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Anesthesia Advanced Circulatory Life Support:

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Introduction:

Advanced Cardiac Life Support (ACLS) was originally developed as an extension of Basic Life Support (BLS), and thus focused on the resuscitation of individuals found unresponsive in the field. It was subsequently expanded to encompass their immediate care in the emergency department, and has been exported to patients found unresponsive anywhere else in the hospital. The initiation of ACLS is predicated upon the discovery of an unresponsive patient who does not have a pulse. ACLS is rhythm oriented and specific to sudden manifestations of cardiac disease during everyday life; it presumes that effective electrical and pharmacological management of a pulseless electrical rhythm will result in the *return of spontaneous circulation* (ROSC).

Cardiac arrest during anesthesia is distinct from cardiac arrest in other settings in that it is usually witnessed, and frequently anticipated. In comparison to other settings, the response is both more timely and focused. In many instances, the prognosis is improved by both a detailed knowledge of the patient and the enormous resources, which can be mobilized in a short time.

In the perioperative setting, patients typically deteriorate into a pulseless arrest over a period of minutes or hours, under circumstances wholly dissimilar to other in-hospital or out-of-hospital scenarios. Consequently, aggressive measures taken to support their physiology can avert, avoid, or forestall the need for ACLS. Additionally, patients in the perioperative period have a different milieu of pathophysiology. For example, hypovolemia is far more common than transmural infarction from plaque rupture and intraoperative myocardial ischemia from O₂ delivery consumption imbalance rarely evolves to full pump failure or ventricular fibrillation in the operating room. The result is a different spectrum of dysrhythmias and desirable interventions in the operating room than in the Emergency Department. The most common cardiac dysrhythmia during general and neuraxial anesthesia is bradycardia followed by asystole (45%). The other life threatening cardiac rhythms are severe tachydysrhythmias including ventricular tachycardia, ventricular fibrillation (14%), and pulseless electrical activity (7%). Remarkably, in 33% of the cases the heart rhythm is not fully assessed or documented.

While the cause of circulatory arrest is usually unknown in patients found down in the field, there is a relatively short list of probable causes in patients who have circulatory collapse in the perioperative period. This certainty produces more focused and etiology-based resuscitation efforts, which frequently do not comply with the more generic algorithms of the ACLS guidelines. While some construe this as sub-standard care, most experts in resuscitation in the operating room regard it as entirely appropriate. In fact it provides care tailored to the patient's unique and specific clinical situation.

When ACLS was first introduced, it was the consensus product of a small multidisciplinary group with a common interest in ACLS. There was little clinical science to guide and shape the guidelines they authored. Fortunately, the scenarios they took an interest in were sufficiently common that they permitted systematic study, which has facilitated subsequent revisions of ACLS guidelines. The current guidelines have their foundation in a large number of directly applicable studies. Unfortunately, these studies have focused upon the issues and circumstances outside the perioperative, thus the guidelines generated translate less well into the perioperative setting. While cardiac arrest in the community remains a common problem, cardiac arrest in the perioperative period is relatively rare. This makes it difficult or impossible to perform large epidemiological studies, and frustrates the generation of evidence-based guidelines. In spite of this, there is a wealth of expertise and experience among anesthesiologists in managing both circulatory crisis and cardiac arrest in perioperative patients.

We offer these guidelines as a consensus statement from a group of experts, hoping that they will inspire the systematic study of how to manage these rare events.

1. Pre-arrest/ Avoiding arrest: Rescue

Failure to rescue is a commonly misidentified 'cause' of cardiac arrest. It is rare that the practitioners caring for a patient fail to realize that they are in crisis. Regrettably, in most instances the problem is not a 'failure' to rescue but rather an inability to rescue: the patients' underlying process was so severe that disaster would have been inevitable in spite of the timely institution of maximal support.

Rescue requires two separate and very distinct components: **comprehension** that the patient is in crisis and **effective action** to manage it. In practice, recognizing that a patient is in crisis is far more difficult than effectively responding. Patients can have poor outcomes in spite of both timely recognition of crisis and the institution of effective therapies. Hindsight bias affords reviewers a clear view of the evolution of a crisis, along with the luxuries of time and access to infinite resources thus making confident proclamations that it might have been averted. Below are some ideas about how to recognize and manage patients in crisis.

Cardiac arrest in perioperative patients typically occurs as a consequence of either hypoxemia or the progression of a circulatory process. Avoiding cardiac arrest requires successfully managing acute anemia, hypoxemia, and all contributing factors to cardiac output: preload, contractility, and afterload. Anesthesiologists as a group are masters of recognizing and treating hypoxemia, and consequently the focus of the remainder of this document will be on the management of cardiopulmonary interactions and the circulation in the rapidly decompensating patient.

Avoiding Cardiac Arrest

- afterload
- contractility
- preload

ACLS/ACLS rhythms

Auto-PEEP: When the lungs choke the circulation

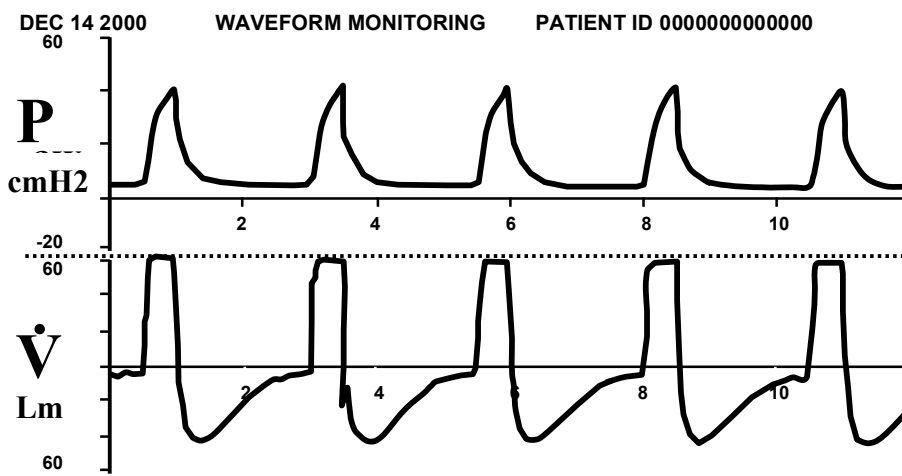
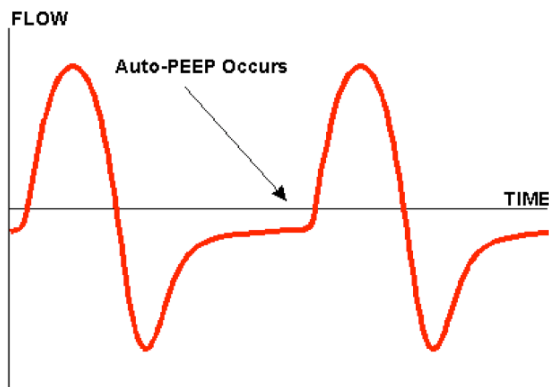
Auto-PEEP, also known as *intrinsic PEEP* and *gas trapping*, is a phenomenon that occurs almost exclusively in patients with obstructive lung disease, both asthma and COPD (emphysema). In these patients, patterns of ventilation which do not allow sufficient time for complete exhalation produce a gradual increase in the end-expiratory volume and pressure in the lung. This pressure is transmitted to the great veins in the thorax and depresses both venous return and cardiac output. As the auto-PEEP increases, the venous return declines. Auto-PEEP is well described as a cause of circulatory collapse, and a very difficult to recognize cause of PEA/EMD (pulseless electrical activity/Electromechanical Disassociation).

Practitioners as a group may be concerned that hypoventilation will have deleterious effects. This has been the conventional wisdom in anesthesia and medicine for the past 50 years, but has been overturned by a variety of clinical observations and studies in the past 20 years. Although none of these studies were of perioperative patients, there are a large number of case series and studies from the past 15 years, all of which suggest a survival benefit to moderate hypoventilation and respiratory acidosis. Hypoventilation is clearly and reproducibly associated with a lower incidence of barotrauma in patients with ARDS or COPD. Furthermore patients who experience cardiac arrest during the perioperative period almost uniformly are receiving some form of supplemental oxygen therapy and as such are less prone to experience 'hypoventilation hypoxemia'.

Capnography is misleading in obstructive lung disease and the more severe the obstructive lung disease, the more misleading the capnography data. Most experts agree upon two things: 1. patients with severe lung disease tolerate hypercarbia and respiratory acidosis very well, and 2. that these patients should be ventilated with high inspiratory flows (and their associated high peak airway pressures) and respiratory rates no higher than 12 breaths a minute. If auto-PEEP is suspected as a cause of circulatory crisis, disconnecting a patient's tracheal tube from the ventilator for a brief time (10-20 seconds) can produce a dramatic improvement in the circulation. Patients who demonstrate dramatic improvement in response to this maneuver will benefit from maximal therapy for obstruction/bronchospasm, and will likely fare best with lower minute ventilations and ventilator rates.

Detecting and decreasing auto-PEEP is a straightforward way to support a sagging circulation. It should be among the first assessments performed in a susceptible patient with an unstable circulation, as the most effective response to the presence of a large amount of auto-PEEP is to decrease it.

Airflow obstruction



In the above figures, the failure of the expiratory waveforms to return to the zero baseline before the next inspiration is indicative of the presence of auto-PEEP.

Importantly, animal models of circulatory crisis and of CPR demonstrate that hyperventilation is almost invariably associated with worsened survival. Ventilation at 20 breaths a minute is associated with significantly lower survival than ventilation at 12 breaths a minute. As a whole, these studies emphasize the principle : *in a low flow state the duration of increased intrathoracic pressure is proportional to the ventilation rate and inversely proportional to blood pressure, coronary and cerebral perfusion.* This is why more recent versions of the ACLS guidelines have recommended much lower levels of ventilatory support, and is the rationale driving the development of technologies to ventilate patients using negative pressure. It is also the genesis of one the recommendations in these guidelines:

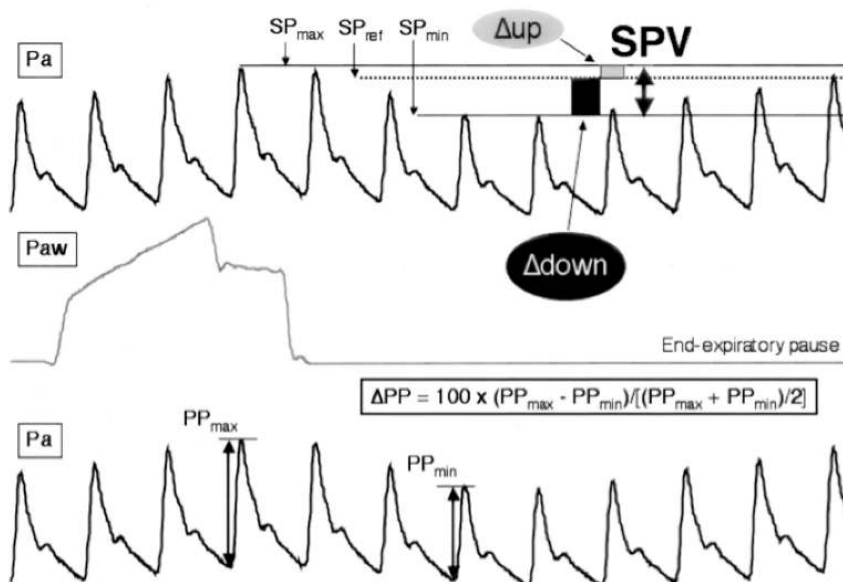
Patients with an unstable circulation should receive sufficient support to adequately oxygenate their blood, but should otherwise be ventilated with the smallest tidal volumes and the lowest rate that their care givers feel is safe.

