



2009 UCSD Medical Center

Advanced Resuscitation Training Manual

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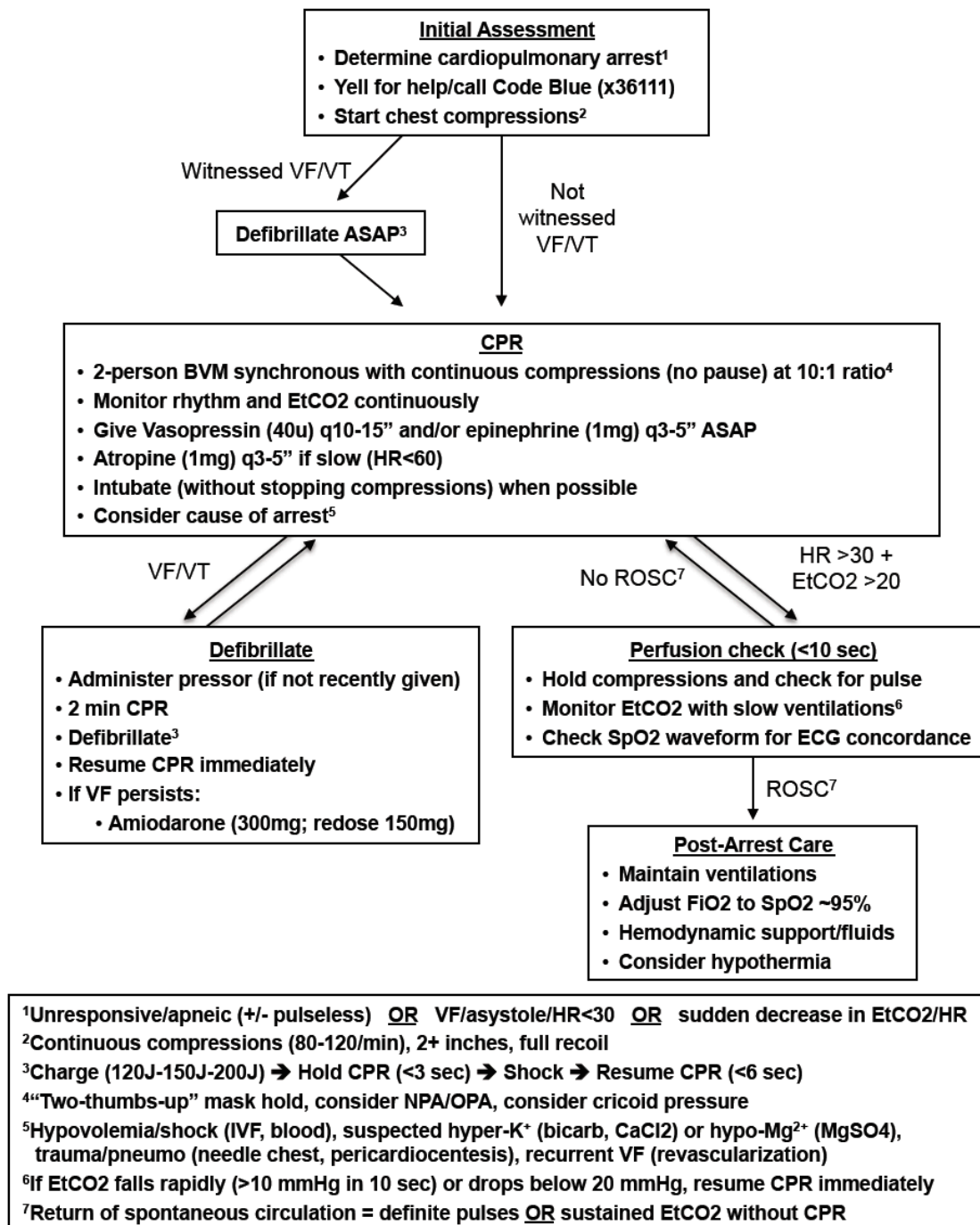
Overview

Resuscitation science is undergoing a renaissance, with a rapid expansion of our understanding of the physiology of resuscitation and a dramatic increase in outcomes-based studies to guide therapeutic interventions. Hospital-based resuscitation has unique features related to the etiologies of arrest, the spectrum of deterioration, and the resources and personnel available to respond. Our discussion here will be structured around the initial assessment and optimal treatment of the cardiopulmonary arrest patient, post-arrest care, and evaluation and treatment of the perfusing patient including rapid response team concepts.

Core Concepts

- I. Resuscitation is rare and stressful event, even for healthcare providers, underscoring the importance of simplicity, consistency, and role-specific training.
- II. There has been an exponential increase in our knowledge regarding the resuscitation physiology and optimal therapy, requiring more frequent updates and a flexible curriculum.
- III. Resuscitation requires increasingly complex teamwork and choreography, which mandates regular simulation-based training.
- IV. Hospital resuscitation is different than prehospital resuscitation, with most arrests representing the final manifestation of hypoxemia and hypotension. This requires a different approach to resuscitation and affords the opportunity to intervene during the pre-arrest period.
- V. Continuous quality improvement efforts and performance evaluations should be closely integrated with resuscitation training.

