Perioperative Management of Cardiac Arrhythmias

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Frequent updates on the treatments for common and infrequent cardiac arrhythmias is needed to maintain proficiency in appropriate management (1). There has been a critical reevaluation of treatment paradigms for these conditions in light of data demonstrating that antiarrhythmic drugs have important proarrhythmic and negative inotropic side effects and that they do not improve long-term survival. ACLS guidelines are thus now based on an objective evaluation of available data on the risk versus benefit of each therapy (Table 1) (2,3). The aims of this lecture are to 1) discuss evidence-based treatments for critical cardiac arrhythmias; 2) provide a focused overview of the management of atrial fibrillation (AF) and Torsade de Pointe (TdP); and 3) summarize special considerations for the perioperative management of cardiac arrhythmias.

ARRHYTHMIA CLASSIFICATIONS

In some situations the precise diagnosis of a cardiac arrhythmia is not readily clear. Tachyarrhythmias are thus often broadly classified as narrow QRS complex and wide QRS complex (interval >120 ms) tachycardias (Table 2). Life-threatening wide QRS complex ventricular arrhythmias include ventricular fibrillation (VF) and ventricular tachycardia (VT). Monomorphic or polymorphic VTs are designations for when the QRS complexes have consistent or repeatedly changing amplitude and polarity, respectively. TdP is a polymorphic VT occurring when there is pre-existing QT interval prolongation.

DEFIBRILLATION/CARDIOVERSION

Elapsed time from the onset of VF to defibrillation is the most critical determinant of return of spontaneous circulation. Prospects of survival decline up to 10% for each delayed minute to defibrillation, but by 3%–5% per minute when immediate CPR is given (2–6). ACLS recommendations for defibrillation have undergone notable revision. Rescuers of a witnessed cardiac arrest should immediately begin CPR until a defibrillating device is available. When the cardiac arrest is not witnessed, five cycles of CPR (for single rescuer, one cycle is a 30:2 compression/breathe ratio; for double rescuer, 15:2) should be given before defibrillation. These recommendations are based on data showing rates of survival are increased when defibrillation is preceded by up to 3 min of CPR and differ from the prior "shock first" teaching (3,6). Another notable change in ACLS guidelines is to immediately resume CPR after the first and subsequent shocks rather than delivering three stacked shocks (3). The emphasis is on limiting delays/interruptions in CPR while awaiting defibrillator recharging.

The energy needed for defibrillation is dependent on the waveform of the delivered electrical current. Biphasic waveform current initially flows in a positive direction, and then, it reverses to flow in a negative direction until completion of delivered energy (2,3,7). In contrast, monophasic current waveform flows only in a positive direction. Biphasic waveforms require less energy and are more effective than monophasic waveforms for defibrillation/cardioversion (7,8). Defibrillation using a biphasic waveform should be with 120–200 J (Class IIa); the optimal energy is dependent on decay properties of the waveform and is thus device specific (3). For monophasic waveforms the energy level should be 360 J. Internal defibrillation during cardiac surgery with 5 J biphasic current is more effective than 10–20 J monophasic current (8). Energy settings for cardioversion are less than that needed for defibrillation. For monomorphic and polymorphic VT the initial settings for *synchronized* cardioversion are 100 and 200 J, respectively (2,3). The dose may be increased if repeated shocks are needed. Synchronized cardioversion of atrial flutter or SVT should be with 50–100 J and for atrial fibrillation 100–200 J (2,3).

Electrode positioning is classically to the right of the upper sternum below the clavicle and at the heart apex at the midaxillary line left of the nipple. Placing the apex electrode anteriorly over the heart and the other electrode posteriorly below the right scapular areas enhances success (3). Self-adhesive electrodes facilitate the latter configuration and have the added benefit of freeing up the hands of the rescuer (9).