Escalating Care:

If, as a practitioner, you feel the urge to consult this outline or initiate therapies outlined in the guidelines and algorithms below, it is also appropriate to give serious consideration to escalating the level of monitoring in parallel with the level of supportive care. The timely insertion of both an arterial line and a central venous line will likely be very helpful in the serial evaluation of patients as outlined below. Insertion of invasive monitors should not take precedence over supportive measures. The decision to escalate the level of monitoring, like the decision to escalate therapies, is ultimately a clinical decision that accounts for a large number of factors, beyond the scope of these guidelines.

Hypovolemia and Systolic Pressure Variation

The most common cause of hypotension in the perioperative period is hypovolemia, and the most reliable indicator of hypovolemia is systolic and pulse pressure variation. The cause of hypovolemia is usually, but not always self-evident in the perioperative patient.



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The greater the fluctuation of the systolic pressure and pulse pressure with respiration, the more likely the patient will respond to volume infusion (SPV figure).

The corollary is also true: minimal or absent systolic and pulse pressure variation with respiration strongly suggest that interventions other than the infusion of volume will be required to support the circulation (add positive inotropes, eliminated negative inotropes). Importantly, large tidal volumes (>8 ml/Kg), higher lung compliance (emphysema) and lower chest wall compliance (3rd degree chest burn) will also cause an increase in systolic pressure variation, requiring practitioners to incrementally adjust their criteria for assessing the need for volume infusion.

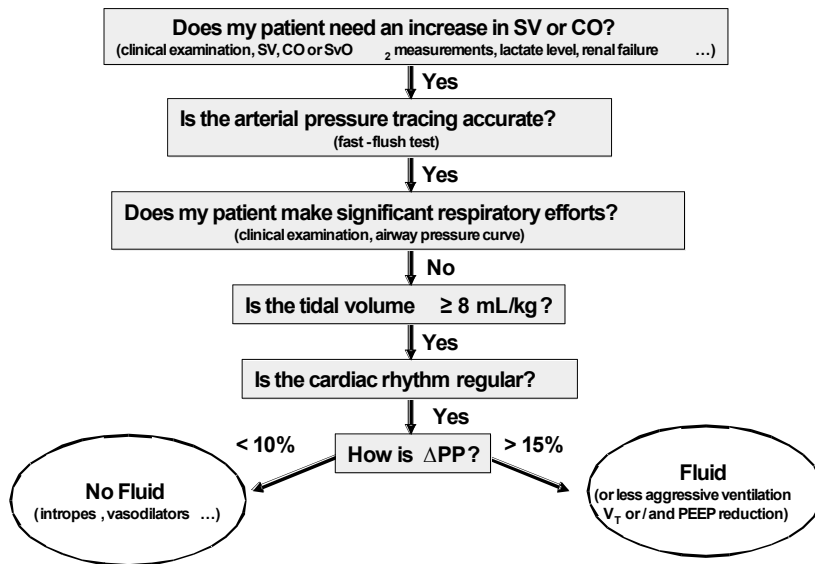


Fig. 9. How to assess the respiratory variation in arterial pressure in clinical practice. CO = cardiac output; Δ PP = arterial pulse pressure variation; PEEP = positive end-expiratory pressure; SV = stroke volume; SvO_2 = mixed venous oxygen saturation; V_T = tidal volume.

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Figure 2 above provides a simple algorithm for choosing between volume infusion and other measures to support the circulation.

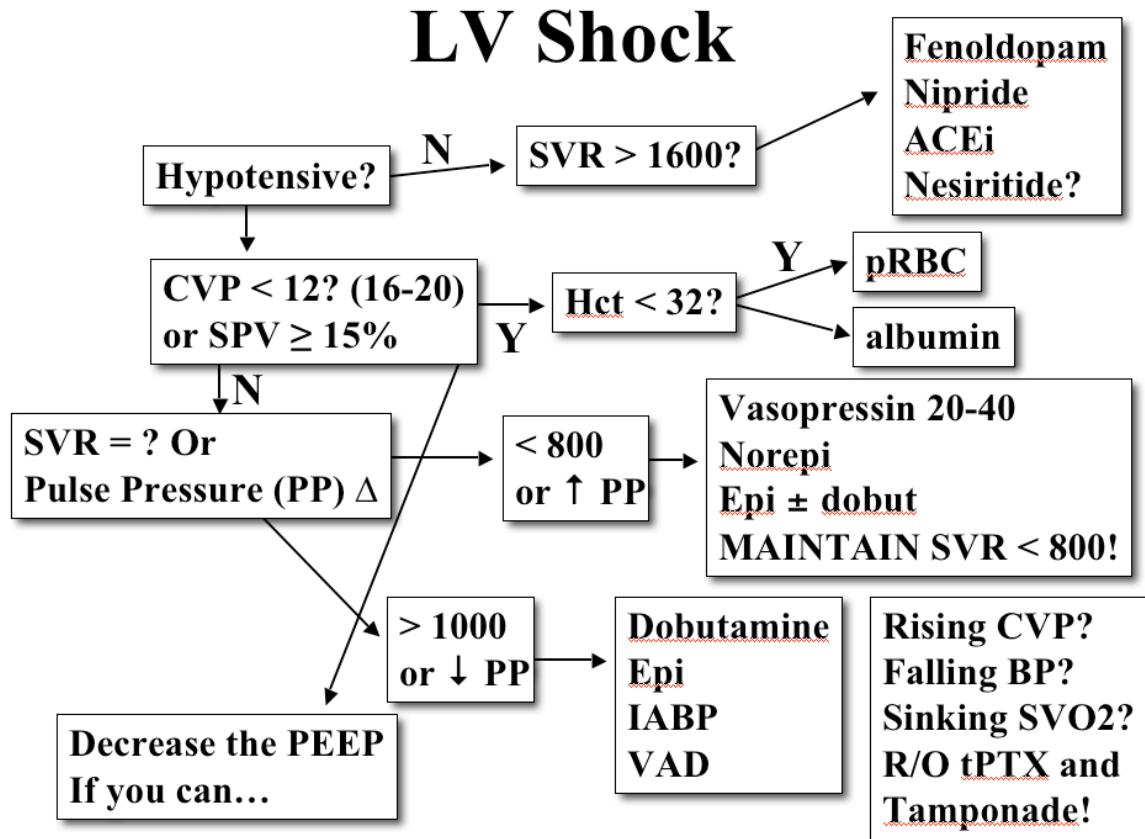
How much volume is too much? This debate continues unabated in the modern era. In the absence of consensus and when presented with a patient in crisis, it is reasonable for practitioners to volume resuscitate as long as there is both clinical evidence the patient might respond and they are not obviously in high pressure pulmonary edema.

Pump Shock

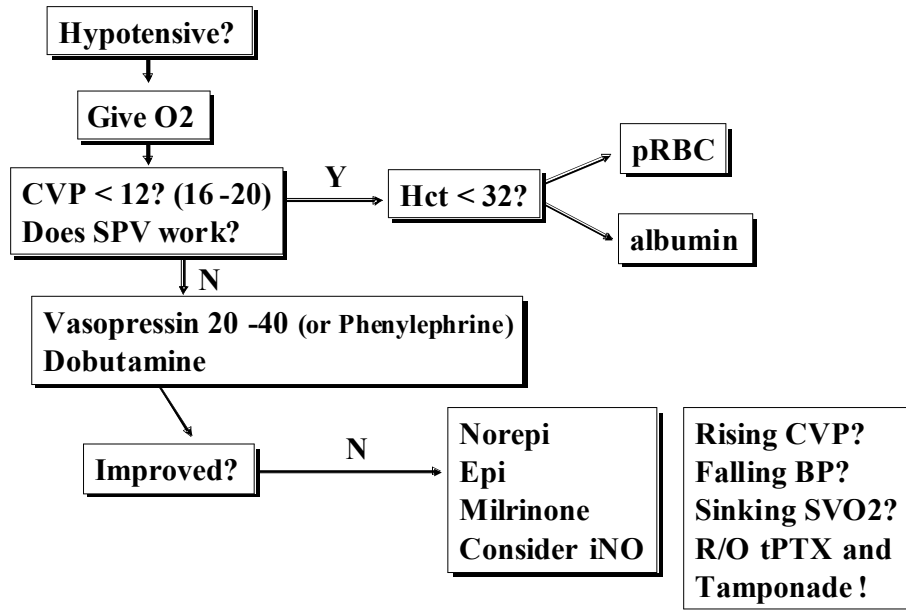
The management of LV failure is substantially different than the management of RV failure. In both settings, an adequate circulating volume is essential for ventricular filling and forward flow. The failing LV is best supported with afterload reduction when possible, followed by positive inotropes. Mechanical assist devices are available in some settings to support the patient with LV failure, but escalation of therapy to that level may not always be possible or clinically appropriate.

The failing RV is best managed with some combination of pulmonary vasodilators and positive inotropes. Unlike the setting of LV failure, the use of systemic arterial vasoconstrictors in this setting is usually associated with improved end organ perfusion and cardiac output. At present, mechanical devices are not part of the management of the vast majority of patients with failing RVs. With the exception of patients with infarction of the right ventricle, the most common causes of an RV limited circulation share the pathophysiology of elevated pulmonary vascular resistance.

- *Primary Pulmonary Hypertension*
- *Massive pulmonary embolism*
- *Recurrent thromboembolism*
- *Severe Obstructive Lung Disease, including COPD & Chronic Bronchitis*
- *Obstructive Sleep Apnea/Sleep Disordered Breathing*
- *Morbid Obesity*



RV Shock



Crisis

Some patients will continue to deteriorate in spite of escalating support. Anesthesiologists typically administer small boluses of 'CODE' drugs in such instances, a practice which is entirely appropriate. Other measures, which might be helpful in this setting, include:

- evaluation of the surgical procedure
- rapid trouble shooting of the anesthesia machine & circuit
- review of medications recently administered
- stat portable chest X-ray to R/O tension PTX
- stat echocardiogram to evaluate ventricular filling, ventricular function, valvular function, and exclude tamponade
- empiric therapy with an H1 and H2 blocker
- empiric therapy with replacement or stress doses of steroid.

In patients who have not been previously treated with steroids, 50mg of hydrocortisone IV and 50 micrograms of fludrocortisone po/ng is an appropriate dose.

- If therapy with catecholamines seems to make things worse instead of better, the possibility that the patient might have previously undiagnosed carcinoid should be entertained, and the circulation supported exclusively with volume and vasopressin.
- Small boluses of vasopressin (0.5 to 2 u IV) may work where catecholamines fail.

