Pediatric Cardiac Intensive Care

Cardiac arrhythmias in Pediatric intensive care unit

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Introduction

Arrhythmias are a common dilemma confronting pediatric intensivists and are most likely to occur in patients with structural heart disease. The inciting factor is usually related to hypoxia, ischemia, catecholamines, electrolyte imbalance or central lines. The physiological impact of given arrhythmia depends on ventricular response rate, duration as well as underlying cardiac function. Patient's underlying cardiac status is the key to management. Bradyarrhythmias may decrease cardiac output in patients with relatively fixed stroke volumes. Similarly, tachyarrhythmias may decrease diastolic filling and reduce cardiac output, resulting in hypotension, in addition to producing myocardial ischemia. In cardiac emergencies, accurate differentiation of ventricular and supraventricular tachyarrhythmia is essential for appropriate management.

This review provides an updated approach to current concepts of diagnosis and acute management of arrhythmias in Pediatric intensive care unit. A systematic approach to diagnosis and evaluation will be presented followed by consideration of specific arrhythmias.

Approach to Arrhythmias in PICU

Artifacts, mechanical problems due to central line and electrolyte problems (potassium, calcium and magnesium related) must be thought of in all cases of arrhythmias in PICU. Rhythm problems once confirmed need to be urgently categorized into two categories: stable or unstable. A twelve lead EKG should be attempted and a rhythm strip printed.

In general atrial arrhythmias such as atrial ectopics, supraventricular tachycardias, first degree AV block, atrial flutter, occasional ventricular ectopics are considered stable but need close monitoring for deterioration. In general all ventricular arrhythmias are potentially unstable. Treatment modalities such as cardioversion, defibrillation and urgent pacing are decided by ventricular response and effect on the cardiac output.

Always remember that first principle in managing arrhythmias is to treat the patient rather than the electrocardiogram. Evaluation heart rate and rhythm quickly and assessing the level of significance in terms of hemodynamic alterations; blood pressure and peripheral perfusion. If the patient loses consciousness or becomes hemodynamically unstable in the presence of a tachyarrhythmia, prompt electrical cardioversion is indicated. If the patient loses consciousness or becomes hemodynamically unstable in the presence of a bradyarrhythmia, prompt medical therapy or cardiac pacing (external or internal) is indicated. If heart rate is below 60 in a newborn or infant, cardiac compression must be instituted in addition to ongoing treatment.

Classification of arrhythmias:

Arrhythmias are commonly classified according to rate, rhythm and electrocardiographic findings. Electrocardiographically, arrhythmias can be characterized as bradycardias, tachycardias or extrasystoles. Bradycardias are further subdivided into the level of dysfunction i.e. sinus node or atrioventricular dysfunction. Tachycardias can be
further classified according to the anatomic level of origin as supraventricular or ventricular and functional mechanism reentry, automaticity or triggered activity.

**Evaluation of Tachyarrhythmias:**

The first step in the evaluation of the critically ill patient with an arrhythmia is to assess hemodynamic stability. Next step in the evaluation is to determine whether the arrhythmia is supraventricular or ventricular in origin based on width of QRS complex. A narrow QRS complex (<120 msec) represents supraventricular tachycardia (SVT). The site of origin may be in the sinus node, the atria, the atrioventricular node, the His bundle, or some combination of these sites (Figure-1).

Pathogenesis involves three electrophysiological mechanisms: (a) reentry (b) Increased automaticity (c) triggered automaticity. Reentry is the most common mechanism. If SVT's are regular and narrow complex, consider reentry. Examples include atrioventricular nodal reentrant tachycardia (AVNRT), atrioventricular reentrant (AVRT), ectopic atrial tachycardia, and atrial flutter. In cases of irregular narrow complex SVT's, consider increased automaticity which includes Atrial Fibrillation (AF), multifocal atrial tachycardia, atrial flutter with variable block, and sinus tachycardia with frequent premature atrial complexes and junctional ectopic tachycardia.

A widened QRS tachycardia (≥ 120 msec) occurs when ventricular activation is abnormally slow. The most common reason that a QRS is widened is that the arrhythmia originates outside of the normal conduction system (eg: ventricular tachycardia). Alternatively, a supraventricular arrhythmia can produce a widened QRS if there are either preexisting or rate-related abnormalities within the His-Purkinje system (eg: supraventricular tachycardia with aberrancy), or if conduction occurs over an accessory pathway. Thus, wide QRS complex tachycardias may be either supraventricular or ventricular in origin.

