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SYMPOSIUM ON PICU PROTOCOLS OF AIIMS

Arrhythmias in Children

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Abstract Arrhythmias in children can be classified according to their effect on central pulse: Fast pulse rate – tachyarrhythmia; Slow pulse rate – bradyarrhythmia; and Absent pulse is pulseless arrest (cardiac arrest). Tachyarrythmia may be narrow complex tachycardia (QRS duration ≤0.08 s): sinus tachycardia (ST), supraventricular tachycardia (SVT), atrial flutter or Wide-complex tachycardia (QRS duration >0.08 s): ventricular tachycardia (VT), SVT with aberrant intraventricular conduction. The choice of therapy depends on the patient's degree of hemodynamic instability. Attempt vagal stimulation, if patient is stable and if it does not unduly delay chemical or electrical cardioversion. Bradyarrhythmias include: sinus bradycardia, sinus node arrest with atrial, junctional and idioventricular escape rhythms and AV block. The emergency treatment of bradycardia depends on its hemodynamic consequences. If heart rate is <60 beats per minute with poor perfusion despite effective ventilation with oxygen, it may be treated with chest compressions, epinephrine through IV or endotracheal tube. If bradycardia persists or responds only transiently, consider a continuous infusion of epinephrine or isoproterenol and plan for emergency transcutaneous pacing. If bradycardia is due to vagal stimulation or primary A-V block, giving atropine may be beneficial.

Keywords Bradyarrythmia · Tachyarrythmia · Cardioversion · Heart block

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Rhythm disturbances (arrhythmias) are not uncommon in sick children admitted in pediatric intensive care unit. These occur as a result of abnormalities in, or insults to, the cardiac conduction system or heart tissues. A rhythm disturbance in a child should be treated as life threatening emergency.

Normal Electrocardiogram (ECG)

The ECG is a graphic representation of the sequence of myocardial depolarization and re-polarization with each normal cardiac cycle consisting of a P, QRS and a T wave (Fig. 1).

Electrical depolarization begins in sinuatrial node at the junction of superior vena cava and right atrium and advances via atrial tissue and the intermodal pathways to the atrioventricular junctional tissue. It then progresses via bundle of His and its divisions to depolarize the ventricular myocardium.

Approach to a Child with Arrhythmia

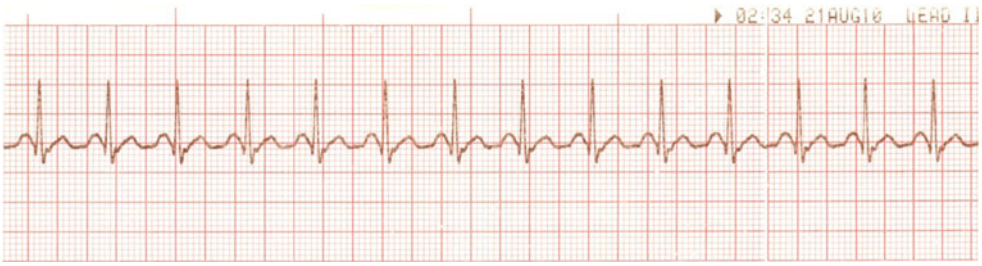
The two most important considerations in evaluating the rhythm disturbance in a child are:

1. Child's typical heart rate and rhythm
2. Child's clinical condition, especially hemodynamic status.

Arrhythmias in children can be classified according to their effect on central pulse:

1. Fast pulse rate = tachyarrhythmia;
2. Slow pulse rate = bradyarrhythmia; and
3. Absent pulse = pulseless arrest (cardiac arrest).

Fig. 1 Normal electrocardiogram



Tachyarrhythmias

Tachyarrhythmias represent a variety of fast abnormal rhythms originating either in the atria or the ventricles of heart. Tachycardia is defined as a heart rate that is fast compared with normal heart rate for the patient's age. Normal heart rate by age is given in Table 1.