2. Arrest

Common Situations Associated with Peri-op Circulatory Crisis are listed below:

Anesthetic

- *Intravenous anesthetic overdose*
- *Inhalation anesthetic overdose*
- *Neuraxial block with high level sympathectomy*
- *Local anesthetic systemic toxicity*
- *Malignant hyperthermia*
- *Drug administration errors*

Respiratory

- *Hypoxemia*
- *Auto PEEP*
- *Acute Bronchospasm*

Cardiovascular

- *Vasovagal reflex*
- *Hypovolemic and/or hemorrhagic shock*
- *Tension Pneumothorax*
- *Anaphylactic Reaction*
- *Transfusion Reaction*
- *Acute Electrolyte Imbalance (high K)*
- *Severe Pulmonary Hypertension*
- *Increased intraabdominal pressure*
- *Pacemaker failure*
- *Prolonged Q-T syndrome*
- *Acute Coronary Syndrome*
- *Pulmonary Embolism*
- *Gas embolism*
- *Oculocardiac reflexes*
- *Electroconvulsive therapy*

Anaphylaxis

Anaphylaxis is a rare but important cause of circulatory collapse in the perioperative period. While there is a wide range of minor allergic reactions, hypotension, tachycardia and bronchospasm can be more easily followed by vasogenic shock when the offending agent is administered as a rapid intravenous bolus, the most common route of drug administration during anesthesia. The preponderance of anaphylaxis in perioperative patients is caused by a small number of drugs.

Anaphylactic shock has been identified as a coexisting or major indeterminate factor for dysrhythmic cardiac arrest during anesthesia occurring in 2.2 to 22.4 per 10,000 anesthetics with 3% to 4% of them being life threatening.

Common Causes:

- IV contrast
- Beta lactam antibiotics
- Latex
- Non-depolarizing neuromuscular blockers

The management of the patient with anaphylaxis consists of measures to interrupt the reaction and support the patient. Surgery should be interrupted when feasible and the patient should be immediately supported with IV fluid and vasopressors. It is imperative to remember that the Epinephrine administered to patients with anaphylaxis is intended to interrupt the reaction, and not support the circulation. Thus it should always be given and at the full recommended dose (0.01 mg/kg or approx 1mg in most adults).

Treatment

- Stop or remove the inciting agent or drug (e.g. IV contrast or latex)
- If feasible, stop surgery
- Oxygen at Flo₂ of 1.0
- Chest compression if no pulse detected for 10 seconds
- 1 mg Epinephrine IV
- ± 2 u Vasopressin IV
- IV fluids/large bore access
- H1 blocker (50 mg diphenhydramine IV)
- H2 blocker (20 mg famotidine IV)
- ± steroid (e.g. 50-150 mg hydrocortisone IV)
- a tryptase level in the blood can be used to confirm the diagnosis

Gas Embolism

Gas embolism remains an important cause of circulatory crisis and cardiac arrest in perioperative patients, and is likely to increase in frequency as greater numbers of procedures are performed utilizing minimally invasive techniques incorporating gas insufflation. While it is difficult to conduct systematic prospective human studies of this problem, there is a growing consensus among the experts reviewing these cases that resuscitative efforts should focus more on supporting a failing right heart, with less emphasis on attempts to remove the offending gas.

Causes

- Laparoscopy
- Endobronchial Laser procedures
- Central Venous Catheterization
- Hysteroscopy
- Pressurized Wound Irrigation
- Prone Spinal Surgery
- Posterior Fossa Surgery
- Pressurized Fluid Infusion

Presentation

- Bradyarrhythmias/Bradycardia
- Cardiovascular Collapse
- Loss of end-tidal carbon dioxide

Treatment

1. Administer 100% oxygen and intubate for significant respiratory distress or refractory hypoxemia. Oxygen may reduce bubble size by increasing the gradient for nitrogen to move out.
2. Promptly place patient in Trendelenburg (head down) position and rotate toward the left lateral decubitus position. This maneuver helps trap air in the apex of the ventricle, prevents its ejection into the pulmonary arterial system, and maintains right ventricular output.

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3. Maintain systemic arterial pressure with fluid resuscitation and vasopressors/beta-adrenergic agents if necessary. See the algorithm on pg.8 for RV failure.

4. Consider transfer to a hyperbaric chamber. Potential benefits of this therapy include (1) compression of existing air bubbles, (2) establishment of a high diffusion gradient to speed dissolution of existing bubbles, (3) improved oxygenation of ischemic tissues and (4) lowered intracranial pressure.

5. Circulatory collapse should be addressed with CPR and consideration of more invasive procedures as described above.

Hyperkalemia

Hyperkalemia can be an elusive but important cause of cardiac arrest in perioperative patients. Patients at greatest risk are those with end-stage renal disease or renal insufficiency requiring the transfusion of red cells, and any patient suffering massive hemorrhage. The management of unexpected cardiac arrest in these patients and in these settings should include treatment for hyperkalemia, with at the very least intravenous administration of calcium and bicarbonate.

It is important for practitioners to appreciate that the majority of patients who sustain a cardiac arrest do not seem to undergo the orderly deterioration of their cardiac rhythm as has been widely taught. Cardiac arrest from hyperkalemia can present as bradycardia, asystole, ventricular tachycardia, ventricular fibrillation, and PEA.

Complications of Central Venous Access

Pneumothorax is a well described and relatively rare complication of central line placement in perioperative patients. Most practitioners astutely suspect this complication in patients who become unstable after undergoing central venous cannulation. More recent analysis from the closed-claims database suggests that both hemo-pneumothorax and tamponade may be important and sometimes unrecognized fatal complications of patients who undergo attempts at central venous cannulation. In those instances where a patient deteriorates following central line placement, echocardiography should be considered in addition to chest radiography.

Anesthetic Techniques and Cardiac Arrest:

Local Anesthetics

Risk of local anesthetic toxicity is difficult to predict. In general, local anesthetics depress the heart in a dose dependent fashion. Amongst the local anesthetics in widespread clinical use, bupivacaine is the most potent myocardial depressant and most often associated with cardiac arrest. Fortunately, most awake patients who are developing systemic toxicity manifest CNS symptoms that alert their caregivers to the possibility of local anesthetic toxicity. In some unfortunate patients, these changes presage cardiac arrest.

Manifestations of local anesthetic toxicity

- ringing in the ears or buzzing in the ears
- metallic taste or peri-oral tingling
- dysphasia
- orthostasis
- confusion
- PVCs
- wide QRS complex EKG which can subsequently deteriorate into EMD/PEA or asystole (bupivacaine)
- bradycardia or atrioventricular block (lidocaine and etidocaine)

Treatment

- Stop the administration of local anesthetic
- CPR as indicated (pulseless for >10 sec)
- Epinephrine 1 mg IV (some experts recommend higher doses)
- Tracheal intubation and ventilation with 100% oxygen
- 20% intralipid, 1.5ml/kg IV load, then 0.25ml/kg/hr IV
- Sodium Bicarbonate to maintain a pH greater than 7.25 in patients who do not respond quickly
- Consider therapy with H1 and H2 blockers
- Consider transcutaneous or intravenous pacemakers for all bradycardic rhythms.
- Most importantly, **continue CPR for at least 60 minutes**, as very good neurologic recovery has been reported in patients after very prolonged cardiac arrests from local anesthetic overdoses.

Neuroaxial Anesthesia

Cardiac arrest in association with neuraxial (spinal or subarachnoid block) anesthesia remains the most mysterious cause of morbidity and mortality in the perioperative period. Its existence would be controversial, except that it has been well documented as an occurrence in younger, otherwise healthy patients undergoing a variety of clinical procedures. Its pathophysiology remains a mystery. Clinically, the only unifying feature of this syndrome is the degree of surprise among the caregivers of these patients. Various hypotheses have been put forward over the years, invoking unrecognized respiratory depression, excessive sedation concurrent with high block, under appreciation of both the direct and indirect circulatory consequences of a high spinal anesthetic, and 'failure to rescue' with airway management and drugs. Hypoxemia from hypoventilation does not appear to be the cause, as there are case reports documenting adequate saturation in these patients. Thus there is a substantial amount of basic science and clinical interest in the effects of high spinal anesthesia on the sympathetic innervation of the heart and the circulation.

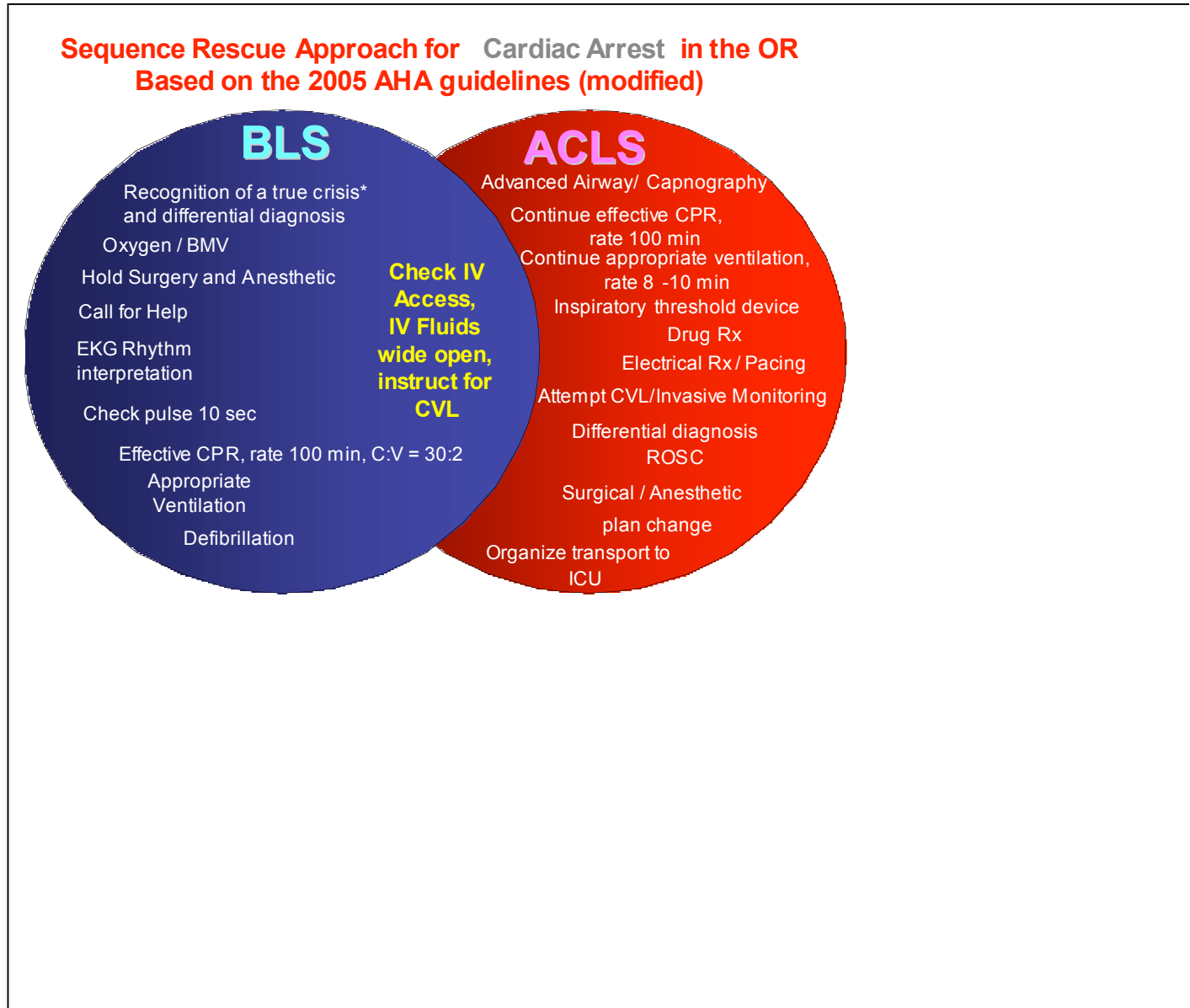
The most recent North American review of the epidemiology of cardiac arrest during neuraxial anesthesia indicates the prevalence of cardiac arrest at 1.8 per 10,000 patients, with more arrests occurring in patients with spinal anesthesia vs. all the other techniques (2.9 vs. 0.9 per 10,000 ; P = 0.041). In almost 50% of the cases cardiac arrest was associated with recurrent specific surgical events (cementing of joint components, spermatic cord manipulation, manipulation of a broken femur, and rupture of amniotic membranes).

