ACLS in the Perioperative Period (AACLS)

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This refresher course is based on the monograph: *Anesthesiology Centric ACLS* by Andrea Gabrielli, Michael F. O’Connor, and Gerald A. Maccioli. It is an approved work product of the ASA’s Committee on Critical Care, and available at:  
http://www.asahq.org/clinical/Anesthesiology-CentricACLS.pdf

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. This can be attributed to the development of better monitoring, safer medications, adoption of clinical standards and advances in training. With detailed knowledge of the patient’s medical history, there is an intuitive difference in a patient’s chance of survival. When cardiac arrest during anesthesia does occur, prompt recognition, and diagnosis can lead to successful management.

The most recent data of cardiac arrest during anesthesia comes from the Mayo Clinic in Rochester (See Sprung J, et al. 2002). In this study, cardiac arrest was defined as the requirement for resuscitation with either closed chest compression or open cardiac massage, after the onset of anesthesia in 518,294 patients. 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) pre-existent cardiac pathology and 3) hypoxia, both at intubation or extubation. Overall 24 cardiac arrests were determined to be secondary 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 complications in the OR, and long term mortality (see Silber, et al 2000). 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 Claims study reported that 57% of hypoxiarelated deaths could have probably been avoided simply by improved awareness of life threatening respiratory complications during anesthesia and the use of pulse oximetry and capnography.

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 settings. Consequently, aggressive measures taken to support patient 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, as a cause of myocardial ischemia, is far more common than transmural infarction from plaque rupture. Intraoperative myocardial ischemia resulting from an imbalance in O2 delivery and consumption rarely
evolves to full pump failure or ventricular fibrillation in the operating room. The result is a different spectrum of dysrhythmias in the operating room than in the ED. 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.

Changes in ACLS
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/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. Recent versions of the ACLS guidelines have recommended lower levels of ventilatory support. This is the rationale driving the development of technologies to ventilate patients using negative pressure.

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 pac patients who are being cardioverted, as some will convert into a very bradycardic rhythm.
- Biphasic defibrillators are more effective and utilize less energy than monophasic defibrillators. Thus biphasic defibrillators have almost completely replaced the monophasic defibrillators discussed in older versions of ACLS.

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 Over the past two decades, there has been increased interest in preserving vital organ perfusion during CPR and restoring it as quickly as possible after there is a return of spontaneous circulation (ROSC). There are multiple animal studies and case series that have suggested that vasopressin or higher doses of epinephrine may be superior to the standard doses of epinephrine recommended in ACLS. Larger clinical studies have failed to demonstrate any consistent benefit to either alternative, but have also not documented worsening of outcome associated with their use. Several of the algorithms in the work product incorporate vasopressin in addition to epinephrine, as it is the opinion of the authors that the combination of the two drugs is likely superior to either alone in those clinical settings.

Avoiding ACLS is as important as performing it well. The monograph offers the algorithms below as reasonable approaches to the management of patients with LV shock and RV shock. 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 are masters of recognizing and treating hypoxemia. 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. Traditional ACLS is intended for caregivers summoned to aid a patient who has suddenly collapsed. In the perioperative setting, the list of causes is substantially larger, and ACLS needs to be managed concurrently with the anesthetic and operation.
Common Causes of ACLS events in the perioperative setting 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

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 – Some key points to remember . . .
- CPR for patients under 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, VT to chest rise, TI one second with 100% oxygen – assess for obstruction, if none, institute mechanical ventilation. If obstruction, suction, fiberoptic bronchoscopy, consider exchanging the airway. Continue CPR.
- Open all IVs to wide open
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.

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 is 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 (neuraxial), with more arrests occurring in patients with spinal anesthesia versus epidural (2.9 vs. 0.9 per 10,000 ; P = 0.041) (Anesth Analg 2005;100:855-865). In 46% (12/26) 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). In 54% (14/26), the anesthetic technique, i.e. subarachnoid block contributed directly to the arrest. 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
N.B. – the full monograph covers a variety of other scenarios, which are not
included in this handout due to space limitations. These include:
- Local Anesthetic Overdose
- Gas embolism
- Tachycardia
- Bradycardia
- Obstetric Patients

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**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

**REFERENCES**

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