If there is tachycardia, look for hemodynamic stability and QRS complex in ECG. The rhythm is unstable if it causes signs or symptoms of poor tissue perfusion (weak pulses, shock with hypotension, respiratory distress, and altered consciousness). Tachyarrhythmias may be classified according to width of the QRS complex:

- Narrow complex tachycardia (QRS duration ≤0.08 s): sinus tachycardia (ST), supraventricular tachycardia (SVT), atrial flutter, or
- Wide-complex tachycardia (QRS duration >0.08 s): ventricular tachycardia (VT), SVT with aberrant intraventricular conduction.

Sinus Tachycardia (ST)

Sinus tachycardia is defined as a rate of sinus node discharge faster than normal for patient's age (Table 1). ST typically develops in response to body's need for increased cardiac output or oxygen delivery. Evaluation of a 12-lead ECG and the patient's clinical presentation should help differentiate sinus tachycardia from supraventricular tachycardia (SVT) (Table 2). Common causes of ST include tissue hypoxia, hypovolemia, fever, metabolic stress, injury, pain, anxiety, toxins, and anemia. Less common causes include cardiac tamponade, tension pneumothorax, and thromboembolism. If the rhythm is ST, search for and treat causes.

Table 1 Normal heart rate by age [1]

Age	Heart rate (per min)	Mean (per min)
Newborn to 3 months	85–205	140
3 months to 2 years	100–190	130
2 years to 10 years	60–140	80
>10 years	60–100	75

Supraventricular Tachycardia (SVT)

SVT [previously known as paroxysmal atrial tachycardia (PAT) and paroxysmal supraventricular tachycardia (PSVT)] is an abnormally fast rhythm originating above the ventricles. SVT is the most common tachyarrhythmia producing cardiovascular compromise during infancy.

Three groups of tachycardia are included in SVT [2]:

1) *AV re-entrant* (or *reciprocating) tachycardia* is not only the most common mechanism of SVT but also the most common tachyarrhythmia seen in the pediatric age group. In SVT caused by re-entry, two pathways are involved; at least one of these is the AV node, and the other is an accessory pathway. Patients with accessory pathways frequently have Wolff-Parkinson-White (WPW) pre-excitation (producing a "delta wave" on ECG).
2) *Ectopic (*or *nonreciprocating) atrial tachycardia* is a rare mechanism of SVT in which rapid firing of a single focus in the atrium is responsible for the tachycardia.

Table 2 Differentiation between sinus tachycardia and supraventricular tachycardia

Parameter		Sinus tachycardia	SVT
Heart rate	Infants	Usually <220/min	Usually ≥220/min
	Children	Usually <180/min	Usually ≥180/min
History		Gradual onset, compatible with known cause e.g. fever, pain, volume loss.	Abrupt onset, vague, non-specific history, symptoms of CHF.
P-wave		Present/normal (upright in I/aVF)	Absent/abnormal (negative in leads II/III/aVF)
R-R interval		Variable with level of activity	Not variable
P-R interval		Constant	Variable
Chest Radiograph		Usually small heart and clear lungs unless pneumonia or underlying heart disease.	Signs of CHF (e.g. cardiomegaly, pulmonary edema) may present.

3) *Nodal* (or AV junctional) *tachycardia* may superficially resemble atrial tachycardia because the P wave is buried in the T waves of the preceding beat and becomes invisible, but the rate of nodal tachycardia is relatively slower (120 to 200 beats/min) than the rate of ectopic atrial tachycardia.

Causes of SVT

1. Idiopathic in about 50% and this type more commonly in young infants than in older children.
2. WPW pre-excitation is present in 10% to 20%.
3. Some congenital heart defects (e.g., Ebstein's anomaly, single ventricle, congenitally corrected transposition of the great arteries) are more susceptible to this arrhythmia.
4. Following cardiac surgeries.

Clinical Features of SVT

Many infants tolerate SVT well. If the tachycardia is sustained for 6 to 12 h, signs of CHF (irritability, tachypnea, poor feeding, and pallor) usually develop. Older children may complain of chest pain, palpitation, shortness of breath, lightheadedness, and fatigue. History and ECG characteristics are helpful in differentiating SVT from ST (Table 2, Fig. 2).