The choice of vasopressors during neuraxial anesthesia is still being debated.

Treatment of Cardiac Arrest Associated with Neuraxial Anesthesia

- Discontinue anesthetic or sedation infusion
- Ventilate with 100% Oxygen, intubate trachea
- Begin CPR if patient has significant bradycardia or is pulseless >10sec
- Treat bradycardia with 1mg Atropine
- Treat with at least 1 mg epinephrine IV (up to 0.1mg/kg)
- Consider concurrent treatment with 40 u vasopressin

A treatment guideline based on BLS and ACLS sequence approach is offered
below (Fig 5)



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Recognizing cardiac arrest in the OR

- EKG with pulseless rhythm (V-tach, V-fib)
- loss of pulse X 10 seconds
- loss of end-tidal CO₂
- loss of plethysmograph

BLS/ACLS in the OR

- CPR for patients undergoing general anesthesia need not be preceded by “Annie!! Annie!! Are you Okay?”
- Instruct appropriate personnel to start effective CPR.
- Discontinue the anesthetic and surgery
- Call for help, defibrillator
- Bag mask ventilation if ETT not in place followed by immediate endotracheal intubation if feasible FiO₂ = 1.0
- Don't stop CPR unnecessarily. Capnography is a more reliable indicator of ROSC than carotid or femoral arterial pulse palpation.
- Capnograph to confirm advance airway positioning and effective CPR
- Hand ventilate rate 8 -10, V_T to chest rise, T_I one second with 100% oxygen – assess for obstruction, if none, institute mechanical ventilation. If obstruction, suctioning, fiberoptic bronchoscopy, consider exchanging the airway
- Open all IVs to wide open
- Blow in capnograph to confirm function

Initiating ACLS Protocols in the OR: Special Considerations

Recognizing that it is time to commence ACLS in the OR is more difficult than it might seem to outsiders for a variety of reasons.

First, false alarms vastly outnumber real events with OR monitors and monitor failure is more common than cardiac arrest in most operating rooms. By far, the most likely cause of ‘asystole’ on an EKG monitor in the OR is an electrode failure or lead disconnect. The operating room is a brutal environment, and the devices we use to monitor patients can fail from heavy use. It is likely that monitor failure is more common than cardiac arrest in most operating rooms.

Second, hypotension and bradycardia are relatively common occurrences in the OR, and most patients recover to an adequate hemodynamic status with minimal intervention.

Third, it can be difficult or impossible to obtain satisfactory monitoring in many patients (vasculopath, hypothermic, morbidly obese).

Ventilation

The growing appreciation of the deleterious effects of excessive positive pressure ventilation has led to the overall single most important change in the resuscitation guidelines for cardiac arrest: the change of the compression-to-ventilation ratio (C:V) to a universal 30:2 for single rescuers for victims of all ages (except newborns) and two-rescuer CPR for adult victims until an advanced airway device (ETT, LMA or esophageal airway) is inserted; and the recommendation to maintain a respiratory rate of no more than 10 breaths/minute with an inspiratory time of one second and a tidal volume limited to “chest rise” (approximately 500 ml in the adult) for intubated patients. The concern that a higher percentage of infants and children frequently develop cardiac arrest secondary to asphyxia has resulted in a more conservative approach to ventilation in that population, with a recommended C:V of 15:2 when two rescuers are available.

PRE-ARREST OR ALGORITHMS

A schematic overview of unstable pre-arrest dysrhythmias is illustrated below (Fig 6)

Pre-arrest Outline of ACLS in Perioperative Patients

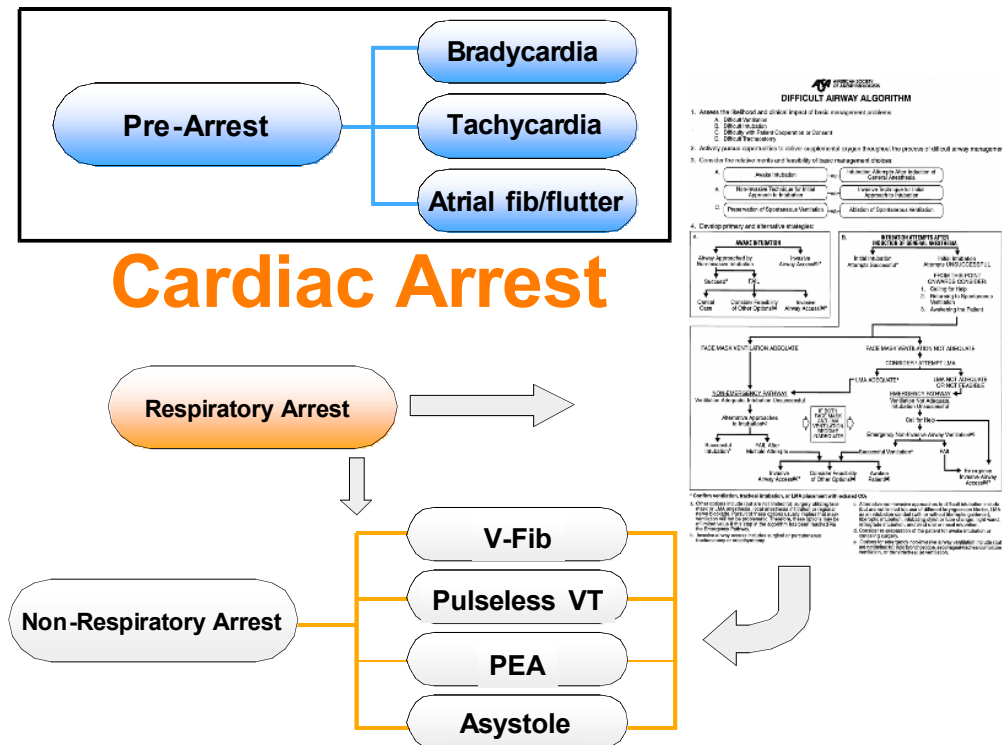


Fig. 6

Bradycardia

A comprehensive algorithm approach is offered below. (Fig 7)

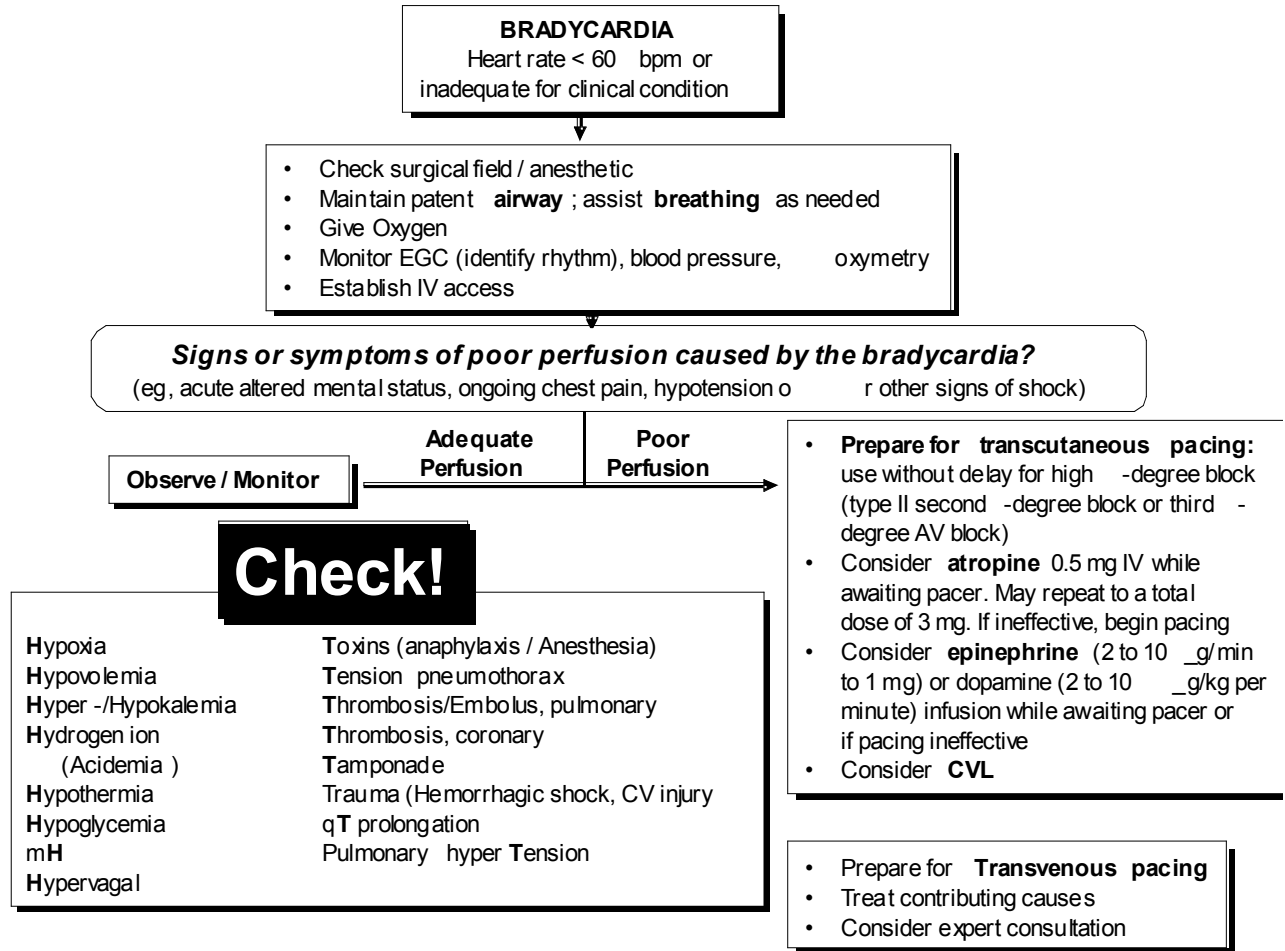


Fig 7: A comprehensive algorithm for treatment of perioperative bradycardia

The different spectrum of causes of bradycardia in the perioperative period makes it more reasonable to attempt pacing in this patient population than in most other settings.