**Regular Rhythms**

Sinus Tachycardia

Sinus tachycardia is a rhythm in which the rate of impulses arising from the sinoatrial (SA) node is elevated. Common potential causes include anxiety, pain, fever, hypovolemia, anemia, hypoxemia, medications, and, occasionally, alcohol withdrawal. Less common causes include thyrotoxicosis, pheochromocytoma and methemoglobinemia. Treatment focuses on identifying and trying to correct the underlying cause.

Atrioventricular reciprocating tachycardia (AVRT):

The most common type of reentrant SVT in children is AVRT, involving both atrial and ventricular tissue. They display a fixed 1:1 AV relationship. Orthodromic reciprocating tachycardia (ORT) is the most common AV reentrant tachycardia in normal infants and is the most common mechanism of SVT. In ORT, antegrade conduction is over AV node, whereas retrograde conduction to the atria occurs via an accessory pathway (Figure-2). QRS morphology and duration are usually normal, with retrograde P wave following each QRS complex.
When the accessory pathway conducts impulses in the antegrade direction (atrium to ventricle) two parallel routes of AV conduction are possible: one is subject to delay through the AV node, and the other occurs without delay through the accessory pathway and results in preexcitation of the ventricle. The result is a characteristic ECG pattern during sinus rhythm consisting of a short PR interval, a "delta wave" (both of which reflect preexcitation), and a widened QRS complex due to the delta wave. This ECG pattern is referred to as the Wolff-Parkinson-White (WPW) pattern (Figure 3). It is important to appreciate that many patients with the WPW pattern do not develop SVT; when episodes of SVT do occur, the patient is said to have the WPW syndrome. A natural history study in young men suggested that, among patients with WPW syndrome, the incidence of SVT is only about 1 percent per year.

Antidromic reciprocating tachycardia (ART) is much less common in which circuit is reversed: antegrade conduction occurs via accessory pathway resulting in preexistent QRS. As a result, ART is not readily distinguishable from Ventricular tachycardia by ECG features alone.

Permanent junctional reciprocating tachycardia (PJRT): In most cases, conduction through the accessory pathway is quite rapid and comparable to the conduction velocity of normal myocardium. Permanent junctional reciprocating tachycardia (PJRT) is a variant of orthodromic AVRT in which the retrograde conduction in the accessory pathway is slow. Slow retrograde conduction through the accessory pathway coupled with the normally slow conduction antegrade through the AV node creates a stable reentrant circuit. As a result, PJRT is an incessant SVT, in contrast to the typically paroxysmal nature of most SVT.

AV nodal reentrant tachycardia (AVNRT): AV nodal reentrant tachycardia (AVNRT) is the most common cause of SVT in older children and adults. It is seen less common in infants. AV nodal reentrant tachycardia (AVNRT) is mediated by the presence, within the AV node, of two conducting pathways that are designated fast and slow. The fast pathway has a short conduction time but long refractory period. The slow pathway has a long conduction time but short refractory period. These distinct pathways allow for a reentrant loop using one pathway in the antegrade direction and one in the retrograde direction. Typical AVNRT utilizes the slow pathway in the antegrade direction and the fast pathway in the retrograde direction. Atypical AVNRT employs the fast pathway in the antegrade direction and the slow pathway in the retrograde direction.

Treatment of Reentrant Tachycardia:
Acute management of a infant or child who presents in the emergency department depends upon hemodynamic status. If the child is hemodynamically unstable, synchronized DC cardioversion with 0.5 -2.0 J/kg should be performed. In
children who are hemodynamically stable and have mild or no symptoms, vagal maneuvers should be attempted. Vagal maneuver for infants or young children include application of a bag filled with ice and cold water over the face for 15 to 30 seconds. This elicits the diving reflex, frequently interrupting the arrhythmia. This maneuver is successful in 30 to 60 percent of cases. If the vagal maneuver is unsuccessful pharmacological agents can be tried. Adenosine is the drug of first choice. It is given by rapid intravenous injection over one to two seconds at a site as close to the central circulation as possible. The usual initial dose is 0.1 mg/kg; if no response is seen within two minutes, the dose is doubled. Adenosine terminates 80 - 95 percent of episodes of AVRT, which accounts for almost three-quarters of episodes of SVT and approximately 75 percent of episodes due to other causes of SVT. Early recurrence of the SVT after termination occurs in 25 to 30 percent of cases. Side effects, include flushing, nausea, vomiting, feeling of discomfort, chest pain, and dyspnea. These are transient and resolve rapidly. Adenosine should be avoided or used with caution in the following settings.

- In patients with Wolff-Parkinson-White (WPW), AF can degenerate into ventricular fibrillation.
- In patients with a wide QRS complex tachycardia, if the specific arrhythmia is not identified, since adenosine can provoke severe hemodynamic deterioration in those who have ventricular tachycardia rather than an SVT.
- In patients with pre-existing second or third degree heart block or sinus node disease where it is contraindicated.