2009 UCSD CARDIOPULMONARY ARREST RESUSCITATION



Initial Assessment

The goal in the initial assessment of the potential arrest victim is to rapidly identify cardiopulmonary arrest, initiate compressions, and call for help. The secondary goals include evaluation for possible defibrillation, initiation of ventilations, and administration of pressor agents.

Responsiveness

In most cases, the arrest victim will be identified by a lack of responsiveness. Use of verbal and tactile stimuli to determine responsiveness should be immediately employed. A victim in cardiopulmonary arrest will be completely unresponsive. Clearly, this approach would not be appropriate for a paralyzed, intubated patient.

Spontaneous ventilation

The cessation of spontaneous breathing is one of the first manifestations of loss of cerebral perfusion. Thus, the lack of spontaneous breaths can be used as an indicator of cardiopulmonary arrest. The “gulping” or “gaspings” respirations in the immediate post-arrest period do not count as spontaneous breathing. Again, this approach cannot be used for a patient undergoing mechanical ventilation or following administration of paralytics.

Pulse check

The absence of a palpable pulse has long been considered the gold standard for determining cardiopulmonary arrest. However, even the most experienced providers routinely err in making this determination. Thus, unresponsiveness and the absence of spontaneous breathing can be considered adequate to initiate chest compressions. It is worth noting that spontaneous breathing may not return immediately upon successful resuscitation, making palpation of a pulse important in determining perfusion status after resuscitation efforts have been initiated. In addition, palpation of pulses may be necessary with mechanical ventilation, since responsiveness and the presence of spontaneous breathing may be difficult to assess in these patients.

Other

Several additional sources of data may be used to help determine cardiopulmonary arrest. A monitor showing asystole, ventricular fibrillation, or a slow ventricular rhythm (<30 beats/min) can be assumed to represent cardiopulmonary arrest. A sudden decrease in end-tidal CO₂ to values <10 mmHg will accompany arrest but must be differentiated from a dislodged endotracheal tube. The pulse oximetry or arterial line waveform will also disappear with cardiopulmonary arrest. A sudden drop in heart rate is the final event during arrest from hypoxemia/hypotension.

Chest Compressions

Chest compressions have become the foundation of resuscitation from cardiopulmonary arrest, regardless of etiology. The primary focus of the resuscitation should be the performance of continuous quality compressions.

Continuous compressions

The goal of chest compressions is to raise aortic pressure and improve perfusion. Unfortunately, it takes some time to reach a plateau pressure, even with good compressions, while interrupting compressions leads to an immediate drop in pressure. Thus, chest compressions should be performed continuously throughout the resuscitation, without pause for ventilation, rhythm analysis, intubation, or vascular access. In addition, compressions should be maintained during defibrillation charge and immediately following each shock.

Compression depth

Deeper compressions with full recoil produce better perfusion. This may limit the rate of compressions for larger patients. The compressor should be directly above the patient, with hands on the lower sternum, for maximum depth.

Compression rate

Compressions should be performed at the fastest rate possible but still allowing maximum depth and recoil, generally between 80/min (larger patients) and 120/min (smaller patients).

Switching compressors

Chest compressions produce significant fatigue when performed properly. Each individual should perform compressions no longer than 2 minutes at a time and may be relieved sooner if compression depth/recoil deteriorate or rate slows. Switching compressors should occur quickly with minimal pause in compressions. In addition, compressors should not be switched at the time of defibrillation to ensure immediate resumption of compressions following each shock.

Defibrillation

Ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) require defibrillation. The importance of compressions in defibrillation must be appreciated.

Compressions and defibrillation

The fibrillating heart must be “primed” with chest compressions prior to defibrillation once arrest times exceed 4-5 minutes. This priming effect decays very quickly (<3 sec), requiring compressions to be performed until the moment of defibrillation. In addition, the heart requires perfusion with compressions immediately (<6 sec) following defibrillation to encourage a viable rhythm. Finally, several minutes of chest compressions (with early administration of a pressor agent) are the most effective “anti-arrhythmic” for a persistent/recurrent VF/VT.

Witnessed arrest

Rapid defibrillation is the top priority for a “witnessed” (including monitored) VF/VT arrest. The closest provider should initiate compressions while yelling for the defibrillator. This provides rapid defibrillation while maintaining a “primed” heart.

Unwitnessed arrest

Once an arrest exceeds 4-5 minutes (common in non-monitored settings) the fibrillating heart must be “primed” before a shock. Compressions should be initiated once cardiopulmonary arrest is confirmed. A “code blue” should be activated, which should result in delivery of a defibrillator. A shock may be delivered immediately if compressions have been performed for several minutes. Otherwise, at least 2-3 minutes of compressions should be performed prior to the first defibrillation attempt.

Energy settings

Biphasic defibrillators are effective at terminating VF/VT at lower energy settings than historical levels. The current recommendations for Zoll defibrillators are 120J for the 1st shock, 150J for the 2nd shock, and 200J for all subsequent shocks. For VF, all defibrillation attempts should be unsynchronized. If perfusion status is uncertain with VF, then Sync Mode can be selected with an initial energy of 120J.