Perioperative Bradycardia: Special Considerations

- Ensure adequate oxygenation via pulse oximetry if possible
- IV fluids wide open
- Initiate CPR for severe bradycardia, as meds will otherwise not reach the heart in a timely fashion.
- Atropine 0.5 mg IV, then Epi 1 mg IV (repeat q 1 minute)
- Call for transthoracic pacemaker and transvenous pacemaker immediately
- Esophageal pacing is a reasonable alternative to transvenous pacing
- Pacing, atropine, and Epi fail: Pace at maximal output (to ensure capture), asynchronous, and at 100 bpm
- Check ETCO₂ and plethysmograph tracing for adequate pacing
- Invasive blood pressure monitoring is appropriate in instances where palpation of the pulse is difficult

Indications for Emergency Pacing

- Hemodynamically symptomatic bradycardia unresponsive to positive chronotropic agents
- Pharmacologically unresponsive bradycardia with escape rhythms, drug overdose, acidosis or electrolyte abnormalities
- Standby for symptomatic sinus node dysfunction, Mobitz type II 2nd degree, 3rd degree, alternating BBB or bi-fascicular block
- Overdrive pacing of supraventricular or ventricular tachycardia refractory to Rx or electrical cardioversion

Tachycardia

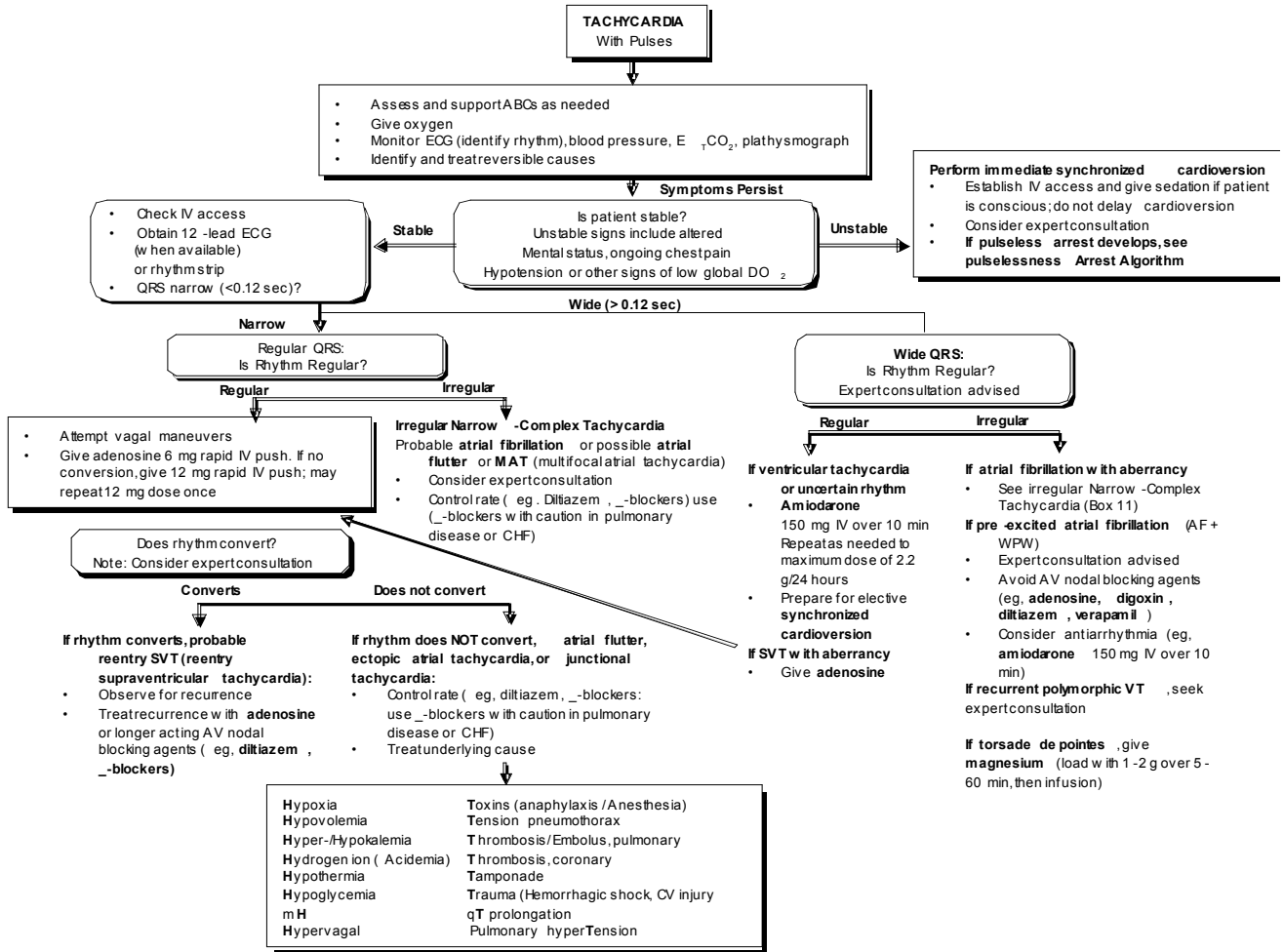


Fig 8: A comprehensive algorithm for treatment of perioperative tachycardia

- Attempt to diagnose the underlying rhythm
- Amiodarone is not always the best drug but it is rarely the wrong drug if the patient has a stable arrhythmia unless it is torsades
- Antiarrhythmics can act as proarrhythmic. So after 1 or 2 antiarrhythmics... Cardiovert. Patients with compromised heart function should not receive multiple antiarrhythmics and should be cardioverted early.
- Biphasic cardioversion is preferable to monophasic cardioversion.
- WPW? Use Amiodarone. Avoid Rx that blocks the AV node: adenosine calcium blockers, beta blockers, digoxin.
- Atrial fibrillation of unknown duration or which is greater than 48 hours old should not be cardioverted.
- Wide Complex Supraventricular Tachycardia is VT unless proven otherwise use amiodarone or procainamide

SVT:

- Narrow complex and irregular? Likely Afib: Amiodarone, cardiovert if unstable
- Narrow Complex and regular? Carotid sinus massage in appropriate patients, then adenosine 12mg IVP. If adenosine fails consider PSVT due to reentry (cardioversion responsive) or an automatic rhythm e.g. ectopic atrial tachycardia, multifocal atrial tachycardia, junctional tachycardia (cardioversion non-responsive, ventricular dysfunction)

Cardioversion: Special Considerations

- Immediate cardioversion is indicated for a patient with serious signs & symptoms related to the tachycardia or if ventricular rate is > 150 bpm (Table 2)
- Always be prepared to externally pace patients who are being cardioverted, as some will convert into a very bradycardic rhythm.

Rhythm	Energy Sequence Monophasic	Energy Sequence Biphasic
PSVT	50 J, 100 J, 200 J, 300 J, 360 J	100 j
A Flutter	50 J, 100 J	50 J
Atrial Fibrillation	200 J, 300 J, 360 J	50 J, 100 J

Table 2. Cardioversion energy sequence of unstable tachycardias

ARREST OR ALGORITHMS

A schematic overview of cardiac arrest pathophysiology is illustrated below (Fig 9)

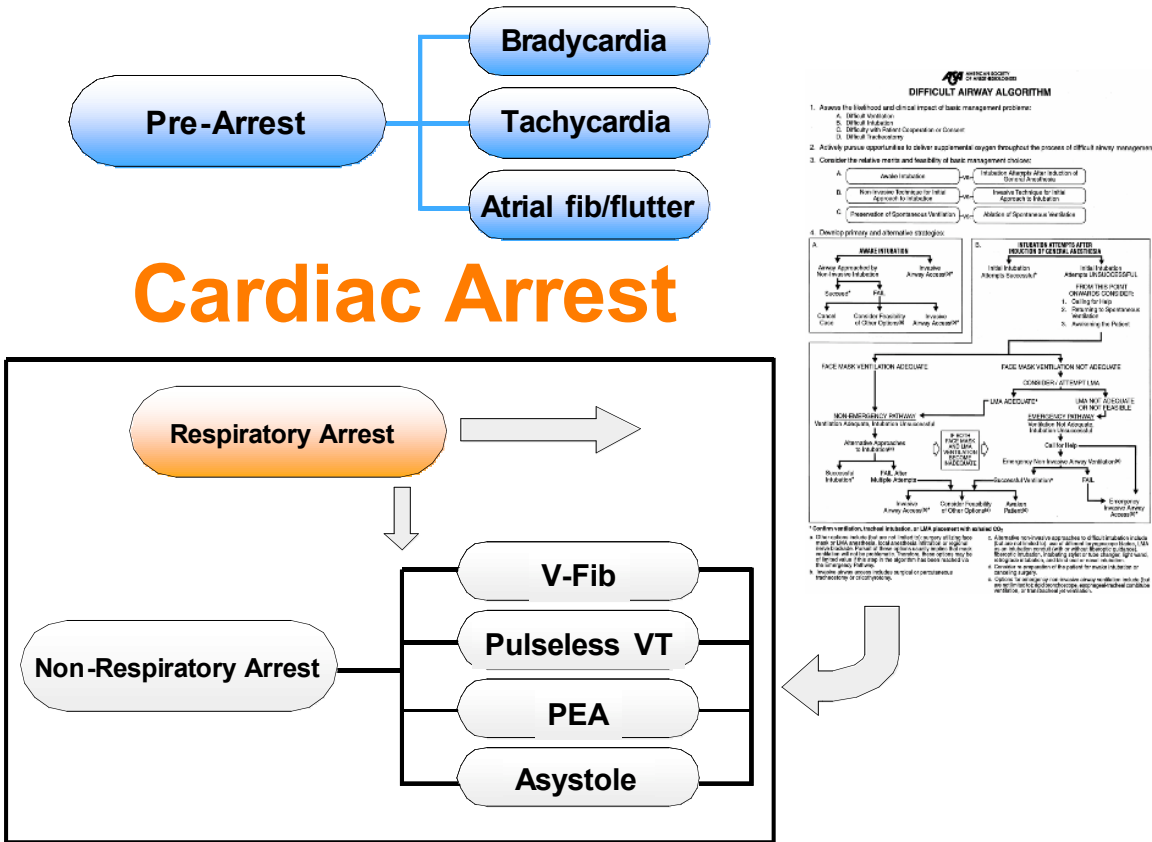


Fig 9 A schematic overview of cardiac arrest pathophysiology in the OR

Cardiac Arrest in the OR

A comprehensive algorithm approach is offered below (Fig 10), including a differential diagnosis of asystole and PEA in the OR (Table 3, 4, 5)

