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.

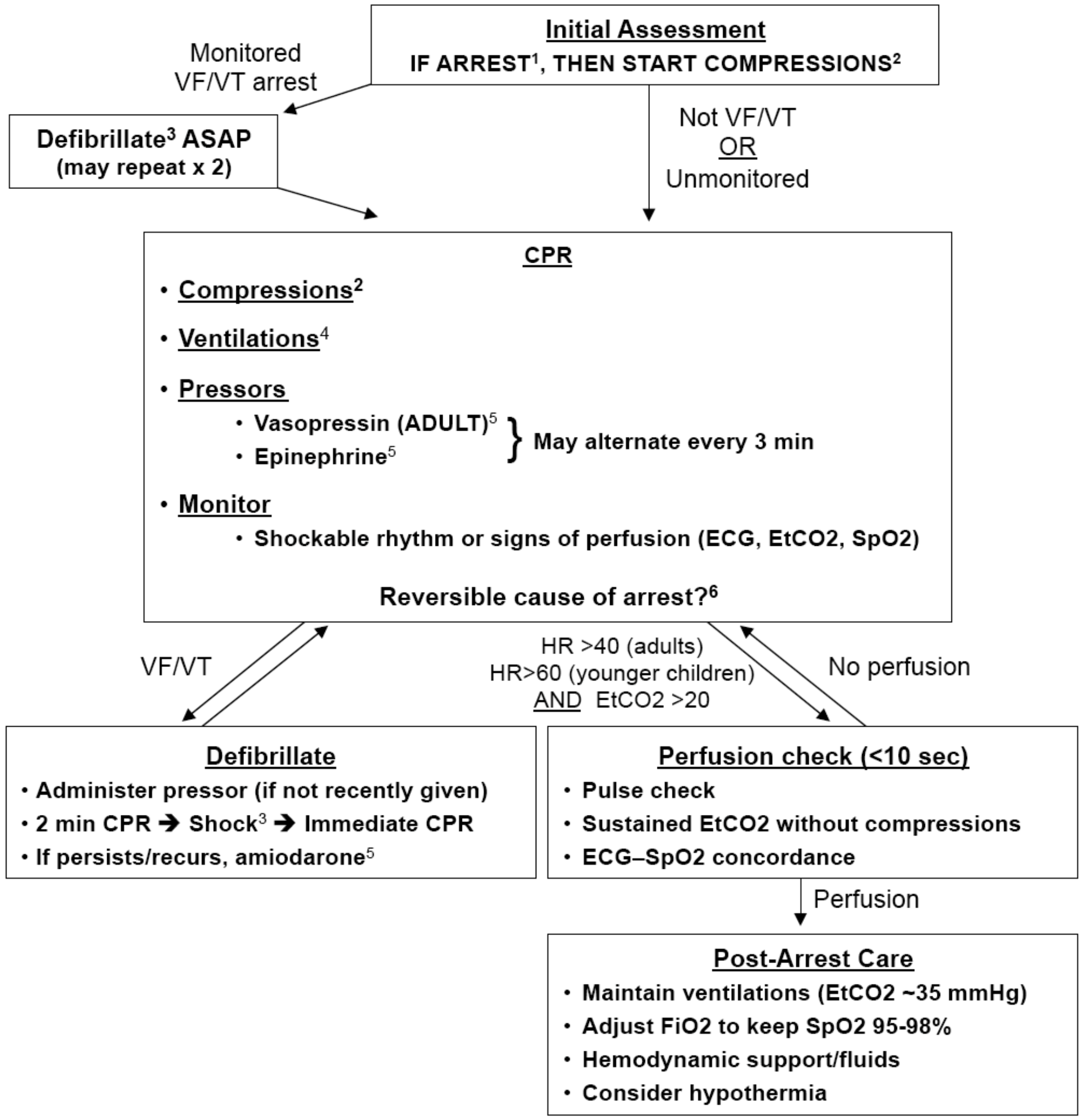
II. There has been an exponential increase in our knowledge regarding the resuscitation physiology and optimal therapy, requiring more frequent updates and a flexible curriculum.

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.

