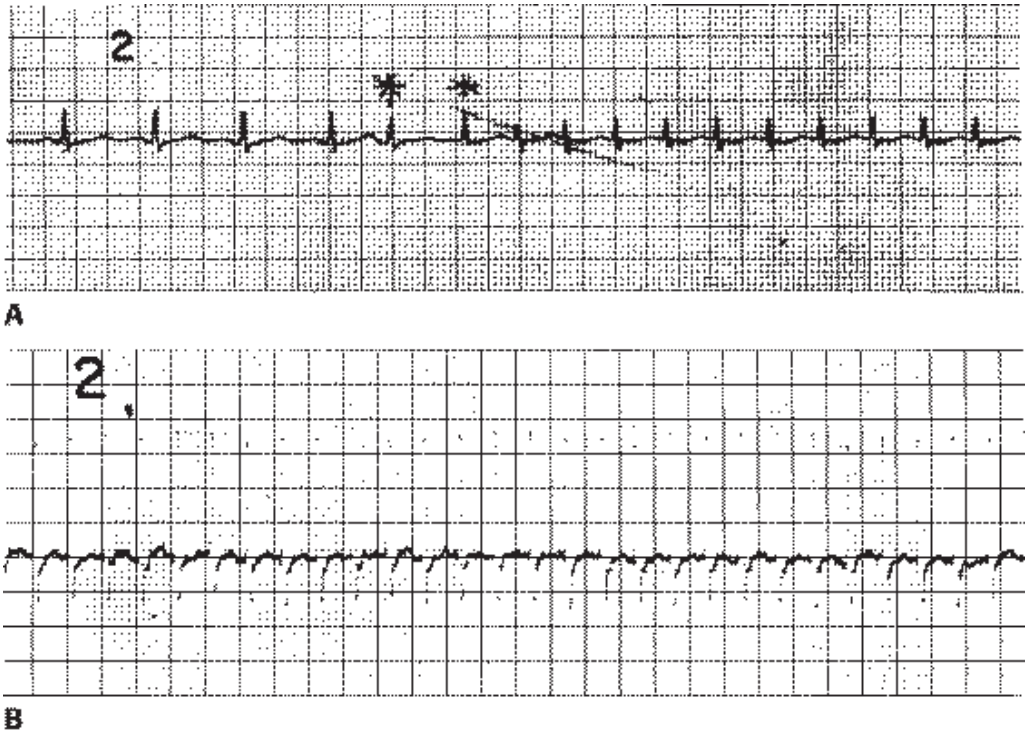


FIGURE 2-13. Reentrant supraventricular tachycardia (SVT). **A.** Second asterisk (*) initiates run of PAT. **B.** SVT, rate 286.

Emergency Department Care and Disposition

1. Perform synchronized cardioversion in any unstable patient (e.g., hypotension, pulmonary edema, or severe chest pain).
2. In stable patients, the first intervention should be vagal maneuvers, including:
   a. Valsalva maneuver: While in the supine position, ask the patient to strain for at least 10 seconds. The legs may be lifted to increase venous return and augment the reflex.
   b. Diving reflex: Have the patient immerse the face in cold water or apply a bag of ice water to the face for 6 to 7 seconds. This maneuver is particularly effective in infants.
   c. Carotid sinus massage: Auscultate to ensure that there is no carotid bruit and massage the carotid sinus against the transverse process of C6 for 10 seconds at a time, first on the side of the nondominant cerebral hemisphere. This should never be done simultaneously on both sides.
3. Administer **adenosine**, 6 mg rapid IV bolus, into a large vein followed by a 20-mL normal saline rapid flush. If there is no effect within 2 minutes, give a second dose of 12 mg IV. Most patients experience distressing chest pain, flushing, or anxiety lasting less than 1 minute. Ten percent of patients may experience transient atrial fibrillation or flutter after conversion. This is first-line treatment for WPW-associated SVT with a narrow QRS complex (orthodromic conduction) but is ineffective in cases of anterograde conduction over an accessory pathway. Adenosine may induce bronchospasm in asthmatics requiring treatment with bronchodilators.
4. Patients with wide complex SVT (antidromic conduction across accessory pathway) should be approached as presumed ventricular tachycardia