VASOACTIVE ADJUNCTS: EPINEPHRINE VERSUS VASOPRESSIN

Epinephrine promotes coronary and cerebral perfusion pressure due to α-adrenergic agonist-mediated vasoconstriction; β-adrenergic agonist properties increase myocardial oxygen demand and may be harmful (10). Vasopressin increases coronary and cerebral perfusion after experimental cardiac arrest without β-adrenergic effects (11). Initial small studies suggested that vasopressin was more effective than epinephrine during cardiac arrest (12,13). A subsequent prospectively randomized

Table 1. American Heart Association Classes of Recommendations (3)

Class I	Benefits ⋙ Risks	Procedure/treatment should be performed/given
Class IIa	Benefits ≫ Risks	Procedure/treatment is reasonable
Class IIb	Benfits ≥ Risks	Procedure/treatment might be considered
Class III	Risk ≥ Benefits	Procedure/treatment should not be performed/given

trial comparing two injections of vasopressin 40 U (n = 589) with two injections of epinephrine 1 mg (n = 597) in out-of-hospital cardiac arrest found no benefit for vasopressin for VF or pulseless VT (14). *Post hoc* analysis suggested a benefit with vasopressin versus epinephrine when the first rhythm was asystole. A 40 U IV/IO injection of vasopressin can be considered instead of epinephrine, but the benefits are not clear (3).

EVIDENCE-BASED TREATMENTS FOR VF/PULSELESS VT

Amiodarone can be considered for refractory VF, pulseless VT, or recurring ventricular arrhythmias but only as *secondary treatment* after defibrillation/cardioversion (Class IIb) (2,3). The initial recommended dose is 300 mg IV/IO followed by one additional dose of 150 mg. These recommendations are based on prospectively randomized, double-blinded clinical trials during cardiac arrest showing that amiodarone use led to higher survival to hospital admission (but not survival to hospital discharge) than placebo or lidocaine (15,16). Amiodarone is recommended for treatment of hemodynamically stable VT (Class IIb) (3). Although lidocaine has a historical position for the treatment of ventricular arrhythmias based mostly on animal experiments, its use during cardiac arrest is supported by only one human trial (2,3,17,18). Lidocaine can be considered as an alternative treatment for VF or pulseless VT, but it has not been shown to have short- or long-term efficacy (2,3). The use of procainamide during VF or pulseless VT is not supported by outcome evidence (3).

EVIDENCE-BASED TREATMENTS FOR NARROW OR WIDE COMPLEX TACHYCARDIA

Cardioversion is the treatment for narrow or wide QRS complex tachycardia when the patient is unstable (hypotension, chest pain, mental status changes, or evidence of hypoperfusion) (2,3). Wide QRS complex tachycardia of unknown etiology but not associated with hemodynamic instability can be treated with amiodarone (3). Regular narrow QRS complex tachycardia that is paroxysmal in onset (PSVT) can be terminated with vagal maneuvers or adenosine (3). Doses of adenosine >6 mg might be needed in the presence of theophylline, caffeine, or theobromine (3). Patients receiving dipyridamole or carbamazepine and those with a transplanted heart are sensitive to adenosine and the dose should be reduced (≤3 mg) (3). Drugs blocking the AV node such as calcium channel blockers or β-blockers (Class IIa) can be considered in the absence of impaired LV function, whereas digoxin (Class IIb) is considered when LV dysfunction is present (3). The latter drug has a limited role perioperatively because its heart rate slowing effects are vagally mediated, are slow in onset, and less effective in the setting of high sympathetic tone. When AV nodal blocking drugs are ineffective, cardioversion should be considered. AV nodal blocking drugs should not be given for regular or irregular tachyarrhythmias in the presence of preexcitation syndromes such as WPW as they may result in a paradoxical increase in ventricular rate.

ATRIAL FIBRILLATION

Atrial Fibrillation (AF) is the most prevalent cardiac arrhythmia in the general population and a common complication after cardiac and thoracic surgery (19). An irregular narrow or wide QRS complex tachycardia is likely AF, but other possibilities include atrial flutter or MAT. Guidelines for the management of patients with AF have been published (20). Treatment goals are heart rate control, restoring sinus rhythm, and assessment for anticoagulation. Rate control is a priority and it can be achieved with AV nodal blocking drugs (e.g., β-blockers, calcium channel blockers). The major risk of restoring sinus rhythm from AF is cerebral and/or systemic embolization of an intramural thrombus. For the most part, rhythm control can be safely performed with low risk for embolization when AF has been present for <48 h. Cardiac thrombus must be excluded with transesophageal echocardiography before cardioversion of AF persisting >48 h (and of unknown duration) or the patient should be anticoagulated for at least 3 wk (INR 2.0 to 3.0) before cardioversion. Anticoagulation should then be continued for 4 wk after cardioversion (20). Amiodarone can be considered for cardioversion or maintenance of sinus rhythm after electrical cardioversion in the setting of preserved (Class IIa) or impaired (Class IIb) LV function (2,3,20). Ibutilide, propafenone, dofetilide, or flecainide actually have a higher level of recommendation than amiodarone for cardioversion of AF (Class I), but they have higher pro-arrhythmic potential, must be administered according to strict monitoring guidelines, and require authorization by a clinical electrophysiologist in most institutions (20).