2012 UCSD ART/PART ARREST ALGORITHM



[1] Unresponsive + apneic/gasping (+/- pulseless) OR VF/asystole OR HR<30 (adults), HR<60 (younger children) OR sudden ↓ HR/EtCO2

[2] Continuous compressions; 2+ inches adult, at least 1/3 AP diameter ped, 80-120/min, full recoil

[3] 120J-150J-200J adult, 2J/kg-4J/kg-4J/kg ped; minimal pause in compressions before/after shocks

[4] Synchronous (10:1) with continuous compressions (no pause); two rescuers; "two-thumbs-up" mask hold; consider OPA/NPA/cricoid pressure; intubate (min compression pause) when possible

[5] Vasopressin (40U adult); epinephrine (1mg adult; 0.01mg/kg (0.1mg/kg ET) ped); amiodarone (300mg-150mg adult; 5mg/kg-5mg/kg-5mg/kg ped); atropine (1mg adult; 0.02mg/kg, min 0.5mg ped)

[6] Hypovolemia (IVF/blood); hyper-K^+ (NaHCO3, CaCl2); hypoglycemia (D50); acidosis (NaHCO3); PE (TPA); tension pneumothorax (needle); vagal (atropine); hypothermia (warming); recurrent VF (PCI)

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 despite an open airway can be used as an indicator of cardiopulmonary arrest. The “gulping” or “gasping” 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. The “shark hook” maneuver can be used to open the airway.

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 (<30-40 beats/min in an unresponsive patient) 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 – at least 2 inches – with full recoil produce better perfusion. This may limit the rate of compressions for larger patients. The mattress may lead to an overestimation of the true compression depth by the defibrillator, particularly if a compression board is not used. The compressor should be directly above the patient, with hands on the lower sternum, for maximum depth. The palm should lift off the chest during recoil phase – enough to “swipe a credit card”.

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.

Monitored VF/VT 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. A “stack” of 3 or more shocks should be delivered for a monitored arrest.

Unmonitored VF/VT (or secondary VF/VT) 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 10^{th} 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 10^{th} 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 10^{th} 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. Are adequate compressions being performed?
2. Are adequate ventilations being performed?
3. Has a pressor agent (vasopressin or epinephrine) been administered?
4. Does the monitor reveal either a shockable rhythm or evidence of perfusion?
5. Is a reversible cause of arrest present?

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 5-10 minutes; epinephrine 1 mg can be given IVP every 3-5 minutes. If a slow rate (<60 beats per minute) due to a suspected vagal event (patient with multiple lines/tubes/catheters being moved or suctioned, commode-related event), atropine 1 mg IVP may be administered early in the resuscitation.

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
- Severe acidosis (sepsis, blood loss, renal failure, asphyxia) – sodium bicarbonate
- Suspected hypoglycemia (diabetic) – dextrose
- Suspected tension pneumothorax (trauma or COPD patient) – needle decompression or chest tube insertion
- Coronary thrombosis (persistent or 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. There may be exceptions early in an arrest that is likely to involve a primary conduction abnormality.

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 (>40/min) and a rise in end-tidal CO_2, with values <20 mmHg unlikely to be associated with ROSC. This rise in end-tidal CO_2 ("mechanical recovery") may take several minutes following the initial rise in heart rate ("electrical recovery"). High-quality CPR can also produce CO_2 values >20 mmHg in the absence of spontaneous perfusion. This can be determined by observing a rapid drop in end-tidal CO_2 during a brief pause in compressions while continuing controlled ventilations. 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<40/min) and/or a low end-tidal CO_2 value (<20 mmHg), compressions should be continued without pausing.

Perfusion check

If ROSC is suspected (organized complexes with a rate >40/min and an increase in end-tidal CO_2 >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 CO_2 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 <12-15 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 95-98%.

2012 UCSD ART/PART PERFUSING PATIENT ALGORITHM

- **Determine perfusing status**
- **Support airway (oxygen, “shark hook”, NPA/OPA, advanced airway)**
- **Obtain vital signs**
- **Ensure vascular access**
- **Monitor (consider placing defibrillator pads), consider ECG**

Circulation Issue
- **Hemorrhage control**
- **Supine (watch airway)**
- **Fluid bolus, blood**
- **Pressors**

Ventilation Issue
- **Oxygen**
- **Upright position**
- **Assisted BVM**
- **Consider Narcan**

Dysrhythmia
- **Supine (watch airway)**
- **Place defib pads**
- **Prep defibrillator for pacing/cardioversion**

Neurological Issue
- **Oxygen, fluids**
- **Check glucose**
- **Consider Narcan**
- **Stroke Code?**

Tachycardia
(assure rhythm is primary cause of symptoms)[1]

Stable

Medications

SVT (narrower, reg)
- **Adenosine[3]**

A-fib/flutter (irreg)
- **Diltiazem[3]**

V-tach (wider, reg)
- **Amiodarone[3]**

Unstable

Cardioversion
- **Set to Sync Mode**
- **Energy settings same as defibrillation[4]**
- **Consider sedation**
- **Confirm Sync Mode before each shock**