(VT; see Ventricular Tachycardia) unless there is a known history of WPW syndrome. Patients with this type of tachycardia are at risk for rapid ventricular rates and degeneration into VF; therefore, agents that preferentially block the AV node such as β-blockers, calcium channel blockers, and digoxin should not be used. Treat stable patients with **procainamide**, 15 to 17 mg/kg IV over 30 minutes up to 50 mg/kg, or until 50% QRS widening is noted (contraindicated in patients with myasthenia gravis since it may increase weakness).

WIDE COMPLEX TACHYCARDIAS

Ventricular Tachycardia

Clinical Features

VT is the occurrence of three or more successive beats from a ventricular ectopic pacemaker at a rate faster than 100 beats/min. The ECG characteristics of VT are (*a*) a wide QRS complex, (*b*) a rate faster than 100 beats/min (most commonly 150 to 200 beats/min), (*c*) a regular rhythm, although there may be some initial beat-to-beat variation, and (*d*) a constant QRS axis (Fig. 2-14). The most common causes of VT are ischemic heart disease and acute MI, accounting for approximately 50% of all cases of symptomatic VT.

Other etiologies include hypertrophic cardiomyopathy, mitral valve prolapse, drug toxicity (digoxin, antiarrhythmics, or sympathomimetics), hypoxia, hypokalemia, and hyperkalemia. In general, all wide complex tachycardia should be treated as VT regardless of clinical symptoms or initial vital signs. Adenosine appears to cause little harm in patients with VT; therefore, stable patients with wide complex tachycardia due to suspected SVT with aberrancy (see previous section) may be treated safely with adenosine when the diagnosis is in doubt. Atypical VT (torsade de pointes, or twisting of the points) occurs when the QRS axis swings from a positive to a negative direction in a single lead at a rate of 200 to 240 beats/min (Fig. 2-15). Drugs that further prolong repolarization—quinidine, disopyramide, procainamide, phenothiazines, and tricyclic antidepressants—exacerbate this arrhythmia.

Emergency Department Care and Disposition

1. Defibrillate pulseless VT with unsynchronized cardioversion starting at 150 to 200 J. Treat unstable patients who are not pulseless with synchronized cardioversion.
2. Treat hemodynamically stable patients with **amiodarone** 150 mg IV over 10 minutes with repeated boluses every 10 minutes up to a total of 2 g. Alternatively, an infusion of 0.5 mg/min over 18 hours may be given

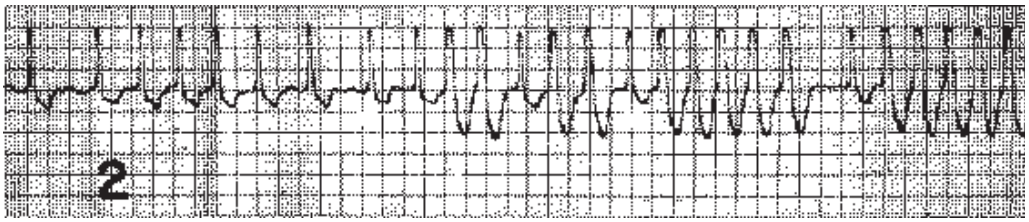


FIGURE 2-14. Ventricular tachycardia.