Management of SVT

The choice of therapy depends on the patient's degree of hemodynamic instability. Attempt vagal stimulation, if patient is stable and if it does not unduly delay chemical or electrical cardioversion [3]. In infants and young children, apply ice to the face without occluding the airway [4]. In older children, carotid sinus massage or Valsalva maneuvers are safe [5]. One method of a Valsalva maneuver is to have the child blow through an obstructed straw [6]. Do not apply pressure to the eye because this can damage the retina. Adenosine is the drug of choice for SVT. If IV access is readily available administer adenosine 0.1 mg/kg (maximum first dose 6 mg) using 2 syringes connected to a T-connector or stopcock; give adenosine rapidly with one syringe and immediately flush with ≥5 mL of normal saline with the other. If there is no reversal, then double the first dose (maximum second dose 12 mg) once. If the patient is very unstable or IV access is not readily available, provide electrical (synchronized) cardioversion. Start with a dose of 0.5 to 1 J/kg. If unsuccessful, repeat using a dose of 2 J/kg. If a second shock is unsuccessful or the tachycardia recurs quickly, consider antiarrhythmic therapy – amiodarone (5 mg/kg over 20 to 60 min) or procainamide (15 mg/kg over 30–60 min) before a third shock. Use extreme caution when administering more than one drug that causes QT prolongation (e.g., amiodarone and procainamide). If there is no effect and there are no signs of toxicity, give additional doses; amiodarone upto 15 mg/kg (maximum 300 mg). Intravenously administered propranolol has been commonly used to treat SVT in the presence of WPW syndrome. Esmolol, other β-adrenergic blockers, verapamil and digoxin have also been used with some success. Do not use verapamil in infants below 1 year because it may cause refractory hypotension and cardiac arrest, and use with caution in children because it may cause hypotension and myocardial depression [7]. Radiofrequency catheter ablation or surgical interruption of accessory pathways should be considered if medical management fails or frequent recurrences occur.

Atrial Flutter (Intra-Atrial Re-entrant Tachycardia)

It is characterized by re-entry circuit within the atria that allows a wave of depolarization to travel in a circle within the atria. There is an atrial rate (F wave with "sawtooth" configuration) of about 300 (range 250 to 400) beats/min, a ventricular response with varying degrees of block (e.g., 2:1, 3:1, 4:1), and normal QRS complexes (Fig. 3). The ventricular rate determines eventual cardiac output; a too-rapid ventricular rate may decrease cardiac output. Thrombus formation may lead to embolic events. Uncontrolled atrial flutter may precipitate heart failure. The flutter may be associated with syncope, presyncope, or chest pain.

Causes include structural heart disease with dilated atria, acute infectious illness, myocarditis or pericarditis, previous surgery involving atria (the Senning procedure, Fontan operation, or atrial septal defect repair), digitalis toxicity, and thyrotoxicosis.