Symptomatic Bradycardia
(assure bradycardia is not due to hypoxia)[2]

Atropine (1st line adult)[5]
OR
Epi/pressors (1st line ped)[5]

No Response

Pacing
- **Set to “Pacing”**
- **Confirm rate (82/min adult, 100/min ped)**
- **Titrate mA until capture**

[1]Dysrhythmia more likely with: sudden change in rate, change in morphology or wide complexes, faster rates (HR>150 adult, HR>180 child, HR>220 infant)

[2]Heart rate will begin to slow with SpO2 <70-80%, in which case oxygen is correct therapy

[3]Adenosine (6mg-12mg adult; 0.1mg/kg-0.2mg/kg ped); diltiazem(10-20mg adult); amiodarone (150mg adult; 5mg/kg ped)

[4]120J-150J-200J adult; 2J/kg-4J/kg-4J/kg (initial attempt at 1J/kg also acceptable) ped

[5]Atropine (0.5mg-1mg q3 min adult; 0.02mg/kg q3 min, min 0.5 mg ped); epi infusion (1-10 mcg/min adult; 0.01-1 mcg/kg/min ped); epi boluses (0.01 mg/kg q3min ped); dopamine (5-20 mcg/kg/min)

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 using the same energy settings as for defibrillation (120J-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 amiodarone 150 mg 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). Lidocaine (100mg IV q5 min x 3) or procainamide (100mg IV q5-10 min until effect) are infrequently used. 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

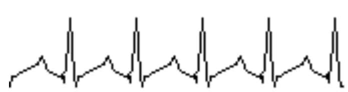
Description: slow or absent electrical activity
Clinical: cardiopulmonary arrest; usually the end result of cardiovascular or respiratory compromise
Treatment: CPR + pressor; atropine; do NOT pace if patient in cardiopulmonary arrest

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.