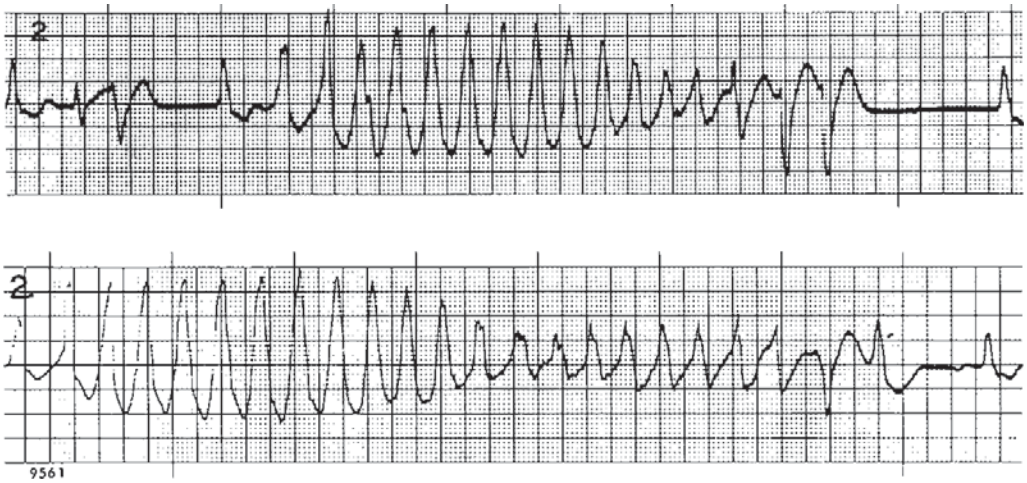


FIGURE 2-15. Two examples of short runs of atypical ventricular tachycardia showing sinusoidal variation in amplitude and direction of the QRS complexes: "Le torsade de pointes" (twisting of the points). Note that the top example is initiated by a late-occurring PVC (lead II).

after the initial bolus. Second-line agents include procainamide (in patients without suspected MI or LV dysfunction) and lidocaine.

3. For patients with torsades de pointes: Try overdrive pacing set at 90 to 120 beats/min to terminate torsades de pointes.
4. **Magnesium sulfate** 1 to 2 g IV over 60 to 90 seconds followed by an infusion of 1 to 2 g/h can be effective.
5. **Isoproterenol**, 2 to 10 μg/min IV infusion, is also used in refractory torsades but carries a risk of increased myocardial oxygen demand.

Undifferentiated Wide Complex Tachycardia

Patients with wide complex tachycardia should be approached as having VT until proven otherwise. Age over 35 years, a history of MI, CHF, or coronary artery bypass grafting strongly favor VT. ECG signs favoring VT include AV dissociation, fusion beats, precordial lead QRS concordance, and a QRS duration longer than 0.14 second. It is a misconception that patients with VT are typically unstable. At the bedside, one should assume any new and symptomatic wide complex tachycardia is ventricular in origin and focus on treating the rhythm as VT, as well as any contributing cause, especially in the unstable patient.

Ventricular Fibrillation

Clinical Features

VF is the totally disorganized depolarization and contraction of small areas of ventricular myocardium during which there is no effective ventricular pumping activity. The ECG shows a fine-to-coarse zigzag pattern without discernible P waves or QRS complexes (Fig. 2-16). VF is seen most commonly in patients with severe ischemic heart disease, with or without an acute MI. It also can be caused by digoxin or quinidine toxicity, hypothermia, chest trauma, hypokalemia, hyperkalemia, or mechanical stimulation (e.g., catheter wire). Primary VF occurs suddenly, without preceding hemodynamic deterioration, and usually is due to acute ischemia or peri-infarct

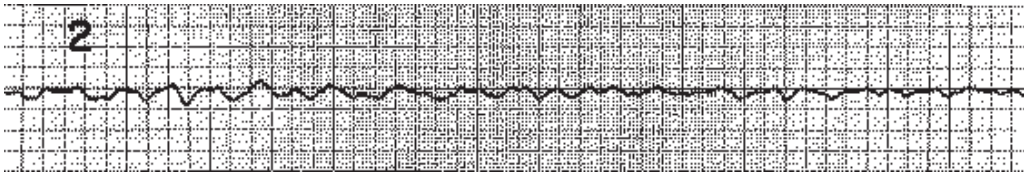


FIGURE 2-16. Ventricular fibrillation.

scar reentry. Secondary VF occurs after a prolonged period of hemodynamic deterioration due to left ventricular failure or circulatory shock.