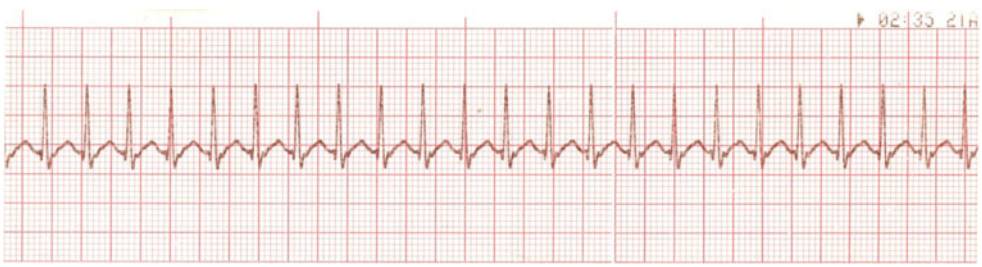


Fig. 2 Electrocardiogram showing supraventricular tachycardia

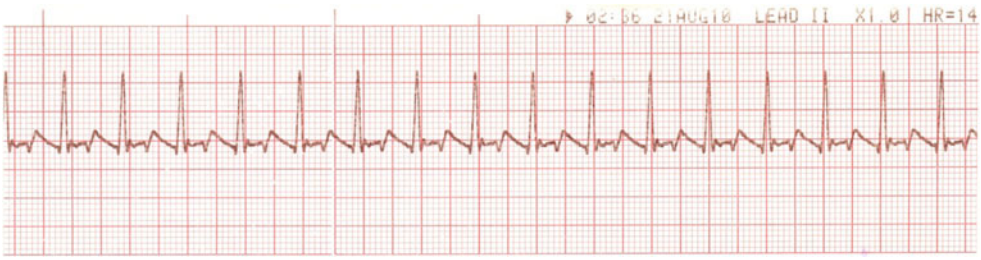


Fig. 3 Atrial flutter

Management

Immediate synchronized DC cardioversion is the treatment of choice for atrial flutter of short duration, if the infant or child is in severe CHF. In some cases, IV amiodarone or IV procainamide may be effective. For chronic cases: anticoagulation with warfarin (with International Normalized Ratio target of between 2.0 and 3.0) is started and cardioversion is delayed for 2 to 3 weeks. After conversion to sinus rhythm, anticoagulation is continued for an additional 3 to 4 weeks.

Atrial Fibrillation

Atrial fibrillation is less common than atrial flutter in children. The atrial excitation is chaotic and more rapid (300–700 beats/min) producing an irregularly irregular ventricular response and pulse with normal QRS complexes (Fig. 4).

Causes include structural heart diseases with dilated atria, such as seen with rheumatic heart disease, Ebstein's anomaly, tricuspid atresia, atrial septal defect, AV valve regurgitation, previous intra-atrial surgery, thyrotoxicosis, pulmonary emboli, and pericarditis. If atrial fibrillation has been present for more than 48 h, anticoagulation with warfarin for 2 to 3 weeks is recommended to prevent systemic embolization of atrial thrombus, if the conversion can be delayed. Anticoagulation is continued for 3 to 4 weeks after the restoration of sinus rhythm. Propranolol, digoxin, quinidine, procainamide or amiodarone may be used to slow the ventricular rate.

Ventricular Tachycardia

Ventricular tachycardia (VT) is a wide-complex tachyarrhythmia generated within the ventricles. Rapid ventricular rate often compromise stroke volume and cardiac output, and they may deteriorate into pulseless VT or Ventricular Fibrillation (VF). Causes include underlying heart disease (or post cardiac surgery), prolonged QT syndrome, myocarditis/cardiomyopathy, electrolyte disturbances (e.g. hyperkalemia, hypocalcemia, hypomagnesemia) and drug toxicity (e.g. tricyclic antidepressants, cocaine). In VT, ventricular rate is at least 120/min and regular, QRS complex is wide (>0.08 s), P waves are often not identifiable; when present there is AV dissociation and T wave are typically opposite in polarity from QRS (Fig. 5).

Management of VT

If hemodynamically stable, give an infusion of amiodarone 5 mg/kg slowly while you monitor the ECG and blood pressure. If there is no effect and there are no signs of toxicity, give additional doses up to 15 mg/kg (max. 300 mg). If amiodarone is not available, consider giving procainamide 15 mg/kg slowly (over 30 to 60 min) or lidocaine 1 mg/kg bolus followed by infusion 20–50 μg/kg/min.

If there is poor perfusion, treat with synchronized electrical cardioversion (0.5 J to 1 J/kg). Repeat with 2 J/kg, if no response. If it does not delay cardioversion, try a dose of adenosine first to determine if the rhythm is SVT with aberrant conduction. If second shock is unsuccessful or if the tachycardia recurs quickly, consider antiarrhythmic therapy (amiodarone or procainamide) before a third shock. If at any time the patient develops pulseless arrest, start CPR.