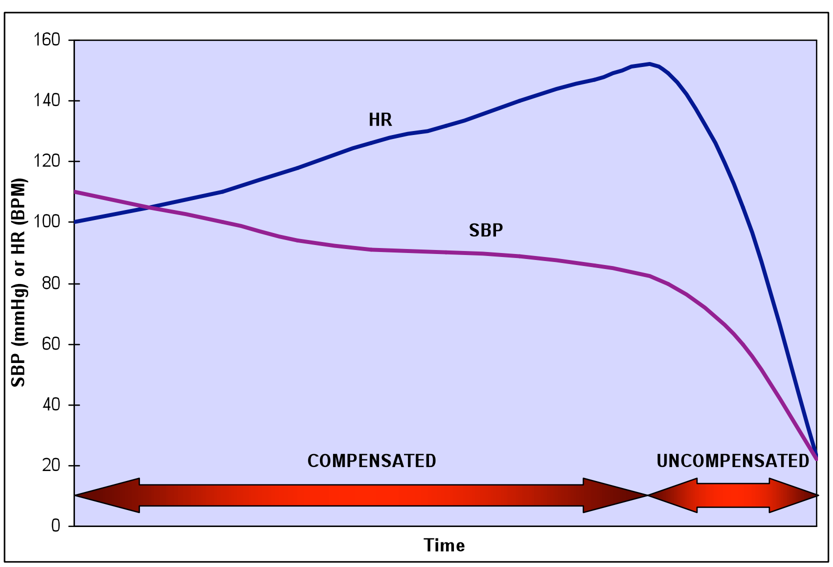
Circulatory issue

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



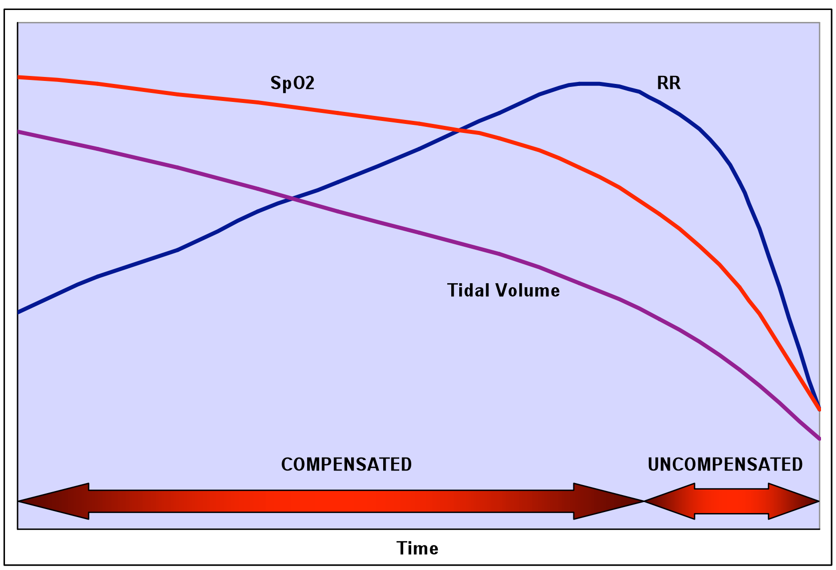
Ventilatory issue

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
- SpO2 <93% with increased FiO2
- 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 (SpO2 <93%)
- Upright positioning
- Assisted ventilation using bag-valve-mask or bilevel ventilation (BiPAP)
- Intubation may be required and should be performed prior to arrest

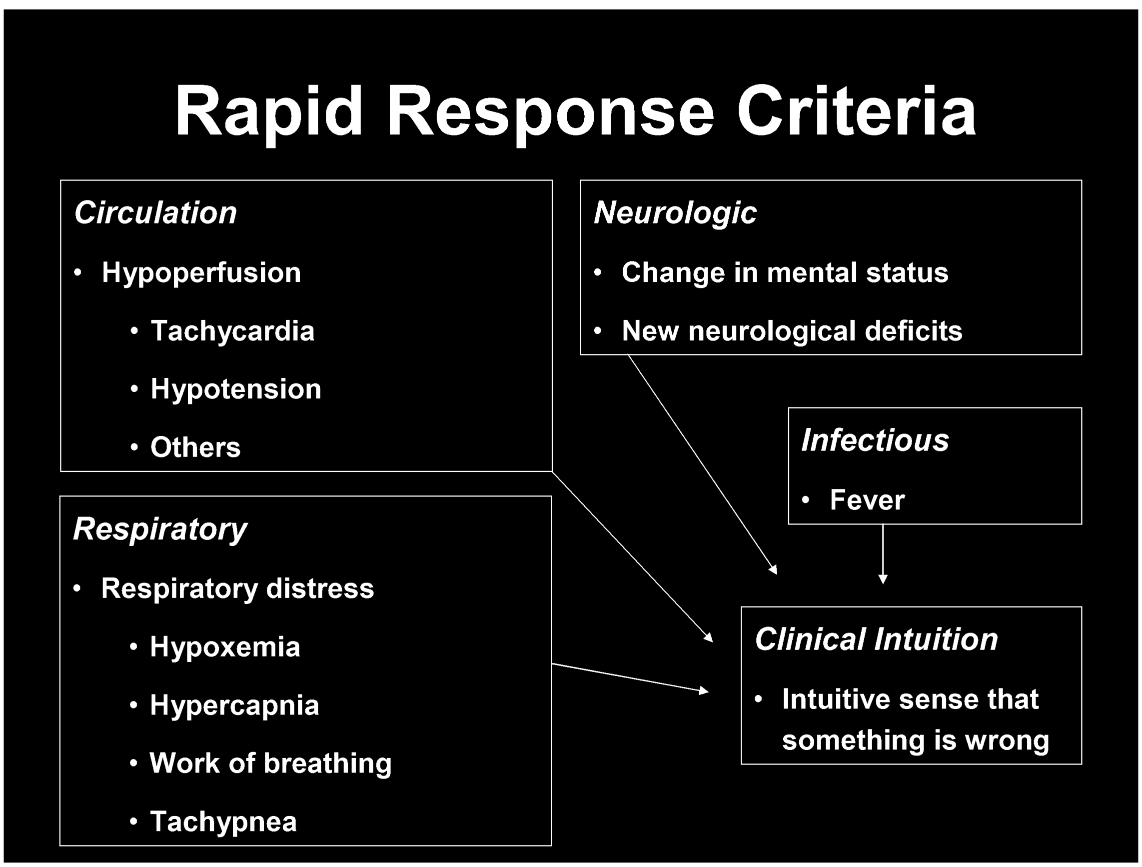


Neurological issue

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^{o}C), hypothermia (T <35^{o}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.



Choking

Choking is managed differently in infants than in adults or non-infant children. Infants do not generate high negative inspiratory pressures, and perhaps because of the narrower subglottic airway are more likely to develop laryngospasm with any peri-glottic foreign body to prevent tracheal entry. This means that while an infant may appear to be obstructed, the object is generally not tightly lodged in the airway. Simply placing the infant into a head-down position and performing multiple (10-20) back blows will usually dislodge the foreign body. Spontaneous respiration (usually crying) will resume once the laryngospasm has spontaneously reversed. Infant ribs are cartilage, making the infant chest wall more distensible. Thus, in the rare occasion of a foreign body lodged tightly in the airway, chest squeezes (3-5 repetitions in the head-down position followed by a return to back blows) will allow direct compression of the lungs to expel the object.