Emergency Department Care and Disposition

1. Perform immediate electrical defibrillation (unsynchronized) at 200 J (biphasic) and 360 J (monophasic) along with immediate vigorous chest compressions to augment coronary perfusion. Keep defibrillation pads on the patient and in the same location because, with successive countershocks, transthoracic impedance decreases.
2. If the initial two cycles of cardiopulmonary resuscitation (CPR) and defibrillation are unsuccessful, administer antiarrhythmic treatment using **amiodarone** 300 mg IV push. **Lidocaine** is second-line and is dosed at 1.5 mg/kg IV followed by 0.75 mg/kg IV for two more doses. Repeat the CPR-defibrillation cycle.
3. If no pulse is present after the third CPR-defibrillation cycle, give **epinephrine** 1 mg IV push, or **vasopressin** 40 units IV push (one time only), followed by a 20-mL normal saline flush and immediate resumption of the CPR-defibrillation cycle.
4. In refractory VF, administer **magnesium sulfate** 1 to 2 g IV over 60 to 90 seconds followed by an infusion of 1 to 2 g/h.

DYSRHYTHMIA-ASSOCIATED CONDUCTION ABNORMALITIES

Wolff–Parkinson–White (WPW) Syndrome

WPW syndrome is the most common form of ventricular preexcitation involving an accessory conduction pathway that bypasses the AV node (Fig. 2-17). The ventricles are activated by an impulse from the atria sooner than would be expected if the impulse were transmitted down the normal conducting pathway. This premature activation causes initial fusion beat morphology with slurring of initial QRS complex, causing the pathognomonic delta wave. Among patients with WPW–PSVT, 80% to 90% will conduct in the orthodromic direction and the remaining 10% to 20% will conduct in the antidromic direction. ECG findings of atrial fibrillation or flutter with antidromic conduction down the bypass tract show a wide QRS complex that is irregular with a rate faster than 180 to 200 beats/min (see Atrial Fibrillation).

Emergency Department Care and Disposition

1. Treatment of the tachydysrhythmia should be based on the QRS duration and regularity of the rhythm. All unstable patient should be cardioverted (synchronized) at 150 to 200 J.