Secondary ventricular fibrillation

When VF appears as a secondary rhythm (i.e., in a patient with an initial arrest rhythm that was not shockable), immediate defibrillation carries a very poor prognosis. Thus, 2 minutes of CPR – ideally with early administration of a pressor agent – should be performed prior to defibrillation with appearance of VF.

Anti-arrhythmics

The preferred agent for persistent or recurrent ventricular fibrillation is amiodarone at 300 mg IVP. Unfortunately, profound hypotension is one of the side effects of the current formulation. Thus, amiodarone should be reserved for use after 2-3 unsuccessful defibrillation attempts. Chest compressions with early administration of a pressor agent are the most effective “anti-arrhythmic” therapy.

Ventilation

The role of ventilation in resuscitation from cardiopulmonary arrest has been de-emphasized due to the relatively lower oxygen requirements in an arrest state and the potential for positive-pressure ventilation to impede cardiac output. The initial rescuer should perform compressions alone, with ventilations initiated by subsequent responders once a bag-valve-mask becomes available.

Compression-to-ventilation ratio

The consequence of stopping chest compressions to provide ventilations appears to be too great to justify “interrupted” CPR. Instead, chest compressions should be continuous, with interposed ventilations delivered every 10th compression.

Bag-valve-mask ventilation

The initial approach to ventilation should include the use of a bag-valve-mask by two rescuers. The first rescuer holds the mask tightly to the patient’s face using the “two thumbs up” approach, with head tilt and jaw thrust. The second rescuer squeezes the bag to deliver interposed ventilations, 1 every 10th compression. There should be no pause in compressions to deliver ventilations. Nasopharyngeal and oropharyngeal airways should be used whenever possible to maintain airway patency. Cricoid pressure can be applied by a third rescuer to minimize gastric insufflation.

Post-intubation ventilation

Once endotracheal tube confirmation has been performed, the ventilator continues to squeeze the bag to deliver 1 interposed ventilation every 10th compression. There should be no pause in compressions to deliver ventilations.

End-tidal CO₂

The end-tidal CO₂ sensor should be placed as soon as possible, between the mask and bag or between the endotracheal tube and bag. End-tidal CO₂ can be used to confirm tube placement, monitor cardiac output during resuscitation, help determine perfusion status, and guide ventilation following return of spontaneous circulation.

All arrest patients

General approach

There are five main considerations for cardiopulmonary arrest that should be reviewed periodically and with any change in the patient's status:

1. Is the patient in cardiopulmonary arrest?
2. If so, are adequate compressions being performed?
3. Is the rhythm shockable?
4. Has a pressor agent (vasopressin or epinephrine) been administered?
5. Are adequate ventilations being performed?

Drug administration

Administration of a pressor (vasopressin and/or epinephrine) should be performed as quickly as possible following initiation of chest compressions for patients in cardiopulmonary arrest. Vasopressin 40 units IVP can be given every 10-15 minutes; epinephrine 1 mg can be given IVP every 3-5 minutes. If rhythm analysis reveals a slow rate (<60 beats per minute) atropine 1 mg IVP may be administered every 3-5 minutes.

Causes of cardiopulmonary arrest

The vast majority of inpatient cardiopulmonary arrest comes as the end result of hypoxemia or hypotension, which are addressed through high quality compressions, early pressor administration, and optimal ventilation. In addition, the underlying etiology of arrest should be considered and attempts made to reverse these whenever possible.

- Hypovolemic/shock – IV fluid bolus and/or blood product administration
- Suspected hyperkalemia (patient with renal disease or receiving potassium/digoxin/spironolactone) – sodium bicarbonate and calcium chloride administration
- Suspected hypomagnesemia (torsade de pointes or malnourishment) – magnesium sulfate
- Suspected pneumothorax (trauma or COPD patient) – needle decompression or chest tube insertion
- Coronary thrombosis (recurrent VF/VT) – expedited revascularization

Pacing

We no longer pace patients in cardiopulmonary arrest due to the interruption in compressions and low likelihood of achieving adequate perfusion.

Assessing for perfusion

The general approach to resuscitation from cardiopulmonary arrest should emphasize continuous chest compressions, with minimal interruptions. Determining return of spontaneous circulation (ROSC) following resuscitation attempts is problematic, as extended periods of time may be spent (inappropriately) determining whether a pulse is present. If ROSC is unlikely, then compressions should be continued without pausing. If ROSC is suspected, then rapid (<10 seconds) confirmation is critical so that compressions can be resumed as soon as possible in the absence of definite evidence of perfusion.

Suspicion of ROSC

Continuous chest compressions should be maintained until ROSC is suspected. This should include the presence of organized complexes at an appropriate rate (>30/min) with a rise in end-tidal CO₂, with values <20 mmHg unlikely to be associated with ROSC. Conversely, end-tidal CO₂ values >30 mmHg should accompany ROSC, although high-quality CPR can also achieve such values in the absence of spontaneous perfusion. Use the filtered ECG waveform on the Zoll defibrillators to see the underlying rhythm while compressions are maintained. If ROSC is doubtful due to a non-perfusing rhythm (VF/VT, HR<30/min) and/or a low end-tidal CO₂ value (<20 mmHg), compressions should be continued without pausing.