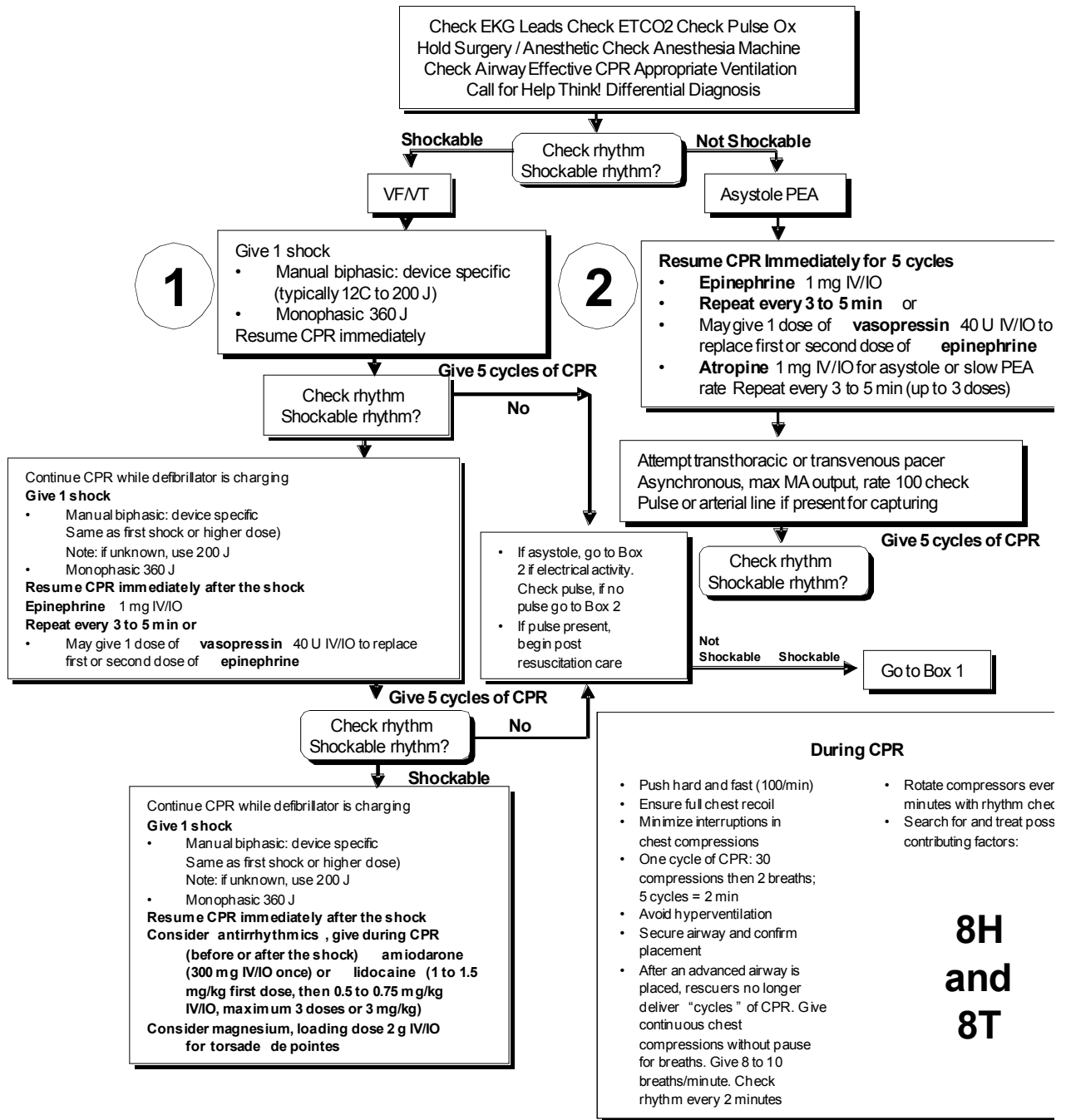


Fig 10: Cardiac arrest in the OR. A comprehensive algorithm from the 2005 AHA guidelines (modified)

Cardiac Arrest in the OR: Special Considerations

- Wide open IV crystalloid without glucose unless hypoglycemia suspected.
- Ventilate by hand or anesthesia machine 10 min V_T 5-7 ml/kg confirmed by spirometry or chest rise asynchronous with chest compressions
- Surgeon stand-by for possible open chest cardiac massage

Differential Diagnosis for perioperative PEA or Asystole: 8H & 8T	
Hypoxia	Trauma/hypovolemia
Hypovolemia	Tension Pneumothorax
Hyper-vagal	Thrombosis of Coronary
Hydrogen Ion	Tamponade
Hyperkalemia	Thrombus in Pulmonary Artery
Malignant Hyperthermia	Long QT syndrome
Hypothermia	Toxins (anaphylaxis)
Hypoglycemia	Pulmonary HTN

Table 3. Differential Diagnosis for perioperative PEA or Asystole

Causes of PEA arrest and the EKG rhythm that most often precedes them	
Cause of PEA	Pre Arrest Rhythm
Hypovolemia	Narrow complex tachycardia or bradycardia
Hypoxia	Bradycardia
Auto-PEEP	Narrow complex Tachycardia that devolves into bradycardia
Vaso-Vagal	Bradycardia, sometimes associated with peaked T waves
Anaphylaxis	Tachycardia
Tension Pneumothorax	Narrow complex tachycardia, then bradycardia
Tamponade	Narrow complex tachycardia
RV arrest	Narrow complex tachycardia which devolves into bradycardia
PA HTN	RBBB, RV strain
Coronary Syndrome/LV arrest	Q waves, ST depression followed by ST elevation, VT, Vfib
Hyperkalemia	peaked T waves, widened QRS, sine wave wide complex PEA
Hypoglycemia	Narrow complex tachycardia
Hypothermia	J or Osborne Waves
Long QT syndrome	Bradycardia
Hypokalemia	Flattened T waves, prominent U waves, widened QRS, prolonged QT, wide complex tachycardia

Table 4. Causes of PEA arrest and the EKG rhythm that most often precedes them

EKG Ventricular Complex Size and the PEA associated with it

<u>Narrow Complex</u>	<u>Wide Complex</u>
Non-Cardiac Causes	Cardiac Causes
e.g. hypovolemia, vasodilation	e.g. drugs and toxins

Table 5. EKG Ventricular Complex Size and the PEA associated with it

Ventricular Tachycardia and Torsades-de-Pointes

Monomorphic vs Polymorphic VT

Monomorphic VT:

- Normal LV function: amiodarone, lidocaine or procainamide
- Impaired LV function: amiodarone, lidocaine

Polymorphic VT:

- Usually self limiting but if recurrent then Mg 2 gm IV,
- Defibrillate if unstable
- Rx usually after spontaneous termination and is directed at preventing the recurrence. If ongoing and unstable go to pulseless VT/VF algorithm
- Prolonged QT? then Mg 2 gm IV
- Not prolonged? See monomorphic VT
- Torsade de pointes: Mg 2 gm, defibrillate if unstable

Ventricular Fibrillation

Defibrillation for Adult and Pediatric Patients

Biphasic Defibrillators are supplanting Monophasic defibrillators in most instances as they are likely more effective, and utilize lower energies in every clinical instance.

<u>Clinical Situation</u>	<u>Biphasic Energy</u>	<u>Monophasic Energy</u>
Ventricular Tachycardia	150 Joules, synchronized	300-360 joules, synchronized
Ventricular Fibrillation	150-200 joules	300-360 joules

Patients who fail to respond to their first round of attempts at cardioversion or defibrillation may respond to the following medications:

<u>Rhythm</u>	<u>1st line</u>	<u>2nd line</u>	<u>3rd line</u>
Ventricular Tachycardia	Amiodarone	Lidocaine	Procainamide
Ventricular Fibrillation	Amiodarone		
Torsades	Magnesium		

Usage:

Amiodarone: 300 mg IV push, then 150 mg IV push, repeat q 5 minutes up to 1.5 grams total.

Infusion rate: 1 mg/min

Lidocaine: Load 1.0-1.5 mg/kg push. Repeat 0.5 to 0.75 mg/kg 3- 5 min later.

Max dose 3 mg/kg

Infusion range: 1-4 mg/min

Magnesium Sulfate: 2 grams IV for Torsades, Hypo Mg or K. May repeat x3.

Procainamide: 20-50 mg/min IV. Max: 17mg/kg.

Infusion rate: 1-4 mg/min

VT, VFib and TdP: Additional Considerations

- Do NOT use adenosine, calcium channel blockers, beta blockers or digoxin
- Consider atropine (1mg IV), epinephrine (1mg IV) and vasopressin (40 U IV) for patients with cardiac arrest associated with neuraxial anesthesia. Repeat q 1 minute x 3 FAST. Do Not Wait for 5 CV cycles.
- Hyperkalemia is a frequently unsuspected cause of a wide complex tachycardia. Consider treating at risk patients with:
 - 1 gram CaCl IV push (typically 10 cc of 100mg/ml)
 - 100 meq NaHCO₃ (2 x 50 cc ampules)
 - 10 units of regular insulin IV and 25 grams of dextrose IV (1- 50cc ampule of D50%w).
- Consider empiric treatment for tension pneumothorax and sub-xiphoid pericardiocentesis in patients at risk for these complications

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Appendix 1: Recommended Infusions

Infusion	Infusion range	Mixed as	mid-range rate(ml/hr for 80 kg pt)	
Dobutamine	1-20 mcg/kg/min	1g/250ml	12	(10 mcg/kg/min)
Norepinephrine	0.05 – 0.5 mcg/kg/min	16mg/250ml	7.5	(0.1mcg/kg/min)
Epinephrine	0.05-0.2 mg/kg/min	16mg/250ml	7.5	(0.1mcg/kg/min)
Dopamine	1-20 mcg/kg/min	400mg/250ml	30	(10 mcg/kg/min)
Vasopressin (AVP – Pitressin)	5-80 milliunits/min	20Unit/100ml	12	(40 milliUnits/min)
Phenylephrine	0.1-3 mcg/kg/min	20mg/250ml	60	(1 mcg/kg/min)
Nitroprusside	0.3-10 mcg/kg/min	50mg/250ml	24	(1mcg/kg/min)
Fenoldopam	0.05-0.3 mcg/kg/min	4mg/100ml	12	(0.1 mcg/kg/min)
Amiodarone	1 mg/minx 6 hr, then 0.5 mg/min	720mg/500ml	20.8	(0.5 mg/min)
Lidocaine	1 mg/min	2g/250ml	7.5	
Milrinone	Load: 50 mcg/kg over 10 min Then 0.375 -0.75 mcg/kg/min	20mg/100ml	12	(0.5 mcg/kg/min)

Appendix 2: CODE drugs

Drug	Formulation	Dose/adminstration
Epinephrine	varies: 1/1000 and 1/10,000	1 mg IV push repeat q 3 min
Vasopressin (Pitressin)	20u/1ml	40 units
Atropine	varies (typically 0.4mg/ml)	1 mg
Adenosine	varies	6mg IVP & 20cc NS may then Rx with 12 mg IVP & 20cc NS qmin x 2
Amiodarone	300 mg in 20-30 ml D5	300 mg IV push, repeat w/ 150 mg IV push in 3 min
Digoxin	0.25mg/ml or 0.1mg/ml in 1 or 2 ml ampule	load 10-15 mcg/kg IBW
Esmolol	varies	0.5 mg/kg over 1 minute then 50 mcg/kg/min for 4 minutes May repeat bolus x1 and increase rate to 100 mcg/kg/min
Lidocaine	varies (typically 100mg in 5cc)	1-1.5 mg/kg – 100 mg IVP
Magnesium sulfate	varies	1-2 grams IV over 5 minutes
Calcium chloride	100mg/ml in 10 ml	500-2000 mg IV push
Bicarbonate Sodium	50 milliequivalents in 50 ml	50 meq IV push

Appendix 3: Resuscitation of the Pregnant Woman in Cardiac Arrest

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There are two potential patients when a pregnant woman arrests, the mother and the fetus. The best hope of fetal survival is maternal survival. The most common pregnancy-related causes of maternal deaths are hemorrhage (hypovolemia), embolism (venous or amniotic fluid), preeclampsia/eclampsia, cardiomyopathy, and cerebrovascular accident. Morbidity and mortality also result from deterioration of medical conditions during pregnancy (e.g., cardiac disease, lupus, diabetes, asthma), coincidental medical conditions (e.g., malignancies, morbid obesity), and automobile accidents, suicide and homicide. The anesthesia team may be called to resuscitate pregnant women in any of these circumstances.