Atrial Tachycardia (AT):
Atrial tachycardia (AT) is a subset of SVT originating entirely from atrial tissue and does not require the atrioventricular (AV) junction, accessory pathways, or ventricular tissue for initiation and maintenance of the elevated heart rate (Figure-4). Atrial fibrillation and atrial flutter, although fulfilling this definition, are usually not included in the designation of AT and are typically identified as specific entities. Atrial ectopic tachycardia (AET) represents 10-20% of SVT in pediatric population. Its an automatic arrhythmia that presents as an incessant rhythm. In addition arrhythmia has been associated with chronic cardiomyopathy. Focal atrial tachycardia (FAT), also due to a single focus, behaves in a paroxysmal manner, starting and stopping abruptly. Microreentry or triggered activity are thought to be the most common cause. Multifocal Atrial tachycardia (MAT) or chaotic atrial rhythm is defined by 3 or more P-wave morphology, atrial rate >400 and ventricular rate of 150-250/min.

Treatment of AET/MAT:
Atrial ectopic tachycardia is generally responsive to medical therapy, and there are high chances of spontaneous resolution. In several case series, spontaneous remission rates vary from 40 to 75 percent for patients up to 18 years of age within 10 to 28 months of diagnosis. Medical treatment involves Digoxin which decreases ventricular response rate by slowing AV conduction but has no effect on ectopic focus. Other drugs that can be used are amiodarone and propafenone. In patients with chronic or frequently recurrent conditions who unresponsive to medical therapy, ablation therapy is suggested.

Multifocal atrial tachycardia resolves completely.
after six months and the prognosis for long-term outcome is excellent in children. In some cases, MAT is seen in patients with structural heart disease, and the outcome is dependent upon the underlying condition. Medical therapy is therapy directed at controlling the ventricular response rate with a combination of oral digoxin, beta blocker, and calcium channel blocker.

Atrial Flutter: May present during utero or newborn period. It is a macroreentry tachycardia which involves electrical circuit between the inferior vena cava and the tricuspid valve annulus, the cavitricuspid isthmus. It manifests as a sawtooth pattern on ECG, usually with atrioventricular block. Atrial flutter in patients who have undergone surgery for congenital heart disease is referred to as Intraatrial Reentrant tachycardia (IART). A high incidence of this arrhythmia has been noted in long-term follow-up, particularly in patients after intraatrial surgeries such as the Mustard or Senning procedure for palliation of d-transposition of the great arteries and the Fontan procedure for single ventricle physiology. Atrial fibrillation (AF) involves multiple, simultaneous atrial reentry wavelets which appear on a rhythm strip as a low amplitude or choppy, irregular baseline with a variable R-R interval.

Ventricular tachycardia (VT)

Ventricular tachycardias include all tachycardias that arise exclusively within the ventricle below the bifurcation of the bundle of His. VT is defined as three or more consecutive beats on ECG. Sustained VT is defined as more than 30 seconds of ventricular beats at a rate of more than 100 bpm. The mechanisms by which ventricular arrhythmias occur are the same as those that result in supraventricular arrhythmias: reentry, automaticity, and triggered automaticity.

VT is most frequently associated in patients with tetralogy of Fallot, although it may occur in patients with other congenital defects (eg, transposition of the great arteries, Ebstein's anomaly, and lesions with left ventricular outflow obstruction). Several genetic disorders are associated with VT and they are classified as channelopathies or cardiomyopathic disorders.

Channelopathy:

The long QT syndrome (LQTS) is a disorder of
myocardial repolarization characterized by prolonged QT interval and T wave abnormalities. QT intervals usually exceed 0.46. Patients with LQTS present with syncope, seizures, or cardiac arrest. A family history of sudden death, seizures, recurrent syncope, or unexplained drowning should heighten the suspicion of LQTS. The LQTS can be congenital, as an inherited disorder usually involving a mutation of an ion channel gene, or can be acquired secondary to drugs, metabolic abnormalities, or bradyarrhythmias. Torsades de pointes is the specific arrhythmia associated with Prolonged QT syndromes and is responsible for the symptoms. This characteristic arrhythmia is recognized by progressive undulation in the QRS axis, resulting in a “twisting” appearance (Figure-6).

Brugada syndrome is associated with characteristic ECG findings of ST segment elevation in leads V1 to V3 and a right bundle branch block pattern in the right precordial leads. Implantation of a defibrillator is the only established effective treatment and is indicated for symptomatic patients.