Perfusion check

If ROSC is suspected (organized complexes with a rate >30/min and an increase in end-tidal CO₂ >20 mmHg), a brief pause in compressions can be performed. It is critical to perform this assessment quickly, as organized complexes suggest a heart that is attempting to reestablish spontaneous perfusion, and cessation of compressions at this critical juncture may be harmful. Pulse checkers should be positioned (carotid or femoral arteries), and the compressor should be instructed to hold compressions. Palpable pulses should be correlated with organized complexes to assure accuracy. Slow ventilations should continue. If end-tidal CO₂ values plummet (>10 mmHg in <10 sec) without compressions, arrest can be assumed and chest compressions restarted immediately. A third strategy to confirm the presence of a pulse is the use of a pulse oximetry or arterial line waveform. If this waveform correlates with organized complexes, the presence of a pulse can be inferred. Ideally, all three strategies are in agreement, and compressions should be restarted with any doubt.

Futility

There are no absolute rules when determining that it is appropriate to stop resuscitative efforts. Once a “Do Not Attempt Resuscitation” order can be confirmed or an appropriate surrogate decision maker makes this request, it is appropriate to stop. Persistent asystole despite “appropriate” efforts – traditionally three rounds of drugs – has been a traditional marker for futility. However, recent data suggest that patients in cardiopulmonary arrest for up to 60 minutes are being successfully resuscitated with neurologically intact survival at increasing rates. Thus, absolute rules for futility do not current exist. The underlying medical condition may give some indication of the likelihood of meaningful survival. End-tidal CO₂ may be a useful adjunct, as values remaining <12 mmHg despite good CPR suggest non-viability. It is worthwhile to achieve consensus among the team and to notify the family when they are present before efforts are withdrawn.

Post-arrest care

The two goals of post-arrest care include preventing re-arrest and mitigating reperfusion injury.