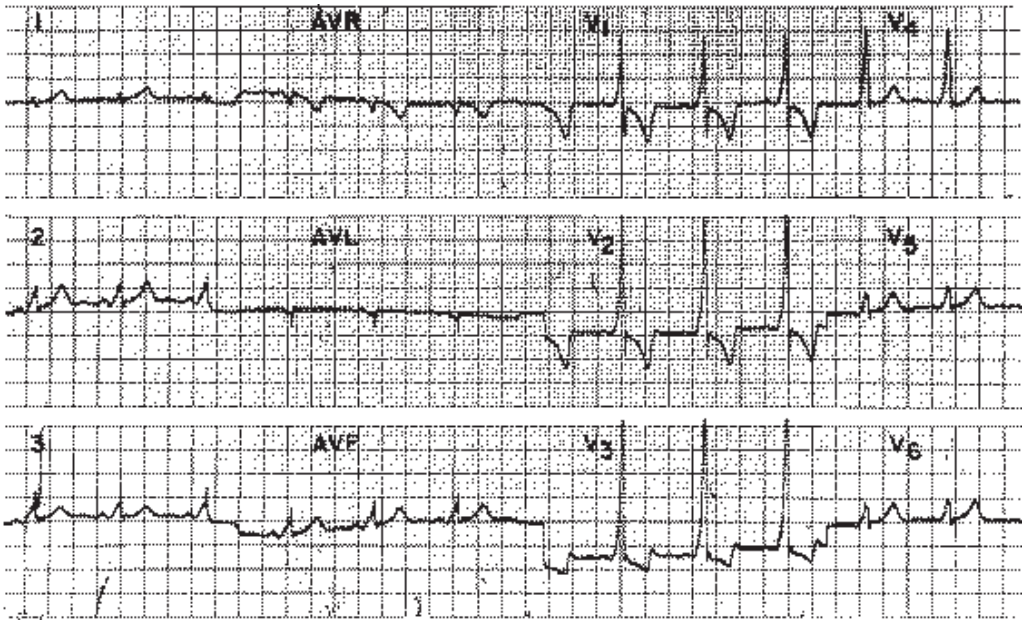


FIGURE 2-17. Type A Wolff–Parkinson–White syndrome.

2. Patients with a narrow regular tachycardia should be treated as SVT (orthodromic conduction) using vagal maneuvers followed by adenosine, if ineffective. Refractory cases may require **procainamide**, 15 to 17 mg/kg IV over 30 minutes up to 50 mg/kg, or until 50% QRS widening is noted.
3. All patients with wide QRS tachycardias, whether regular (SVT with antidromic conduction) or irregular (atrial fibrillation with antidromic conduction), should be treated with procainamide.
4. Nodal blocking agents such as β-blockers and calcium channel blockers should be avoided due to the risk of enhancing conduction across the bypass tract.
5. Patients with stable recurrent episodes may be monitored after cardioversion and discharged to outpatient follow-up. Instability, loss of consciousness, or other concerning features warrant observation on monitoring for recurrence. Asymptomatic patients with WPW, found incidentally, warrant outpatient referral to a cardiologist for further evaluation.

Brugada Syndrome and Long-QT Syndrome

Brugada syndrome and long-QT syndrome increase the risk of spontaneous VT/VF and require evaluation for implantable cardiac defibrillator placement when diagnosed. Brugada syndrome is a genetic disorder of fast sodium channels causing an RBBB pattern in the early precordial leads (V1–2) with a pathognomonic J-point elevation and saddle-shaped or sloped ST segment (Fig. 2-18). Long-QT syndrome is characterized by a QT interval greater than 470 milliseconds in men and greater than 480 milliseconds in women and may be congenital or acquired, leading to an increased risk of torsades de pointes. The risk of arrhythmia increases significantly with QTc durations greater than 500 milliseconds.

Emergency Department Care and Disposition

1. Recognition of the ECG pattern should prompt close outpatient follow-up with a cardiologist, especially in stable symptomatic patients.