Catecholaminergic polymorphic ventricular tachycardia (CPVT):
Patients with CPVT has characteristic feature of ventricular tachycardia with beat-to-beat alternation of the QRS axis occurring with physical or emotional stress and can be asymptomatic. Mutations in the cardiac ryanodine receptor gene (RYR2) or cardiac calsequestrin (CASQ2) underlie catecholaminergic bidirectional ventricular tachycardia. Therapies include beta blockers or Intracardiac defibrillator.

Cardiomyopathy: VT is often associated with inherited cardiomyopathies such as hypertrophic cardiomyopathy. Arrhythmogenic right ventricular cardiomyopathy (ARVC) and myotonic dystrophy.

Other causes: Include acquired ones like coronary heart disease, myocarditis, Chagas disease, electrolyte and metabolic disturbances. Hyperkalemia can lead to variety of conduction abnormalities and arrhythmias. Tricyclic antidepressants (TCA) toxicity can cause widening of the QRS complex and ventricular arrhythmias.

Sodium bicarbonate is the antidote of choice for TCA toxicity.

Treatment:
Treatment is determined by the degree of hemodynamic compromise. Acute management of a child who presents with wide QRS complex tachycardia and is based upon the 2005 American Heart Association, American Academy of Pediatrics, and International Liaison Committee on Resuscitation (AHA/AAP/ILCOR) guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) of pediatric patients. In stable patients with VT, the guidelines recommend that i.v amiodarone at a dose of 5 mg/kg (maximum dose 300 mg) be given slowly over 20 to 60 minutes. Additional doses can be given in patients who remain in VT and do not have signs of toxicity. If amiodarone is not available i.v procainamide can be administered as a 15 mg/kg bolus over 30 to 60 minutes ECG and blood pressure monitoring are required as amiodarone and procainamide can prolong the QT interval and cause hypotension. Synchronized cardioversion (0.5-1 joules/kg) can be used to treat perfusing ventricular arrhythmia, if the rhythm is pulseless defibrillate immediately (2-4 joules/kg). If the rhythm is consistent with torsades de pointes, intravenous magnesium at a dose of 25 to 50 mg/kg may be given.
**Bradyarrhythmias:**

Bradycardia is defined as heart rates below the lowest normal values set for age. There are two main mechanisms for bradycardia. Either it is due to sinus node defect resulting in sinus bradycardia or it may be due to abnormalities of AV conduction including first, second or third degree heart block.

**Sinus Bradycardia:** Sinus bradycardia is present when there is a normal sinus appearing P-wave on the 12-lead ECG but the rate is below normal for age. Causes include increased vagal tone, raised intracranial pressure, hypoxia, hypothyroidism, Drugs (digoxin, β blocker, calcium channel blocker) and post cardiac surgery

**Abnormalities of AV conduction:**

Atrioventricular (AV) block is defined as a delay or interruption in the transmission of an atrial impulse to the ventricles. The conduction can be delayed, intermittent, or absent. Heart block is divided into three categories.

1. **First-degree AV block** occurs when the PR-interval is greater than the upper limits of normal for age. (Figure-8)

   ![Figure-8](image1)

2. **Second-degree AV block** is further divided into two categories based on ECG findings, Mobitz type 1 and type 2.

   In Mobitz type 1 block (also referred to as Wenckebach block), there is progressive prolongation of the PR-interval until a P wave fails to be conducted. (Figure-9)

   ![Figure-9](image2)

3. **Third-degree AV block** is also referred to as complete heart block. On ECG, there is complete dissociation of the atrial and ventricular activity. (Figure-11)

   ![Figure-11](image3)

**Treatment:**

First thing is to seek the underlying cause. It is particularly important in intensive care setting where airway compromise is the most common cause of acute bradycardia. Raised ICP, electrolyte imbalance produces bradycardia which may require intervention. No intervention is needed for sinus bradycardia as long as cardiac output is maintained. No treatment is necessary for first degree or Mobitz type-I heart block. Mobitz type-II and third degree are always pathological.

For hemodynamically significant bradycardia, after managing airway, initial treatment is usually pharmacological, whether the cause is sinus node slowing or AV nodal block. Atropine transiently ameliorates bradycardiac effect. Continuous infusion of epinephrine and isoproterenolol may be instituted. Consider cardiac pacing, particularly if a conduction defect is detected or suspected. In PICU, temporary pacing is most commonly used after surgery.

**Conclusion:**

Artifacts, mechanical problems due to central line and electrolyte problems must be thought of in all cases of arrhythmias in PICU. Rhythm problems once confirmed need to be urgently categorized into two categories: stable or unstable. If the patient...
loses consciousness or becomes hemodynamically unstable in the presence of a tachyarrhythmia, prompt electrical cardioversion is indicated. However, if the patient loses consciousness or becomes hemodynamically unstable in the presence of a bradyarrhythmia, prompt medical therapy or cardiac pacing (external or internal) is indicated.

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