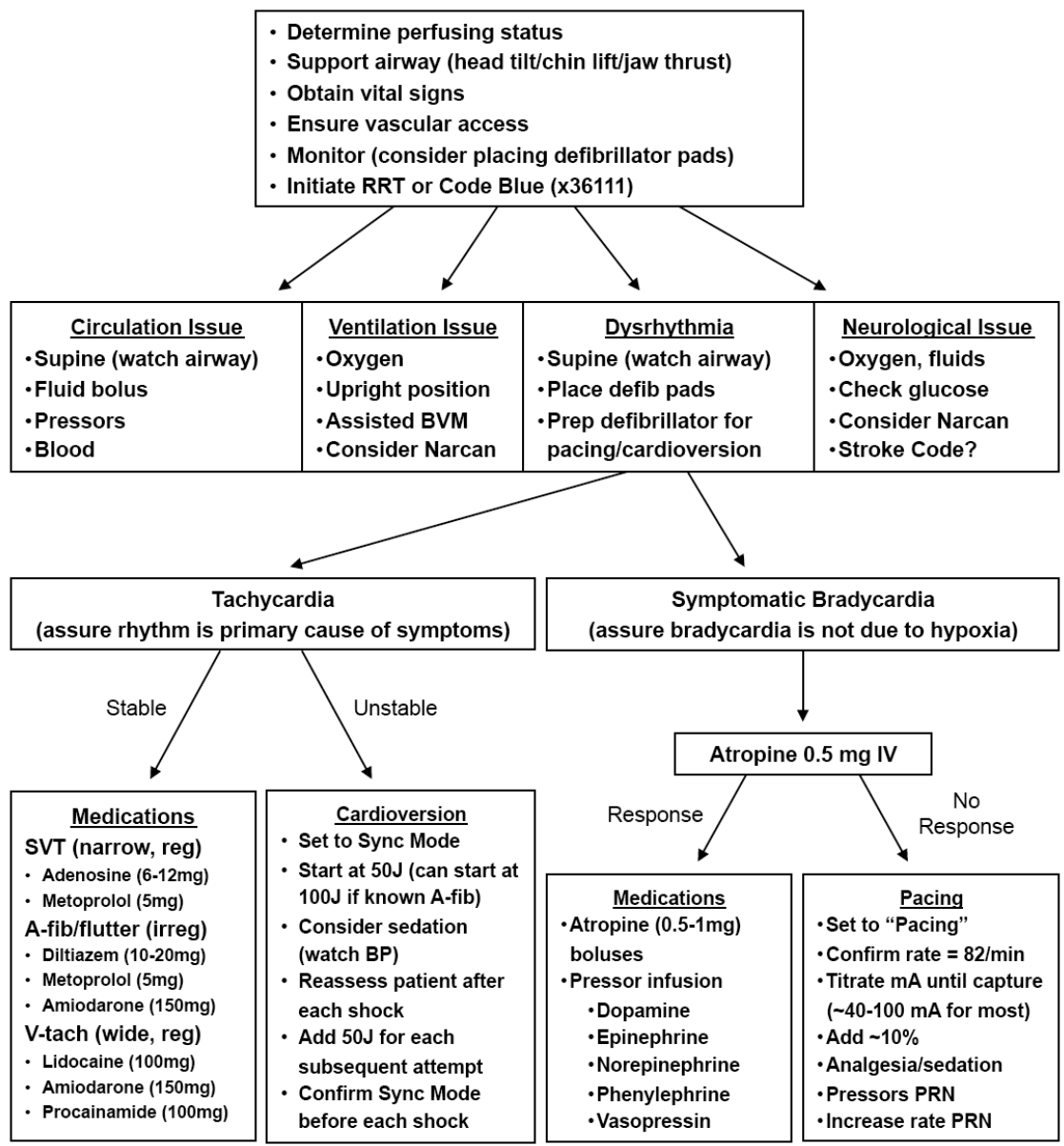
Preventing re-arrest

Maintaining perfusion and oxygenation are the most important factors in preventing deterioration into cardiopulmonary arrest. Ventilations should be continued at a slow-to-moderate rate to avoid the hemodynamic effects of overaggressive ventilation. End-tidal CO₂ should be used to guide ventilation, with a target of 35-40 mmHg. There may be a brief overshoot of end-tidal CO₂, representing a “washout” phenomenon. Fluids and early pressor infusions should be considered with hemodynamic instability, especially with systolic blood pressure values <70 mmHg. End-tidal CO₂ will plummet with re-arrest and should be monitored closely in the post-arrest period.

Reperfusion injury

Much of the damage that accompanies cardiac arrest comes at the time of reperfusion. Many experimental treatments are undergoing investigation but are not yet ready for clinical use. The one therapy that is available currently to prevent reperfusion injury is hypothermia, with substantial improvements in outcome accompanying its use. All cardiopulmonary arrest victims with ROSC should be considered for hypothermia. In addition, excessive oxygen following reperfusion may favor the formation of reactive oxygen species. Thus, FiO₂ should be titrated immediately to achieve target SpO₂ values ~95%.

2009 UCSD PERFUSING PATIENT RESUSCITATION



Perfusing Patients

The goals of resuscitation for perfusing patients are to prevent arrest and minimize ongoing injury, usually from hypoperfusion and/or hypoxemia. Conceptually, patients requiring resuscitation can be categorized into the following: circulatory issue, ventilatory issue, primary dysrhythmia, and neurological issue. A code blue response should be activated for patients in a pre-arrest state, while the rapid response team targets patients further upstream with the first indication of impending deterioration.

Circulatory Issue

Indication of hypoperfusion include hypotension, tachycardia, altered mental status, and serum indicators of acidosis (low pH, elevated lactate, low bicarbonate, wide anion gap, elevated base deficit). Acute coronary syndrome and stroke represent focal circulatory issues. Diagnostic and therapeutic interventions should occur in parallel to avoid further deterioration. These include supine positioning (which may create problems with airway patency in obtunded patients), fluid boluses, blood transfusion, and the use of pressor agents. It is worth noting that a 500 mL saline bolus acutely improves perfusion through a 10% increase in plasma volume, while increasing total body water by only 1%, minimizing the likelihood of complications such as pulmonary edema.

Ventilatory issue

Hypoxemia is one of the most common etiologies of deterioration and cardiopulmonary arrest in the hospital environment. This may occur in patients with known pulmonary disease (pneumonia, COPD, asthma) or with the airway obstruction that accompanies a decrease in mental status (hypotension, hypoxemia, hypoglycemia, analgesia/sedation, sleep apnea). Initial efforts should focus on achieving airway patency (head tilt, chin lift, jaw thrust, nasopharyngeal airway, reversal of altered mental status). Supplemental oxygen should be administered with any degree of hypoxemia (SpO₂ <93%). Patients in respiratory failure have very low tidal volumes and may benefit from upright positioning and assisted ventilation using bag-valve-mask (small volume synchronized to spontaneous breaths) or bilevel ventilation (BiPAP). Ultimately, intubation may be required and should be performed prior to cardiopulmonary arrest.

Neurological issue

The majority of acute neurological deficits within the inpatient population are secondary to hypoperfusion, hypoxemia, hypoglycemia, or seizures. Once these have been excluded, the major objective is to identify patients with acute stroke for possible thrombolysis. A “stroke code” should be activated with any concern about acute stroke. The rapid response or code blue teams should be activated with altered mental status or other signs of hypoperfusion/hypoxemia.

Dysrhythmias

The most difficult challenge in managing patients with bradycardia or tachycardia is determining whether the dysrhythmia is the primary cause of the problem or secondary to another process.

Tachycardia

Sinus tachycardia is not a primary dysrhythmia but instead represents a physiological response to some other process (shock, fever, pain/agitation, hypoxemia). Any tachycardic patient not in sinus rhythm with signs of hypoperfusion (altered mental status, hypotension, severe dyspnea) should undergo immediate synchronized cardioversion (50J, 100J, 150J, 200J). Stable patients requiring cardioversion should be sedated before attempts.