Patients may occasionally present for surgery with previously undiagnosed or new onset AF. Inquiry of the patient's history should include questions regarding palpitations or sensation of an irregular heart rate. A 12-lead ECG is needed to confirm the rhythm, exclude pre-excitation, evaluate for LVH, and assess for prior MI (20). A transthoracic echocardiogram should be performed to evaluate for valvular heart disease, right and left ventricular function and chamber size, LV hypertrophy, estimation of PA pressure, LA thrombus (low sensitivity), and pericardial disease (26). Thyroid, renal, and hepatic function should be evaluated (20).

Table 2. Wide QRS (Duration >120 ms) and Narrow QRS Complex Tachyarrhythmias

Wide QRS complex Tachycardia	Narrow QRS complex Tachycardia
Monomorphic VT	Sinus tachycardia
Polymorphic VT	Atrial fibrillation
Supraventricular tachycardia with aberrant ventricular conduction (e.g., bundle brand block)	Atrial flutter
Tachycardia with accessory bypass tract	AV nodal re-entry
	Accessory pathway tachycardia
	Multifocal atrial tachycardia (MAT)
	Junctional tachycardia
	Atrial tachycardia (ectopic or re-entrant)

Table 3. Transient Imbalances Contributing to Ventricular Arrhythmias

Perioperative stress-laryngoscopy, pain	Myocardial reperfusion injury
Direct mechanical stimulation—CVP, PA catheters, chest tubes	Hypothermia
Electrolyte abnormalities—K^+, Mg^{++}	Cardioactive drugs-inotropic drugs, β-blocker withdrawal
Airway complications/ $\downarrow po_2$, $\downarrow$ pH, $\uparrow Pco_2$	Proarrhythmia
Medication errors/drug toxicity	Pneumothorax/pericardial tamponade
Trauma	Pulmonary embolism
Myocardial ischemia/infarction	Macro- or microshock

There is a great deal of interest in pharmacologic strategies to prevent AF after cardiac surgery. A critical evaluation of available trials supports the use of β-blockers before and after surgery if not contraindicated (Class I) (19–21). Multiple trials have suggested that amiodarone is effective prophylactic therapy for postoperative AF (22). A major limitation of many trials is the failure to account for β-blocker use before and after surgery in patients randomized to the placebo group. Treatment benefit might simply be explained by β-blocker withdrawal in the control patients, a situation avoided in amiodarone-treated patients. Postoperative AF prophylaxis with amiodarone can be considered (Class IIa), but the benefits over a pure β-blocker have not been clearly established (19–22). Of interest is data from a prospectively randomized trial showing that atorvastatin (40 mg/day) started 7 days before cardiac surgery was effective in reducing the frequency of postoperative AF compared with placebo (23). The high AF rate in the control versus the amiodarone group must be acknowledged (57% vs 35%, $P = 0.003$).

TORSADES DE POINTES

TdP is a polymorphic VT in the setting of pre-existing QT interval prolongation. Long QT syndromes are classified as congenital or acquired. Congenital long QT syndromes result from mutations in genes encoding Na^+ and K^+ channels (24,25). The resultant "ion channelapathies" result in abnormalities of cardiac repolarization and refractoriness. Polymorphisms for genes coding components of ion channels might further be responsible for some cases of acquired long QT syndromes (24,25). In this instance, affected individuals are "silent carriers" and only manifest prolonged QT intervals in the presence of a precipitating drug. Of interest, mutant forms of a specific K^+ ion channel ($KCNQ1_{A344V}$) were found to have increased sensitivity to local anesthetics suggesting a pharmacogenetic susceptibility to local anesthetic cardiotoxicity (26).