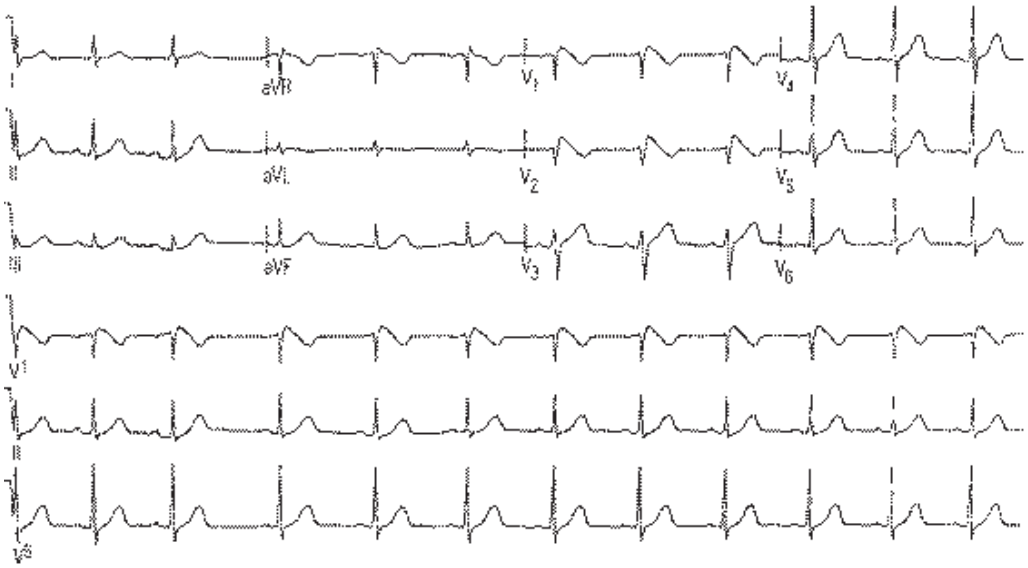


FIGURE 2-18. Brugada syndrome.

2. Patients presently with unstable rhythms or concerning clinical presentations (e.g., drop attacks) should be admitted for monitoring and cardiology consultation.
3. Patients should be advised to avoid any medications that may potentially worsen QT prolongation (http://www.brugadadrugs.org). Patients with long-QT syndrome should be advised to avoid strenuous exercise, particularly swimming, which can increase the chance of sudden death.

PRETERMINAL RHYTHMS

Pulseless Electrical Activity

Pulseless electrical activity is the presence of electrical complexes without accompanying mechanical contraction of the heart. Potential mechanical causes should be diagnosed and treated, including severe hypovolemia, cardiac tamponade, tension pneumothorax, massive pulmonary embolus, MI, and toxic ingestions (e.g., tricyclic antidepressants, calcium channel blockers, β-blockers). In addition, profound metabolic abnormalities such as acidosis, hypoxia, hypokalemia, hyperkalemia, and hypothermia also should be considered and treated.

After intubation and initiating CPR, administer **epinephrine** 1 mg IV/IO (1:10,000 solution) every 3 to 5 minutes. If giving via endotracheal tube, increase the dose 2 to 2.5 times and follow with several rapid ventilations to disperse the drug. Treatment is guided by rapid identification and treatment of the underlying cause. Use agents with α-adrenergic activity, such as norepinephrine and phenylephrine, to improve vascular tone when indicated. Electrical pacing is not effective.

Asystole (Cardiac Standstill)

Asystole is the complete absence of cardiac electrical activity and carries a grim prognosis. Treatment is the same as that for pulseless electrical activity.

CARDIAC PACEMAKERS AND AUTOMATED INTERNAL CARDIAC DEFIBRILLATORS (AICDS)

Pacemakers, AICDs, or combination units may be used in patients with a history of sudden death, heart failure, or cardiomyopathy. Malfunction can occur at any level of the device, including infection or hematoma in the pocket housing the device, lead infection/displacement, failure to pace, failure to sense, overpacing, or inappropriate defibrillation. Most pacemakers will have a magnetic switch which, when triggered by magnet application to the unit, will cause the pacemaker to function in a fixed asynchronous mode.

Emergency Department Care and Disposition

1. Evaluation should include an ECG, electrolytes, and chest x-ray to assess lead position and integrity. Arrangements should be made for electrical interrogation of the unit.
2. Patients with pacing failure may require treatment based on their underlying rhythm and associated symptoms.
3. Patients with overpacing may require magnet application to convert the pacemaker to asynchronous mode pacing at a lower rate.

FURTHER READING

For further reading in *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 8th ed., see Chapter 18, "Cardiac Rhythm Disturbances," by William J. Brady, Thomas S. Laughrey, and Chris A. Ghaemmaghami; Chapter 19, "Pharmacology of Antiarrhythmics and Antihypertensives" by Sara Shields, Rachel M. Holland, R. Dustin Pippin, and Benjamin Small; Chapter 20, "Pharmacology of Vasopressors and Inotropes" by Sara Shields and Rachel M. Holland.