Torsades de Pointes

This polymorphic VT is seen in patients with hypomagnesemia, long QT interval, which may be congenital or may

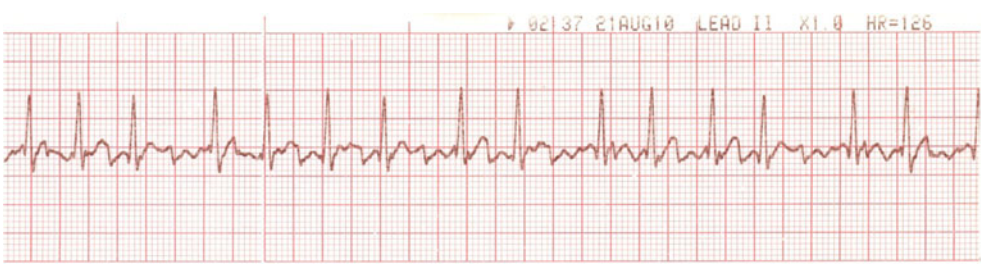


Fig. 4 Atrial fibrillation

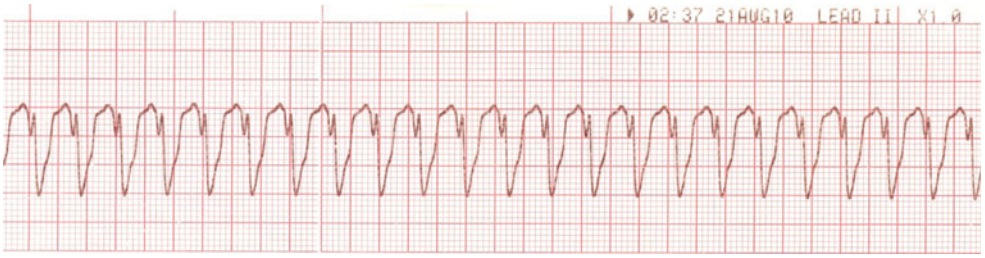


Fig. 5 Electrocardiogram showing ventricular tachycardia

result from toxicity with type IA antiarrhythmics (e.g., procainamide, quinidine, and disopyramide) or type III antiarrhythmics (e.g., sotalol and amiodarone), tricyclic antidepressants, digitalis, or drug interactions [8]. In torsades the QRS complexes change in polarity and amplitude, appearing to rotate around the ECG iso-electric line (Fig. 6). Regardless of the cause, treat torsades de pointes with IV infusion of magnesium sulfate 25–50 mg/kg over 20–30 min.

While managing tachyarrhythmia, look for and treat possible contributing factors which can be remembered as Hs and Ts [1]: Hs – hypovolemia, hypoxia/ventilation problem, hydrogen ion (acidosis), hypo-/hyperkalemia, hypoglycemia, hypothermia and Ts – toxins, tamponade (cardiac), tension pneumothorax, thrombosis (coronary or pulmonary) and trauma (hypovolemia).

Bradyarrhythmias

Bradycardia is defined as a heart rate that is slow compared with normal heart rates for patient's age. Clinically significant bradycardia is defined as a heart rate less than normal for patient's age associated with poor systemic perfusion. Bradyarrhythmias are the most common pre-arrest rhythm in children. They are often associated with conditions such as hypoxia, hypotension, and acidosis. Examples of bradyarrhythmias include: sinus bradycardia, sinus node arrest with atrial, junctional and idioventricular escape rhythms and AV block.

Sinus Bradycardia

It is usually an incidental finding in otherwise healthy persons, particularly in young adults, sleeping patients, and well-conditioned athletes. Pathologic causes of sinus bradycardia include hypoxia (most common), poisoning, electrolyte disorders, infection, sleep apnea, drug effects, hypoglycemia, hypothyroidism, and increased intracranial pressure.