Immediate cardioversion is necessary when TdP is associated with hemodynamic instability. Even when initially stable, TdP can rapidly progress and hemodynamics can abruptly become compromised. Treatment is directed at stopping all medication that prolong the QT interval and correcting electrolyte abnormalities. Internet query using the term "torsade de pointe" is helpful in identifying lists of potential offending agents. Of interest to anesthesiologist is the "black box" warning issued by the FDA for the prophylactic use of droperidol against emesis based on a review of 74 reported cardiac events including five cases of TdP (27). When TdP is not associated with hemodynamic instability, there are several treatment modalities, but most lack substantial evidence of efficacy. Drug induced TdP may respond to 1–2 g of magnesium IV, but efficacy of this treatment is supported by only observational studies (3,28,29). Overdrive atrial pacing or increasing the heart rate with isoproterenol lacks convincing supportive data for treating TdP (2,3). Class I and III antiarrhythmic drugs prolong the QT interval and should be avoided.

SPECIAL CONSIDERATIONS FOR PERIOPERATIVE ARRHYTHMIAS

Transient imbalances occurring perioperatively may contribute to the onset and/or maintenance of cardiac arrhythmias warranting a careful search for metabolic and other precipitants (Table 3). *It can not be underemphasized that a search for reversible causes of arrhythmias must be undertaken whenever a cardiac arrhythmia develops.* Ventricular premature beats and nonsustained ventricular tachycardia seldom require pharmacologic treatment (in the absence

of structural cardiac abnormality) and removal of transient imbalances may be all that is necessary. In the setting of cardiac disease, the latter arrhythmias identify individuals at risk for sudden cardiac death; consultation with an electrophysiologist may be warranted after surgery. Myocardial ischemia must always be considered as a cause of ventricular tachyarrhythmias even after surgical revascularization. Nonetheless, tachyarrhythmias often occur without evidence of myocardial ischemia/infarction, or hemodynamic alterations (1,30).

CARDIAC ARREST DURING REGIONAL ANESTHESIA

Cardiac arrest during regional anesthesia may result from local anesthetic cardiovascular toxicity possibly complicated by sympathetic blockade and acidosis/hypoxia from accompanying sedation (31). Blockade of cardiac sympathetic nerves leads to vagal predominance and venodilation reducing cardiac preload. Use of ephedrine in this circumstance might increase myocardial contractility further enhancing vagal stimulation via myocardial mechanoreceptors (Bezold-Jarisch reflex). Thus, therapy aimed at vasoconstriction and increasing cardiac preload has been recommended (31). A retrospective review from the Mayo Clinic found that the frequency of cardiac arrest during neuroaxial anesthesia declined between 1983 and 2002 (32). Cardiac arrest was more common with spinal than epidural anesthesia, more frequent during hip surgery, often occurred well after the regional block was established, and was commonly associated with an event such as blood loss or cement placement.

Cardiovascular toxicity of local anesthetics include effects on vascular smooth muscle, myocardial contractile state, and ventricular conduction. Bupivacaine has a narrow margin of safety between plasma concentrations producing CNS toxicity and cardiovascular toxicity possibly due to slow dissociation from Na^+ channels promoting conduction delay and re-entry (33). VT/VF due to bupivacaine may be refractory to conventional treatment (34). An advance in treating refractory cardiac arrest associated with bupivacaine toxicity may be "lipid rescue." In animal models, lipid infusion allowed for resuscitation from bupivacaine-induced cardiac arrest, possibly by effects on mitochondrial carnitine-dependent lipid transport mechanisms (35). In a case report, administration of 100 mL of 20% intralipid IV resulted in return of sinus rhythm during cardiac arrest after bupivacaine/mepivacaine interscalene block (36). It must be noted that doses of propofol (prepared as 10% lipid emulsion) needed for a similar effect would be associated with cardiovascular compromise particularly in a patient in cardiac arrest (35).

Cardiotoxicity from ropivacaine is intermediate between lidocaine and bupivacaine (37). Cardiac arrest during peripheral nerve block with ropivacaine has been reported and is noteworthy for presenting with bradycardia and asystole in contrast to refractory ventricular arrhythmias seen with bupivacaine (38). Prevention of local anesthesia toxicity is the most prudent recommendation including practices such as using a test dose, fractionated dosing, use of lowest effective volume and concentrations, adherence to maximum dose guidelines, and careful patient vigilance for toxicity.

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