- Supraventricular tachycardia (SVT; narrow, regular) is usually not accompanied by hemodynamic instability and responds extremely well to adenosine (6 mg, 12 mg, 12 mg rapid IVP); resistant or recurrent SVT may require a beta blocker (metoprolol 5mg IV) or cardioversion.
- Atrial fibrillation/flutter (AF; irregular) may or may not be accompanied by hemodynamic instability. If unstable, then rapid cardioversion should be performed. Otherwise, symptoms may improve if the ventricular rate is slowed using bolus doses of medication (diltiazem 10-20 mg IV, metoprolol 5 mg IV, or amiodarone 150 mg IV) or continuous infusions (diltiazem or esmolol).
- Ventricular tachycardia (VT; wide, regular) is often accompanied by hemodynamic instability and generally requires aggressive treatment. The “standard” medical treatment is amiodarone (150 mg IV), which can produce profound hypotension and should not be used with any hemodynamic instability. Lidocaine (100mg IV q5 min x 3) or procainamide (100mg IV q5-10 min until effect) may be preferable with any instability. Many VT patients will require cardioversion, with or without sedation depending upon hemodynamic status.

Bradycardia

The most common etiology for bradycardia among inpatients is hypoxemia, and the acute treatment should focus on supporting ventilation/oxygenation rather than administering medications or pacing. If ventilation is not the primary cause of the bradycardia, then a test-dose of atropine (0.5 mg IVP) should be administered. A response (improved heart rate and perfusion) indicates that medical therapy will likely be effective (boluses of atropine 0.5-1.0 mg IV, infusions of dopamine 10-20 mcg/kg/min or epinephrine 2-10 mcg/min). Transcutaneous pacing should be initiated if medications are not effective. The rate should default to 82/min. Current should be titrated upward until capture occurs (wide complex beat following each pacer spike), then increase current by ~10% to avoid loss of capture. Mechanical capture and hemodynamic stability should be confirmed. Although increasing rate may improve blood pressure, this should be avoided if cardiac ischemia is suspected. Fluids and pressors should also be considered. Sedation/analgesia may be administered once hemodynamic stability is ensured.

VENTRICULAR FIBRILLATION

Description: very fast (250-350), disorganized, variable amplitude

Clinical: cardiopulmonary arrest, often sudden, may be associated with cardiac ischemia

Treatment: defibrillation (direct countershock); CPR + pressor is most effective at making v-fib more shockable; amiodarone (300mg IV) may be used for persistent/recurrent v-fib

**VENTRICULAR TACHYCARDIA**

Description: fast (120-220), regular, wide; torsade is polymorphic (below, right)

Clinical: variable [arrest OR unstable OR stable (monitor closely)], may reflect cardiac ischemia

Treatment: defibrillation/CPR if arrest; cardioversion if unstable; amiodarone if stable (watch HoTN); lidocaine/procainamide are alternatives; Mg and cardioversion (or defibrillation if arrest) for torsade

**BRADYASYSTOLE**

Description: slow or absent electrical activity

Clinical: cardiopulmonary arrest; usually the end result of cardiovascular or respiratory compromise

Treatment: CPR + pressor; atropine; do NOT pace if patient in cardiopulmonary arrest

**PERFUSING BRADYCARDIA**

Description: slow rhythm

Clinical: often indicates hypoxemia; may also be related to medications or cardiac ischemia

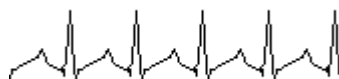
Treatment: try atropine first; if responsive, use atropine PRN or pressor (dopamine, epinephrine) drip; if unresponsive, pace at ~80/min and use pressor infusions following capture

**SUPRAVENTRICULAR TACHYCARDIA**

Description: fast (150-220), regular, narrow (unless aberrancy)

Clinical: usually stable or mildly unstable; usually reflects underlying electrophysiological abnormality

Treatment: vagal maneuvers; adenosine; cardioversion if unstable; Bblocker to prevent recurrence

**ATRIAL FIBRILLATION/FLUTTER**

Description: usually fast (130-200), irregular (afib) or regular ~150 (aflutter), narrow or wide (block)

Clinical: can be stable or unstable

Treatment: cardioversion if unstable; slow with diltiazem or metoprolol/esmolol (watch HoTN); occasional chemical cardioversion (ibutilide, procainamide) if recent onset (<12-24 hours)



Rapid Response Teams

The majority of hospital arrests result from hypoperfusion and/or hypoxemia. In addition, the majority of these patients manifest vital sign abnormalities for several hours prior to arrest. Finally, much of the morbidity that accompanies diseases such as sepsis and acute coronary syndrome can be prevented with early intervention. The triggers for rapid response team activation fall into the following categories: circulation, ventilation, neurological, and infectious.

