Intraoperative and Postoperative Arrhythmias: Diagnosis and Treatment

Karen L. Booth, MD, Lucile Packard Children’s Hospital

Arrhythmias are common after congenital heart surgery [1]. Postoperative electrolyte imbalances, myocardial stress and dysfunction, and intracardiac incisions and suture lines all play a role in the etiology of new rhythm disturbances. Both bradycardias and tachycardias occur, but fortunately most are self-limited.

The most common bradycardia is sinus bradycardia. Patients separating from cardiopulmonary bypass may be hypothermic with core temperatures of 32-34 °C; they will remain bradycardic until their temperature rises closer to normal. Patients at risk for sinus bradycardia are those undergoing atrial baffles such as a lateral tunnel type Fontan for a single ventricle defect or atrial switch procedures for transposition of the great arteries. Patients with sinus venosus and primum atrial septal defect repairs also commonly have slower sinus rates. Extremely bradycardic patients can be treated with pacing with temporary epicardial wires placed before separating from cardiopulmonary bypass.

Surgeries near the AV node such as ventricular septal defect repairs, AV canal repairs, resection of subaortic muscle, and tetralogy of Fallot repairs create risk for injury to the conduction system. Usually this is manifest as bundle branch block, most commonly right bundle branch block, after tetralogy of Fallot repair. Left bundle branch block can occur with subaortic surgeries. Rarely there is more significant block such as complete heart block. Fortunately, it resolves in the majority of patients (< 2% incidence overall, with recovery in 2/3 of affected patients by two weeks postoperatively) without need for permanent pacemaker placement [2].

Common tachyarrhythmias include supraventricular tachycardia and junctional tachycardia. Patients with accessory pathways will often first develop a tachyarrhythmia in the postoperative period. Ectopic tachycardias are also common after congenital heart surgery [3]. High catecholamine states, intracardiac catheters and sutures lines, and electrolyte abnormalities all contribute to the appearance of postoperative
tachyarrhythmias in previously asymptomatic patients. The onset of tachycardia may provide useful information regarding its etiology. Tachycardias with a sudden onset that generate a sustained heart rate with minimal variability are likely to be reentrant, using an accessory pathway, in nature. Tachycardias with a gradual increase in heart rate with some beat-to-beat variability are likely to be automatic (or ectopic) in nature. Transesophageal echocardiography (M mode) is useful in diagnosing atrial arrhythmias with variable ventricular conduction such as atrial flutter and atrial fibrillation (see Figure 1). Direct inspection of the heart is also extremely useful as the atrial appendages may be seen fibrillating in the operative field. Temporary epicardial atrial wires can be used to obtain intracardiac atrial electrograms to examine the relationship of the P waves to the QRS complex to further aide in diagnosis (see Figure 2). Atrial wires can also be used to terminate reentrant rhythms by overdrive pacing. In the operating room, the initial treatment of choice for reentrant rhythms or atrial fibrillation is usually cardioversion with internal paddles, external pads, or atrial wires. Cardioversion will be ineffective with automatic rhythms such as ectopic atrial tachycardia and junctional tachycardia (see below). If the arrhythmia persists, pharmacological therapy should be considered. For reentrant and ectopic supraventricular tachycardias, β-blockers, such as esmolol, are usually the first-line therapy and are well tolerated. Digoxin can be considered in patients with apparent accessory pathways that are not pre-excited or for rate control in rapid atrial rhythms. Second-line agents include procainamide and amiodarone. Amiodarone should be used with caution, as the incidence of serious side effects including circulatory collapse, heart block, and proarrhythmia is high [4]. Once the tachycardia is controlled and the patient has progressed to extubation in the intensive care unit, intravenous agents can be transitioned to oral agents (e.g., the β-blocker propranolol can be substituted for esmolol). More refractory supraventricular tachycardias may be treated with the class III agent sotalol, or class IC agents flecainide or propafenone. The length of oral treatment will vary with the nature of the tachycardia, but should be considered for at least 1-3 months postoperatively.

Junctional ectopic tachycardia (JET) is a narrow complex rhythm that arises just below the AV node. It is most common after ventricular septal defect closure in tetralogy of Fallot and AV canal repairs. JET usually occurs in the first 48 hours postoperatively.
and is almost always self-limited. The typical electrocardiographic finding is AV dissociation (see Figure 3) with a more rapid ventricular rate (typically > 180 bpm), but there can be retrograde P waves with a 1:1 relationship. Because of the lack of AV synchrony, this rhythm can lead to elevated atrial pressures and low cardiac output with hypotension. Treatment is directed towards reducing catecholamine states by weaning vasoactive infusions such as dopamine, providing sedation, analgesia and possibly paralysis, and avoidance of hyperthermia with active cooling. Atrial pacing above the junctional rate (if < 180 bpm) can restore AV synchrony and improve hemodynamics. The most efficacious pharmacologic therapy includes procainamide in combination with cooling [5]. Amiodarone has been reported to be successful in the treatment of JET [6], but it should be used with caution due to acute cardiac side effects. Because JET is a self-limited tachycardia, once the patient has regained normal sinus rhythm, a gradual reduction in therapy starting with rewarming and removal of deep sedation and/or paralysis can occur. Pharmacologic support should then be weaned over 24-48 hours.

Ventricular ectopy after congenital heart surgery can occur, especially in the setting of electrolyte abnormalities. Monomorphic premature ventricular beats or ventricular bigeminy or trigeminy is usually benign and self-limited. Sustained ventricular tachycardia that is rapid and polymorphic is dangerous and concerning for myocardial ischemia. Pharmacological treatment with lidocaine or amiodarone may be attempted, but an urgent evaluation of myocardial function and perfusion must be pursued or a cardiac arrest may be imminent.

References:

**Figure 1.** M-mode echocardiogram of atrial flutter. Upper arrows mark the atrial contraction and the lower arrows mark ventricular contraction. The atrial rate is twice the ventricular rate. This is atrial flutter with 2:1 ventricular conduction.

**Figure 1.** The surface electrocardiogram (lower tracing) reveals an irregular, narrow complex rhythm. Atrial depolarizations (P waves) are not easily discernable. The unipolar atrial electrocardiogram (upper tracing) reveals a rapid ectopic atrial tachycardia with many blocked atrial premature beats. Atrial depolarization is denoted by “A” and ventricular depolarization is denoted by “V”.
Figure 2. The surface electrocardiogram (upper tracing) reveals a regular, narrow complex rhythm with possible atrioventricular (AV) disassociation. The unipolar atrial electrocardiogram (lower tracing) demonstrates AV dissociation with a ventricular rate that is more rapid than the atrial rate. This is junctional ectopic tachycardia. Atrial depolarization is denoted by “A” and ventricular (or junctional) depolarization is denoted by “V”.