Sinus Node Arrest

It is characterized by absent pacemaker activity in the sinus node and a subsidiary pacemaker in atrium, AV junction or ventricles initiating cardiac depolarization with following rhythms:

1. Atrial escape rhythm—Impulse arises from a subsidiary nonsinus atrial pacemaker. There is late P wave of different morphology.
2. Junctional escape rhythm—Impulse originates in AV node and produces a slow, uniform, narrow QRS complex because the ventricles are depolarized through normal conduction system. Retrograde P wave may or may not be present.
3. Idioventricular escape rhythm—Impulse originates in ventricles when there is significant bradycardia or high grades AV block and produce slow (usually between 30–40/min) wide complex.

AV Block

It is a disturbance of electrical conduction through the AV node. It is classified as following (Fig. 7):

1. First degree—Characterized by a prolonged PR interval representing a slow conduction through AV node. Causes include intrinsic AV nodal disease, myocarditis, electrolyte disturbance (hyperkalemia), drugs (digoxin, β-blockers, calcium channel blockers), acute rheumatic fever and may also in healthy persons. It is usually asymptomatic.

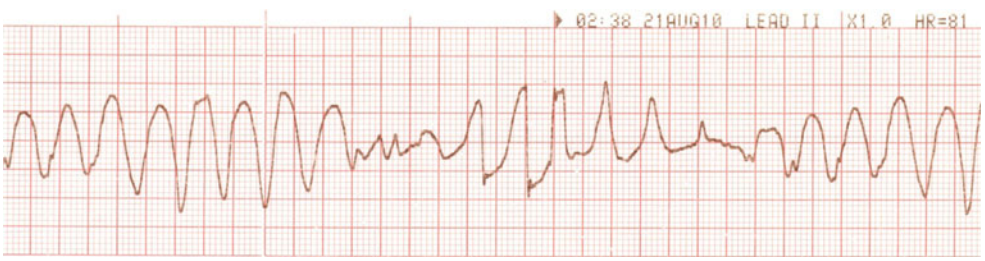


Fig. 6 ECG showing torsades de pointes

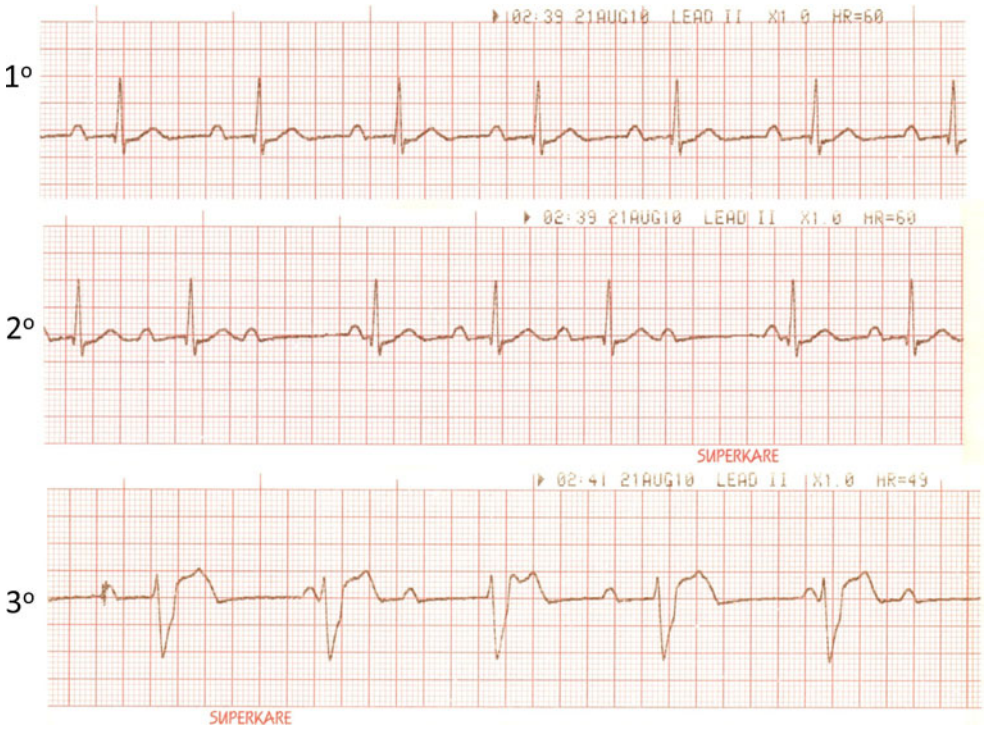


Fig. 7 Conduction blocks

2. Second degree—Some but not all atrial impulses are conducted to ventricles. It is of two types:
    a. Mobitz type I block (Wenckebach phenomenon): Block occurs at AV node and there is progressive prolongation of PR interval until an atrial impulse is not conducted to ventricles. As a result, one P is not followed by a QRS complex. Causes include drugs (digoxin, β-blockers, calcium channel blockers), any condition that stimulates parasympathic tone. It rarely causes dizziness.
    b. Mobitz type II: Block occurs below AV node and it is characterized by consistent inhibition of a specific proportion of atrial impulses, 2:1 or 3:1 atrial to ventricular rate. It typically results from organic lesion in the conduction pathway or acute coronary syndrome. It may cause faintness or lightheadedness.
3. Third degree—It is complete heart block and none of the atrial impulse is conducted to ventricles. There is no relationship between P waves and QRS complex. Ventricular rhythm is maintained by subsidiary pacemaker. Causes include extensive conduction system disease or injury (post surgery), congenital block, myocarditis, myocardial infarction, toxic drug effects. Symptom may include fatigue, syncope.

Management of Bradyarrhythmias