Adults and non-infant children can generate higher negative inspiratory pressures and will typically alternate between violent coughing and “whooping” to move a foreign body away from the vocal cords, which are the narrowest portion of the airway. A choking victim who is still moving air in and out, as evidenced by the coughing and whooping sounds, should be allowed to clear his or her airway spontaneously. With near-complete or complete obstruction, which is associated with a lack of airflow during coughing or whooping, rescuer attempts to expel the foreign body should be initiated. Because of the ossification of ribs, the most effective strategy to compress the lungs is from beneath the rib cage, using the Heimlich maneuver in an upright patient or abdominal thrusts in a supine patient. Both maneuvers should be applied so as to direct the umbilicus upward and inward toward the diaphragm.

In infants, non-infant children, and adults, progression to full cardiopulmonary arrest (unresponsive and apneic) should result in the immediate implementation of chest compressions. A single attempt to open the mouth and visualize the foreign body can be made, with a finger sweep used to remove the object ONLY if it is visible. Either way, chest compressions become the most important therapy once cardiopulmonary arrest has developed. It is possible that the foreign body will be expelled with chest compressions, which appear to generate higher intra-tracheal pressures than the Heimlich maneuver or abdominal thrusts.

Pediatric Arrest

The general principles of resuscitation in adults are also relevant to pediatric patients (up to age 8). Immediate recognition of arrest and rapid initiation of chest compressions are basic tenets of pediatric resuscitation. However, the etiology of arrest in children differs from that in adults. Most pediatric cardiopulmonary arrest occurs as a result of asphyxia, either from Sudden Infant Death Syndrome (SIDS) in infants or as a result of traumatic brain injury in toddlers and young children. Because of this, the general approach to pediatric resuscitation from cardiopulmonary arrest emphasizes ventilation to a greater degree than in adults.

The specific actions recommended for pediatric inpatient resuscitation are covered in the Pediatric Advanced Resuscitation Training (PART) course. The initial resuscitation of a pediatric patient should include an initial assessment that is identical to that for adults – responsiveness followed by airway opening and assessment of spontaneous respiration. Pulse checks can be even more challenging in pediatric patients; thus, the absence of responsiveness and spontaneous breathing is adequate to determine cardiopulmonary arrest and initiate chest compressions. The depth of compressions should be determined by the individual patient's chest (one-third to one-half the AP diameter), with full recoil allowed between compressions. The rate should be as fast as possible, as long as adequate depth and recoil are maintained. In an inpatient setting, compressions may be initiated by the first rescuer, with ventilations applied once additional providers arrive. An adult bag-valve-mask can be used to ventilate a pediatric patient, as long as the mask is placed against the face rather than over the chin, using the "two thumbs up" approach described previously. Continuous compressions with a breath inserted between compressions at a 10:1 ratio should be performed when bag-valve-mask is available. If no bag-valve-mask is available, mouth-to-nose/mouth ventilation should be performed at a 30:2 ratio with a single rescuer and 15:2 ratio with multiple rescuers.

ART SKILLS CHECKLIST

Name: ____ Date: _____________

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 100-120/min (guided by depth/recoil)
- ☐ Switch every 2 min (or less) with minimal delays

Defibrillation

- ☐ Identification of shockable rhythm
- ☐ Monitored arrest – stacked shocks
- ☐ Unmonitored arrest or 2^{o} VF/VT – single shocks (CPR + pressor before each shock)
- ☐ Shock with minimal pre- and post-shock pauses
- ☐ Energy sequence 120-150-200

Ventilations

- ☐ One breath every 10^{th} compression, synchronous with no compression pause
- ☐ Proper mask hold ("two thumbs up" with head tilt/jaw thrust)
- ☐ Use of NPA/OPA

All Patients

- ☐ Continuous compressions
- ☐ Early pressors (Vasopressin 40u IVP, epinephrine 1mg IVP)
- ☐ Consideration for other drugs (atropine, amiodarone)

Perfusion Check

- ☐ Appropriate indications (HR >40, EtCO2 rise >20)
- ☐ Hold compressions and check for pulse
- ☐ Ventilate slowly and observe EtCO2 pattern
- ☐ Pulse oximetry waveform concordance with ECG
- ☐ Arterial line waveform if available

Post-resuscitation Care

- ☐ Slow ventilations (guided by EtCO2)
- ☐ Fluids/pressors for hypotension
- ☐ Consider PCI, hypothermia, EEG monitoring

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)