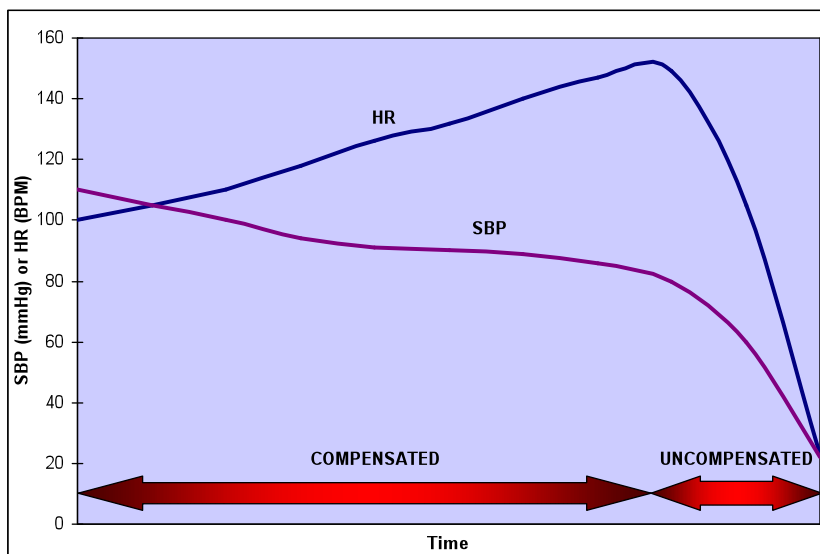
Circulatory issue

Indications of hypoperfusion include hypotension, tachycardia, altered mental status, and serum indicators of acidosis (low pH, elevated lactate, low bicarbonate, wide anion gap, elevated base deficit). Acute coronary syndrome and stroke represent focal circulatory issues. Circulatory triggers include:

- $90 > SBP > 170$ or acute drop in SBP
- $55 > HR > 120$ or acute rise in HR
- Acute chest discomfort
- Acute blood loss

While a diagnostic workup may be indicated, therapeutic interventions should occur in parallel to prevent further deterioration. These include:

- Supine positioning, which can create problems with airway patency in patients with altered mental status
- Fluid boluses; it is worth noting that a 500 mL saline bolus acutely improves perfusion through a 10% increase in plasma volume, but increases total body water by only 1%, minimizing the likelihood of acute pulmonary edema
- Blood transfusion
- Pressor agents



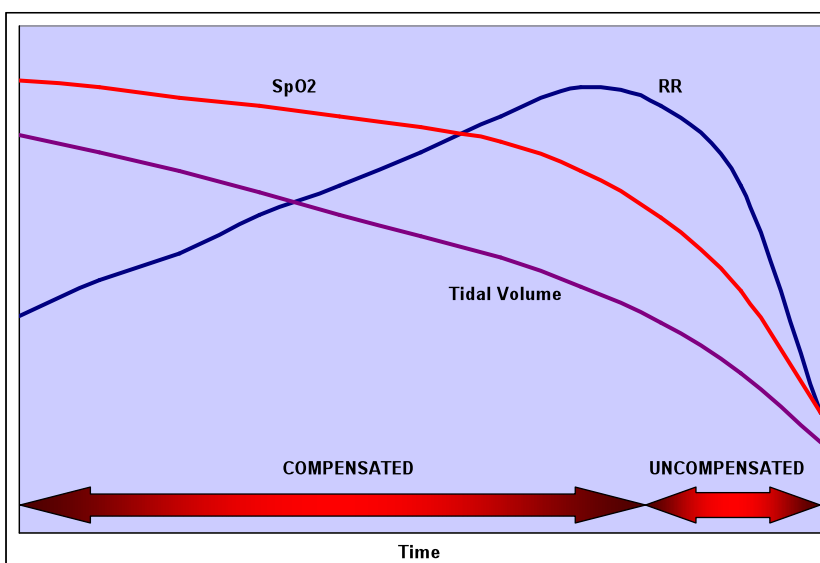
Ventilatory issue

Hypoxemia is one of the most common etiologies of deterioration and cardiopulmonary arrest in the hospital environment. This may occur in patients with known pulmonary disease (pneumonia, COPD, asthma) or with the airway obstruction that accompanies a decrease in mental status (hypotension, hypoxemia, hypoglycemia, analgesia/sedation, sleep apnea). Respiratory triggers include:

- Increased work of breathing
- Stridor/noisy breathing
- $12 > RR > 28$ or acute rise in RR
- $SpO_2 < 93\%$ with increased FiO_2
- ABG for respiratory concerns

While a diagnostic workup may be indicated, therapeutic interventions should occur in parallel to prevent further deterioration. These include:

- Maintaining airway patency (head tilt, chin lift, jaw thrust, nasopharyngeal airway, reversal of altered mental status)
- Supplemental oxygen with any hypoxemia ($SpO_2 < 93\%$)
- Upright positioning
- Assisted ventilation using bag-valve-mask or bilevel ventilation (BiPAP)
- Intubation may be required and should be performed prior to arrest