CHAPTER 3

Resuscitation of Children and Neonates

Marc F. Collin

Children primarily develop cardiac arrest secondary to hypoxia from respiratory arrest or shock syndromes. Because of age and size differences among children, equipment sizes also differ (Table 3-1).

PEDIATRIC CARDIOPULMONARY RESUSCITATION

Securing the Airway

The airway in infants and children is smaller, variable in size, and higher and more anterior than that in the adult. The prominent occiput and relatively large tongue and epiglottis may lead to obstruction when the child is in the supine position.

Mild extension of the neck in the sniffing position opens the airway. This may be maintained by placing a towel beneath the shoulders. Chin lift or jaw thrust maneuvers may relieve obstruction of the airway related to the tongue. Oral airways are not commonly used in pediatrics but may be useful in the unconscious child who requires continuous jaw thrust or chin lift to maintain airway patency. Oral airways are inserted by direct visualization with a tongue blade.

A bag-valve-mask system is commonly used for ventilation. Minimum volume for ventilation bags for infants and children is 450 mL. The tidal volume necessary to ventilate children is 8 to 10 mL/kg. Observation of chest rise and auscultation of breath sounds will ensure adequate ventilation.

Endotracheal intubation usually is performed with a Miller straight blade with a properly sized tube. Resuscitation measuring tapes have been found to be the most accurate for determining tube size. The formula 16 plus age in years divided by 4 calculates approximate tube size. Uncuffed tubes are commonly used in children up to 8 years, but cuffed tubes can be used in younger children as well.

Respiratory rates should be started at 20 breaths/min for infants beyond the neonatal period, 15 breaths/min for young children, and 10 breaths/min for adolescents unless hyperventilation is required.

Rapid Sequence Intubation

Rapid sequence intubation is the administration of an intravenous (IV) anesthetic with a neuromuscular blocking agent to facilitate endotracheal intubation, and is associated with the highest success and lowest complication rates, compared to other methods. See Table 3-2 for common medications for rapid sequence intubation.

1. All equipment and supplies must be prepared. A well-functioning IV line must be in place. A cardiac monitor and oximetry should be used, and if available, noninvasive capnometry as well. The laryngoscope light source must be checked. Suction equipment should be turned on and immediately available.

TABLE 3-1 Length-Based Equipment Chart (Length = Centimeters*)

Item	54–70	70–85	85–95	95–107	107–124	124–138	138–155
Endotracheal tube size (mm)	3.5	4.0	4.5	5.0	5.5	6.0	6.5
Lip–tip length (mm)	10.5	12.0	13.5	15.0	16.5	18.0	19.5
Laryngoscope	1 straight	1 straight	2 straight	2 straight or curved	2 straight or curved	2–3 straight or curved	3 straight or curved
Suction catheter	8F	8F–10F	10F	10F	10F	10F	12F
Stylet	6F	6F	6F	6F	14F	14F	14F
Oral airway	Infant/small child	Small child	Child	Child	Child/small adult	Child/adult	Medium adult
Bag-valve mask	nfant	Child	Child	Child	Child	Child/adult	Adult
Oxygen mask	Newborn	Pediatric	Pediatric	Pediatric	Pediatric	Adult	Adult
Vascular access (gauge)							
Catheter	22–24	20–22	18–22	18–22	18–20	18–20	16–20
Butterfly	23–25	23–25	21–23	21–23	21–23	21–22	18–21
Nasogastric tube	5F–8F	8F–10F	10F	10F–12F	12F–14F	14F–18F	18F
Urinary catheter	5F–8F	8F–10F	10F	10F–12F	10F–12F	12F	12F
Chest tube	10F–12F	16F–20F	20F–24F	20F–24F	24F–32F	28F–32F	32F–40F
Blood pressure cuff	Newborn/infant	Infant/child	Child	Child	Child	Child/adult	Adult

Directions for use: (1) measure patient length with centimeter tape; (2) using measured length in centimeters, access appropriate equipment column.