When performing BLS/ACLS, rescuers must understand the anatomic, mechanical and physiologic changes caused by pregnancy. Multiple gestations are associated with greater changes and correspondingly less reserve when crises occur. Hypoxia, hypercapnia and acidosis develop extremely rapidly during pregnancy because of decreased FRC, increased O₂ demand/CO₂ production and decreased buffering capacity. Difficult intubation and pulmonary aspiration often complicate resuscitation efforts because of suboptimal positioning, airway edema, gastroesophageal reflux and lack of the usual prophylactic measures. **Most important, after 20 weeks of gestation the pregnant uterus compresses the inferior vena cava and aorta when the mother is supine, markedly decreasing venous return and cardiac output.** This can cause prearrest hypotension or shock and in the critically ill patient may precipitate arrest. During cardiac arrest, aortocaval compression can impede venous return and cardiac output such that cardiac compressions are ineffective. Displacing the uterus 15-30° by tilting the patient or displacing the uterus laterally may alleviate obstruction in the prearrest situation. **During cardiac arrest, resuscitation may prove impossible until the fetus is delivered.**

BLS/ACLS for the Pregnant Woman

The recommendations in the Anesthesia Advanced Circulatory Life Support with respect to intubation and ventilation, medications (including epinephrine, vasopressin and dopamine) and defibrillation doses apply equally to the pregnant patient. In addition, the following modifications to BLS and ACLS are appropriate:

- Displace the uterus 15-30° by placing the mother on her left side, placing a wedge under her right hip or by manually moving the uterus laterally.
- Secure the airway early using a smaller than usual endotracheal tube and cricoid pressure (if possible).
- Perform chest compressions slightly above the center of the sternum to adjust for the elevation of the diaphragm
- Defibrillate using standard ACLS doses but remove fetal or uterine monitors before shocking.
- Do not use the femoral vein for administration of medications, as there may be no effective flow until the fetus is delivered.
- At gestational age <20 weeks, urgent Caesarean delivery need not be considered, because a gravid uterus of this size is unlikely to significantly compromise maternal cardiac output.
- At gestational age approximately 20—23 weeks, initiate emergency hysterotomy to enable successful resuscitation of the mother, not survival of the delivered infant, which is unlikely at this gestational age.
- After 20-24 weeks gestation, perform immediate hysterotomy (cesarean delivery) within 5 min of cardiac arrest if no response to BLS and ACLS to enable successful resuscitation of the mother and fetus .
- For cardiac arrest secondary to hemorrhagic shock (ectopic pregnancy, placental abruption, placenta praevia and uterine rupture) consider the following additions to the resuscitation protocol:
 - a. oxytocin and prostaglandins to correct uterine atony
 - b. uterine compression sutures
 - c. radiological embolization of uterine blood supply
 - d. hysterectomy
 - e. aortic cross-clamping

Providers should try to identify reversible pregnancy-specific and incidental causes of cardiac arrest when deciding whether to proceed to cesarean delivery. For example, spinal hypotension is often treatable with oxygenation, ventilation and aggressive use of vasopressors, including epinephrine or vasopressin (see neuraxial anesthesia section above), eclamptic seizures may be self-limiting, and magnesium overdose should respond to one or repeated doses of calcium chloride, 1 gm iv. In contrast, the cardiac failure after amniotic fluid embolism (also called anaphylactoid syndrome of pregnancy) rarely resolves over the course of several minutes. Inotropic support is often required and patients who survive the first minutes typically have uterine atony and a consumptive coagulopathy.

Emergency Hysterotomy (Perimortem Cesarean Delivery)

Aorticaval compression from the gravid uterus may result in no venous return and therefore no response to BLS/ACLS. In this situation, immediate delivery (within 4-5 min) may prove life saving to the mother. Beneficial changes after delivery include immediate relief of aorticaval compression with consequent improved venous return and cardiac output, improved pulmonary mechanics, and decreased oxygen demand. Also, the fetus of ≥ 24 weeks gestational age has the best chance of intact survival when delivery occurs less than 5 min after maternal cardiac arrest. The American Heart Association's 2005 guidelines state that when maternal cardiac arrest is not immediately reversed by BLS and ACLS: ***"The resuscitation leader should consider the need for an emergency hysterotomy (cesarean delivery) protocol as soon as a pregnant woman develops cardiac arrest."*** They further emphasize: ***"... you will lose both mother and infant if you cannot restore blood flow to the mother's heart. Note that 4 to 5 minutes is the maximum time rescuers will have to determine if the arrest can be reversed by BLS interventions. The rescue team is not required to wait for this time to elapse before initiating emergency hysterotomy."*** When uterine size corresponds to < 20 weeks gestational size, immediate delivery may not be indicated. Between 20 and 23 weeks (before fetal viability) it is likely to benefit only the mother; after 24 weeks it may benefit both mother and fetus. Even when delivery cannot be accomplished within 5 minutes, performing it as soon as possible usually will confer maternal benefit. In a 20-year review of maternal cardiac arrests by Katz et al., maternal pulse and blood pressure returned after CD in 12 out of 18 cases in which hemodynamic status was reported; in no case was there deterioration of maternal condition.

To optimize the chance of maternal survival with good neurologic outcome, advance preparations designed to facilitate urgent CD in non-operating room locations are necessary. Transferring a patient to an operating room during BLS/ACLS (rather than performing CD on-site) is logistically challenging and time-consuming, will almost certainly result in interruption of chest compressions and monitoring, and overall probably will decrease maternal and fetal survival. Plans for performing emergency CD should be made in collaboration with obstetric, anesthesia, neonatal, and nursing personnel to determine what is feasible in that particular institution.

Appendix 4: Pediatrics - forthcoming

Appendix 5: Epidemiology and Pathophysiology of ACLS in the perioperative period.

The epidemiology of cardiac arrest in the anesthesia world is unique and special. In fact hypoxemic or dysrhythmic cardiac arrest is rarely observed when sedation, regional or general anesthetics are provided. There are also intuitive differences in a patient's chance of survival when the health care provider has prior knowledge of a his medical history, is instantly aware of cardiovascular and respiratory vital sign changes, can immediately recognize the probable cause of arrest and begins medical management within seconds.

Cardiac arrest during anesthesia has been labeled a rare event. In fact, the development of better monitoring, safer medications, adoption of clinical standards, and advances in knowledge and training have all had a significant impact on patient safety. Despite this, cardiac arrest during anesthesia still occurs but with prompt recognition, diagnosis, and treatment can be successfully managed.

The most recent data of cardiac arrest during anesthesia comes from the Mayo Clinic in Rochester. Cardiac arrest was defined as the requirement for resuscitation with either closed chest compression or open cardiac massage, after the onset of anesthesia. Cardiac arrests after transport to the ICU were not included. The two outcome variables were survival of at least one hour after initial resuscitation and survival to discharge from the hospital. All probable causes of cardiac arrest were grouped into three categories: 1) intraoperative hemorrhage, 2) permanent cardiac cause and 3) hypoxia, both at intubation or extubation. Overall 24 cardiac arrests were determined to be attributed to anesthesia (0.5/10,000 anesthetics).

If one extrapolates this number to the 20 million anesthetics performed annually in the United States, it translates to at least 1000 patients/year, or about three patients a day going from "sleep" to cardiac arrest!

This number is probably a gross underestimation since the many prestigious academic institutions in the US and abroad that report their experiences do not necessarily reflect the incidence of this problem in the "real world," i.e. outside academic boundaries or abroad.

The impact on favorable outcome of having an anesthesiologist present or immediately available during a surgical procedure is clear. In a large retrospective review, the adjusted alteration for death and failure to rescue were greater when care was not directed by a physician anesthesiologist (alteration for death = 1.08, $p < 0.04$; alteration for failure to rescue = 1.10, $P < 0.01$), suggesting that anesthesiologist-directed anesthesia care has a significant positive effect on the outcome for cardiac arrest, and long term mortality.

Appropriate vigilance and monitoring is often the key to recognition and timely response to such a crisis. For example, in the late 80s the ASA closed claimed study reported that 57% of hypoxia related deaths could have probably been

avoided simply by a better awareness life threatening respiratory complications during anesthesia and the use of pulse oximetry and capnography.

Asphyxial Cardiac Arrest

Respiratory complications and their variations have been described as an important cause of cardiac arrest and death during anesthesia, but the information became widespread only when, at the end of the 1980s, new confidentiality agreements between scientist and government institutions allowed the development of a massive database on anesthetic deaths.

For all anesthesiologists, hypoxia as a main cause of cardiac arrest can be more frequently seen within the context of a “cannot intubate – cannot ventilate” scenario. Hypoxia during anesthesia occurs in airway management failure such as misplacement of the endotracheal tube (esophageal or endobronchial; accidental tracheal extubation) aspiration of gastric contents, laryngospasm mainly due to mechanical irritation during inadequate depth of anesthesia, severe bronchospasm because of anaphylactic or intrinsic reactions, and errors in providing oxygen supply (hypoxic gas mixture).

Failure of adequate ventilation was observed in the 80s in about 35% of the cases of cardiac arrest and has continued to increase in the 90s, when the American Society of Anesthesiologists started recording nationwide insurance claims for major anesthesia complications reported voluntarily. In spite of the limitations of voluntary reports, the claims confirmed the unrecognized difficult airway as a major cause of cardiac arrest in approximately 25% of the cases.

In the most recent review, airway and ventilation-related cardiac arrests, both at intubation or extubation, amounted to approximately 45% of all cases. In this series 24 cardiac arrests were directly attributed to anesthesia management.

Irreversible hypoxic or ischemic brain damage is the clinical, most crucial consequence when - at normothermia - the brain is not receiving adequate oxygen delivery for more than five to seven minutes during cardiac arrest. Hypoxic brain damage can be an unexpected finding following prolonged hypotension (low global oxygen delivery) or inadvertent administration of an unrecognized hypoxic gas mixture.