The emergency treatment of bradycardia depends on its hemodynamic consequences. If at any time the patient develops pulseless arrest, start CPR. If bradycardia is causing cardiorespiratory compromise, support airway, breathing, and circulation as needed, administer oxygen, and attach a monitor/defibrillator. Reassess; if pulses, perfusion, and respirations are normal, no emergency treatment is necessary. Monitor and proceed with evaluation. If heart rate is <60 beats per min with poor perfusion despite effective ventilation with oxygen, start chest compressions. Continue to support airway, ventilation, oxygenation (and provide compressions as needed) and give epinephrine 0.01 mg/kg IV/IO (1:10,000; 0.1 ml/kg) or 0.1 mg/kg through endotracheal tube (1:1,000; 0.1 ml/kg) and repeat every 3–5 min. If bradycardia persists or responds only transiently, consider a continuous infusion of epinephrine (0.1–0.3 μg/kg/min) or isoproterenol.

If bradycardia is due to vagal stimulation or primary A-V block, give atropine 0.02 mg/kg IV/IO (minimum dose 0.1 mg; maximum single dose 0.5 mg for child and 1 mg for adolescent) or 0.04 to 0.06 mg/kg through endotracheal tube. Repeat dose in 5 min, if no response (maximum total dose, 1 mg for child and 2 mg for adolescent). Emergency transcutaneous pacing may be lifesaving if the bradycardia is due to complete heart block or sinus node dysfunction unresponsive to ventilation, oxygenation, chest compressions, and medications, especially if it is associated with congenital or acquired heart disease [9]. Pacing is not useful for asystole or bradycardia due to post-arrest hypoxic/ischemic myocardial insult or respiratory failure. While managing bradyarrhythmia, look for and treat possible

contributing factors which can be remembered as Hs and Ts (see above).

Cardiac Arrest

It, also called cardiopulmonary arrest or pulseless arrest, is the cessation of circulation of blood as a result of absent or ineffective cardiac mechanical activity. Clinically, the patient is unresponsive, apneic, with no detectable pulse. In children, cardiac arrest is more often caused by progression of respiratory distress, respiratory failure, or shock than primary cardiac arrhythmias [10]. Two presentations of cardiac arrest in children are: hypoxic/asphyxial arrest (common) and sudden cardiac arrest (uncommon, usually associated with VF or pulseless VT). Asystole and bradycardia with a wide QRS complex are most common in asphyxial cardiac arrest [10]. Cardiac arrest is associated with one of the following rhythm, also known as arrest rhythm:

1. Asystole
2. Pulseless electrical activity (PEA)
3. Ventricular Fibrillation (VF)
4. Pulseless VT (including torsades de pointes)

Management of Cardiac Arrest

If there is cardiac arrest, support ABC and start high quality CPR immediately (start with chest compressions, push hard and push fast, minimize interruptions of chest compressions with a ratio of 15 compressions to 2 breaths. Once an advanced airway is secured, give continuous chest compressions at a rate of 100 per min without pauses for ventilation, that is, at a rate of 8–12/min. Rotate the compressor role, approximately every 2 min to prevent compressor fatigue and deterioration in quality and rate of chest compressions). Give supplementary oxygen and attach ECG monitor/defibrillator. Determine the rhythm whether it is "shockable" (i.e., VF or pulseless VT) or "not-shockable" (i.e., asystole or PEA).

"Shockable Rhythm": Ventricular Fibrillation (VF)/ Pulseless VT

VF is a chaotic, disorganized series of depolarization that results in a quering myocardium without organized contractions (Fig. 8). VF occurs in 5% to 15% of all pediatric victims of out-of hospital cardiac arrest and is reported in up to 20% of pediatric in-hospital arrests at some point during the resuscitation [11]. Defibrillation is the definitive treatment for VF with an overall survival rate of 17% to 20% [11]. Use a dose of 2 J/kg for the first attempt and 4 J/kg for subsequent attempts.

Give 1 shock (2 J/kg) as quickly as possible and immediately resume CPR. Give 5 cycles of CPR in 2 min. Check the rhythm. If a shockable rhythm persists, give 1 shock (4 J/kg), resume CPR immediately. Give a dose of epinephrine. Use a standard dose of epinephrine for the first and subsequent doses. Repeat epinephrine about every 3 to 5 min during cardiac arrest. After 5 cycles of CPR, check the rhythm. If the rhythm continues to be "shockable," deliver a shock (4 J/kg), resume CPR immediately, and give amiodarone (5 mg/kg) or lidocaine (1 mg/kg). Continue CPR for 5 cycles before again checking the rhythm and attempting to defibrillate if needed with 4 J/kg. If there is an organized rhythm at any time, check for a pulse and if pulse present, begin post-resuscitation care. If defibrillation is successful but VF recurs, continue CPR while you give another bolus of amiodarone before you try to defibrillate with the previously successful shock dose. Search for and treat reversible causes.

"Nonshockable Rhythm": Asystole/PEA

The most common ECG findings in infants and children in cardiac arrest are asystole and PEA. PEA is organized electrical activity—most commonly slow, wide QRS complexes—without palpable pulses. This subcategory (formerly known as electromechanical dissociation [EMD]) is more likely to be treatable. For asystole and PEA: resume CPR and continue with as few interruptions in chest compressions as possible. A second rescuer gives epinephrine while the first continues CPR. Use a standard dose for the first and subsequent doses. Search for and treat reversible causes.

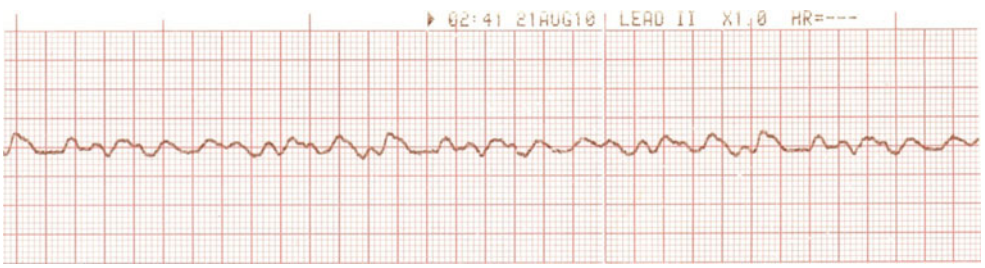


Fig. 8 Ventricular fibrillation

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