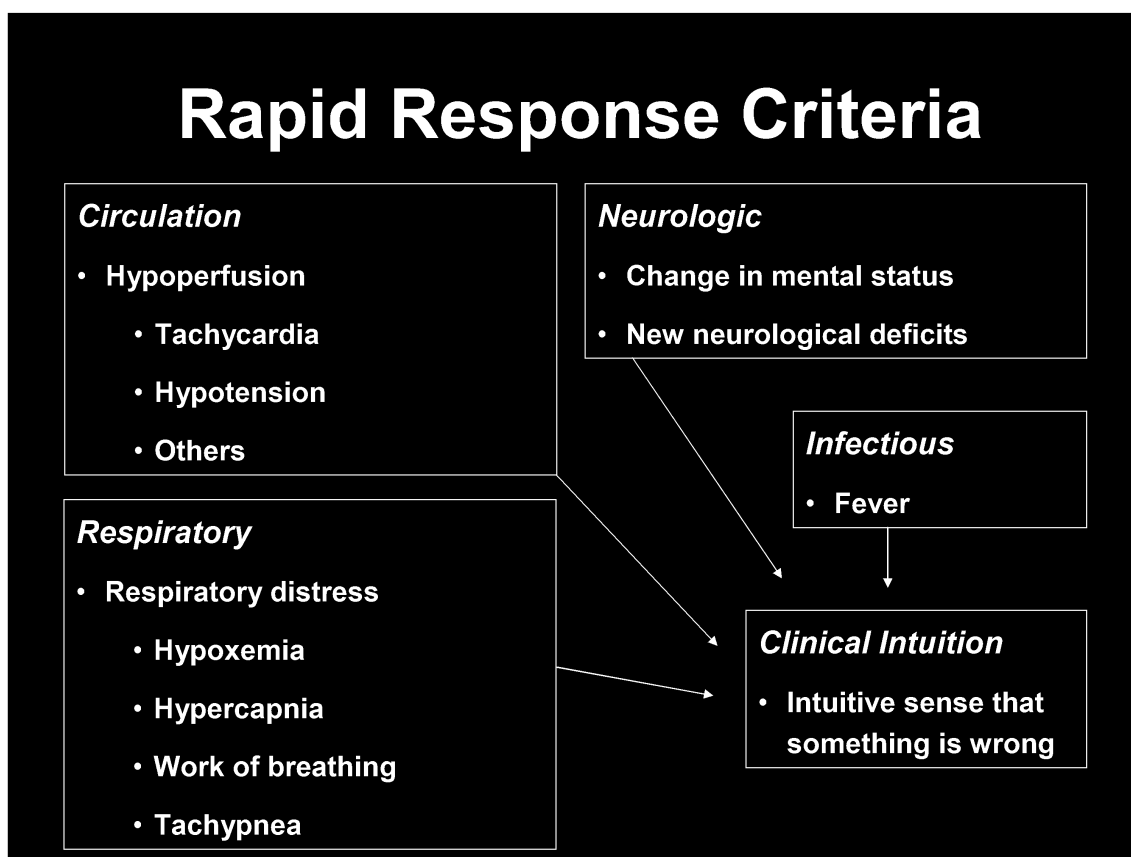


Neurological issue

The majority of acute neurological deficits within the inpatient population, especially global alterations in mental status, are secondary to hypoperfusion, hypoxemia, hypoglycemia, or seizures. A stroke code should be activated with any concern about acute stroke, especially with focal neurological deficits. The rapid response or code blue teams should be activated with altered mental status or other signs of hypoperfusion/hypoxemia, including restlessness/agitation or confusion.

Infectious issue

Hyperthermia ($T >39.5^{\circ}\text{C}$), hypothermia ($T <35^{\circ}\text{C}$), or clinical suspicion of systemic infection should prompt a workup as well as an aggressive approach to therapy to avoid decompensated sepsis. Early sepsis criteria include indications of hypoperfusion along with clinical suspicion of infectious etiology. Interventions include appropriate hemodynamic monitoring, early administration of antibiotics, aggressive volume replacement, transfusion, use of pressors to promote perfusion (dobutamine), and consideration of steroids with suspected adrenal insufficiency.



ART SKILLS CHECKLIST

Name: _____ Date: _____

Unit: _____ Email: _____ Instructor: _____

Initial Assessment

- Responsiveness
- Open airway and check for spontaneous breathing
- Pulse check (optional)
- Call for help (Code Blue or 9-1-1)
- Start compressions

Compressions

- Appropriate depth & recoil
- Rate 80-120/min (guided by depth/recoil)
- Switch every 2 min (or less) with minimal delays

Defibrillation

- Identification of shockable rhythm
- Charge while compressions performed
- Shock with minimal pause
- Restart compressions immediately

Ventilations

- No pause in compressions
- One breath every 10 compressions
- Proper mask hold ("two thumbs up" with head tilt/jaw thrust/NPA/OPA)

All Patients

- Continuous compressions
- Early pressor
- Consideration for other drugs (atropine, amiodarone)

Perfusion Check

- Appropriate interval (2 min) since last perfusion check or shock
- Organized complexes >40/min (with rise in EtCO₂)
- Hold compressions and check for pulse
- Ventilate slowly (and watch for fall in EtCO₂)
- Pulse oximetry waveform

Post-resuscitation Care

- Slow ventilations (guided by EtCO₂)
- Fluids/pressors for hypotension

Perfusing Patients

- Pacing
- Synchronized cardioversion
- Ventilatory support (upright positioning, bag-valve-mask assist)
- Circulatory support (supine positioning, fluids)

General

- Overall comfort
- Ability to function effectively as team member
- Pediatrics & choking (if necessary)