The electrophysiologic aspect of cardiac arrest during hypoxemia is unique. An initial brief sympathetic stimulation aggravated by preexisting hypercarbia when present, is followed by severe bradycardia. Increased serum potassium, acute metabolic (lactic) and respiratory acidosis potentiates the cardiovascular depressant effect of the anesthetic, if present at the time of the hypoxic event. The result of this cascade if left uncorrected is asystole or more rarely PEA potentiated by vagal stimulation and increased serum potassium. If this vicious cycle is recognized and hypoxia is corrected in a timely manner, the process can be successfully reversed.

During the first few minutes of dysrhythmic adult cardiac arrest ventilation is not fundamental to restore spontaneous circulation and has been somewhat de-emphasized in the new AHA guidelines in favor of more effective chest

compressions. The attention of the rescuer of a hypoxemic a cardiac arrest is now focused on providing hemodynamic support while attempting to re-establish oxygenation and *normal* ventilation. Since the most frequent evolution of severe hypoxia is an unshockable rhythm, immediate pharmacological support with epinephrine is fundamental while attempting to secure the airway.

Furthermore, when the patient is in full arrest and an ETT is in place resulting in an unobstructed airway a small amount of tidal volume is exchanged, estimated to about 50 ml for compression. At a frequency of 100 compressions per minute, “involuntary” minute ventilation by simply compressing the chest would approximate 4.5 liters. Unfortunately, the effect of increased dead space ventilation on these small and frequent volumes cannot be easily anticipated.

When asphyxia is clearly the cause of cardiac arrest, such as in a “witnessed no ventilation no intubation scenario or in most of the pediatric population, oxygen consumption has proceeded to near complete exhaustion, and carbon dioxide and lactate have significantly accumulated just before cardiac arrest. In these cases, oxygen content of the tissues is minimal and providing ventilation with an FiO₂ of 1.0 is essential for survival.

Animal studies of asphyxial cardiac arrest (clamping of the endotracheal tube in an anesthetized pig with preexisting good oxygenation) showed that the arterial partial pressure of oxygen is maintained within the normal range for only approximately one minute in a model of chest compressions without ventilation and more importantly that return of spontaneous circulation was noted only when ventilation was added to chest compression.

This is in contrast to ventricular fibrillation, in which hypoxemia and acidemia become significant only several minutes after the onset of cardiac arrest.

However, positive pressure ventilation comes as a tradeoff of venous return during low flow states, including cardiac arrest and ventilation needs to be “matched” to the current lung perfusion.

When systemic blood flow decreases, lung perfusion decreases. In a low flow state, with less venous CO₂ delivered to the lungs, less is available for elimination via exhalation and the concentration of CO₂ in exhaled gas decreases. Because CO₂ elimination is diminished, it accumulates in venous blood and in the tissues. Mixed venous PCO₂ thus reflects primarily systemic and pulmonary perfusion and is an indicator of the tissue acid-base environment.

Positive pressure ventilation produces positive intrathoracic pressure during inspiration, reducing venous return to the chest and, as a result, reducing cardiac preload and subsequent cardiac output. For a given airway pressure, pleural pressure transmission of positive pressure ventilation increases when the lung is more compliant and when the chest wall is rigid. Furthermore, airway pressure pleural transmission increases on a number of variables including inspiratory flow rate and time, tidal volume, ventilation rate and degree of intrinsic positive end-expiratory pressure (auto-PEEP).

During low flow conditions, arterial PCO_2 and PO_2 reflect primarily the adequacy of alveolar ventilation. In these conditions, if alveolar ventilation is excessive, blood flowing through the pulmonary capillary bed is over-ventilated resulting in a large ventilation-perfusion mismatch. Adequate ventilation with an advanced airway in place and an F_iO_2 of 1.0 include a respiratory rate of 8 -10/min, V_T to chest rise (usually 5-7 ml/Kg), T_I of one second. The immediate consequence of unnecessary hyperventilation during cardiac arrest is further decrease of preload, coronary and cerebral perfusion without significant change of the acid base balance.

Local Anesthetic Toxicity

- Local Anesthesia

Local anesthetic toxicity is often unpredictable since the administration of the drug can result either in local constriction or systemic dilatation depending on the dose. Systemic effect can also vary, but in general the principal toxic effect is the result of cardiac depression or dysrhythmia. Local anesthetics affect either fast or slow calcium channels. While this latter characteristic has been debated for years, a study shows that in general all local anesthetics have a drug specific negative inotropic effect. When local anesthetic toxicity is studied in animal myocardial preparations, bupivacaine is associated with the most severe depression of cardiac conduction suggesting an extensive block of cardiac sodium channels as principal etiology of its cardio toxicity.

Bupivacaine exhibits a higher potency than the average anesthetic possibly by inhibition of myocardial energy metabolism in several ways, including blockade of the respiratory chain, inhibition of ATPases, uncoupling of oxidative phosphorylation and inhibition of ATP-ADP translocation.

Clinically, systemic toxicity from local anesthetic overdose can be subtle and nonspecific. Initial unexplained dysrhythmias such as uni or multifocal PVCs and mild neurocognitive dysfunction and "auras" of tinnitus, metallic taste, or dysphasia, might be followed by generalized seizure activity. The specific EKG rhythm feature of bupivacaine is widening of the QRS complex preceding a malignant ventricular dysrhythmia, typically electromechanical dissociation or asystole. Lidocaine and etidocaine more often progress to severe bradycardia or atrioventricular block. Rarely, local anesthetic toxicity leads to unidirectional block and re-entry, which in turn can produce ventricular tachycardia and fibrillation.

- Neuraxial anesthesia

It has been said that more than 50% of episodes of cardiac arrest during regional anesthesia could be avoided if inadequate ventilation were expeditiously recognized and avoided.

A nationwide study of closed insurance claims for major anesthetic mishaps has been retrieved from the database of the American Society of Anesthesiologists closed claim study, a project of the American Society of Anesthesiologists committee on professional liability.

Interesting clinical trends were revealed. In each single case, the event was unexpected, the patient ASA status was low and the outcome was, in general, poor.

In 30% of the 14 cases reviewed, a spinal anesthetic was chosen in an emergency procedure. The use of tetracaine seemed to be the most commonly associated with cardiac arrest. Most of the anesthesiologists involved in these cases were reasonably experienced. Despite the obvious selection bias of these self reported cases, the following were common features of patients suffering cardiac arrest: 1. The use of intraoperative sedation to achieve deep, sleep-like sedation to a level the patients would not vocalize; a combination of opioids, benzodiazepines and hypnotic agents was commonly the drug cocktail of choice. 2. Cardiac arrest was detected on a range of 5 to 25 minutes after the last administration of drug. 3. Often, cardiac arrest was preceded by a few minutes of unexplained and under treated progressive tachycardia and hypotension. 4. Cyanosis was noticed in a majority of the cases inferring that a mechanism of respiratory depression was added to the sympathetic blockade with the highest documented sensory level of T4 ± 1. When blood gases were available during an arrest, hypoxemia was confirmed, although immediately corrected by endotracheal intubation. 5. While immediate placement of an advanced airway was achieved in most of the patients, CPR seemed to have been delayed several minutes after the probable arrest. 6. Ephedrine was the most common first choice of vasopressor used to allow recovery of heart rate and blood pressure, mostly with minimal therapeutic success. 7. The administration of a more powerful direct catecholamine such as epinephrine averaged five minutes after the initial diagnosis of arrest.

To summarize the above observations, despite the presence of an anesthesiologist immediately available and a clear relationship between the anesthetic management and the cardiac arrest, the recognition of the crisis was in general late and the treatment not very effective. The result was a surprisingly poor neurological recovery, with only four patients regaining consciousness but with various degrees of cognitive dysfunction. It can be easily speculated that hypoventilation induced by concurrent use of opioids, benzodiazepines or hypnotics could have expedited a sympathetic blockade produced by the high spinal anesthesia and that the anesthesiologist's level of awareness of this combination was low.

Since the introduction of pulse oximeters, a few episodes of cardiac arrest have been documented with normal saturation readings and thus impossible to explain using the hypoxia theory. Therefore, alternative mechanisms to the hypoxia/hypercarbia theory should occasionally be considered. However, the lack of early recognition of a high level of neuraxial block in a patient silent and sedated, combined with delayed administration of direct acting catecholamines have been identified as typical patterns in the development of cardiac arrest. The mechanisms behind circulatory collapse during central neuraxial blockade or "total spinal anesthesia" have been recently reviewed.

In spinal and epidural anesthesia preganglionic efferent sympathetic nerve fibers are blocked. When the autonomic sympathetic fibers of the heart are denervated at the T1-T4 level, the release of endogenous catecholamines is blunted by blockage of the efferent sympathetic adrenal medulla fibers from T5-L2. This results in vasodilatation of the venous and arterial side and uncompensated sympathetic blockade of the adrenal medulla. Vagal influence on the heart becomes predominant. Overall the major determinate of severe hypotension during central neuraxial blockade is the decrease in venous return, with venous pooling occurring mostly in the splanchnic circulation. Pre-existing low heart rate by virtue of a healthy physical status or the

use of negative chronotropic agents may lead to severe bradycardia and asystole, without clear correlation between the severity of bradycardia and the level of blockade.

Several other factors may acutely decrease the heart rate. They include a decreased stimulation of receptors located in the atria and the venoatrial junction (a “reversed” Bainbridge phenomenon), direct stretching of the sinoatrial node from atrial emptying and increased non-myelinated vagal nerve afferents firing. The left ventricular wall stretch sensor is also reacting to decreased stretch, a phenomenon still debated in originating severe bradycardia and known as Bezold-Jarisch reflex. While all neuraxial anesthesia techniques have been described in the context of cardiac arrest, spinal anesthesia has clearly the worst track record.

In animal studies, cardiac arrest induced by high spinal anesthesia seems to respond best to a higher dose of epinephrine. Spinal anesthetic blunting of the neuroendocrine response to catecholamines during cardiac arrest is believed to contribute to the need for a high dose of epinephrine.

An initial intravenous administration of epinephrine of 1 mg can be supplemented by escalating bolus doses up to a total of 0.1 mg/kg. The use of a higher dose than 1 mg of epinephrine, while anecdotally associated with good recovery, needs to be carefully evaluated in view of its possible deleterious effect on the myocardium. Animal studies have shown that epinephrine can increase myocardial oxygen consumption, ventricular rhythm, ventilation perfusion mismatch, and post-myocardial dysfunction, all undesirable adverse effects during resuscitation in anesthetized patients.

Furthermore, in an animal model of epidural neuraxial anesthesia where V fib was induced by electrocution, a single dose of vasopressin appeared immediately comparable to mega dose epinephrine achieving better organ perfusion (heart and brain) at five minutes. While this is an interesting observation, it cannot be easily generalized to the common scenario described in humans, where cardiac arrest under neuraxial block is typically a unshockable rhythm. Therefore, the potential beneficial effect on a “high spinal” anesthetic has never been tested. The end tidal CO₂ can be used as a marker of successful resuscitation. An end tidal CO₂ of less than 10 mmHg, despite an appropriate dose of epinephrine during CPR, correlates with poor outcome.

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