Chapter 05
Cardiovascular Emergencies

Objectives
- Define the following terms: afterload, preload, cardiac output, and stroke volume.
- List assessment findings consistent with circulatory compromise.
- Define shock (hypoperfusion).
- Discuss the common causes of shock in infants and children.
- Describe the clinical classifications of shock.
- Describe the assessment findings that indicate shock in infants and children.
- Differentiate between compensated and decompensated shock.

Objectives
- Describe the initial management of hypovolemic, cardiogenic, distributive (septic, anaphylactic, neurogenic), and obstructive shock in infants and children.
- Describe assessment findings that indicate cardiopulmonary failure or arrest in children.
- Discuss the primary etiologies of cardiopulmonary arrest in infants and children.
- Identify the major classifications of pediatric cardiac dysrhythmias.
- Identify four essential questions to ask in the initial emergency management of a pediatric patient with a dysrhythmia.
Objectives

- Recognize the following dysrhythmias: bradycardia, sinus tachycardia, supraventricular tachycardia, ventricular tachycardia, ventricular fibrillation, and asystole.
- Differentiate sinus tachycardia from supraventricular tachycardia and supraventricular tachycardia from ventricular tachycardia.
- Recognize a “sick” (unstable) and “not sick” (stable) infant or child with a cardiac dysrhythmia.
- Discuss the dysrhythmias associated with pediatric cardiopulmonary failure or arrest.

Objectives

- Discuss the management of cardiac dysrhythmias in infants and children.
- Discuss the pharmacology of medications used during shock, symptomatic bradycardia, stable and unstable tachycardia, and cardiopulmonary arrest.
- Given a patient situation, formulate a management plan (including assessment, airway management, CPR, pharmacological, and electrical interventions where applicable) for a patient in shock, or presenting with symptomatic bradycardia, stable or unstable tachycardia, or cardiopulmonary arrest.

Review of the Cardiovascular System
Coronary Arteries

Blood Vessels
- Arteries are conductance vessels
- Arterioles are resistance vessels
- Capillaries are exchange vessels
- Veins are capacitance vessels

Arteries
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- Infants and children are capable of more effective vasoconstriction than adults are.
- As a result, a previously healthy infant or child is able to maintain a normal blood pressure and organ perfusion for a longer time in the presence of shock.
Perfusion

- Perfusion
  - Circulation of blood through an organ or a part of the body
  - Delivers oxygen and other nutrients to the cells of all organ systems and removes waste products

- Hypoperfusion (shock)
  - Inadequate circulation of blood through an organ or a part of the body

Heart Rate

- Autonomic nervous system
  - Sympathetic division
    - Mobilizes the body
    - Allows body to function under stress
    - "Fight or flight" response
  - Parasympathetic division
    - Responsible for conservation and restoration of body resources
    - "Feed and breed" response

Cardiac Output

- Cardiac output = Stroke volume x Heart rate

- Normal cardiac output
  - Neonates: 200 mL/kg/min
  - Infants and children: 150 mL/kg/min
  - Adolescents: 100 mL/kg/min
Cardiac Output

- Changes in heart rate OR stroke volume can affect cardiac output
  - ↑ stroke volume or heart rate → ↑ cardiac output
  - ↓ stroke volume or heart rate → ↓ cardiac output
- Tachycardia is the initial compensatory response to the demand for increased cardiac output

Blood Pressure

- Systolic pressure
- Diastolic pressure
- Pulse pressure

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- An early sign of impending shock is a slight increase in diastolic pressure without a change in the systolic pressure (i.e., narrowed pulse pressure).
Vascular Resistance

Stroke Volume

- Stroke volume is determined by:
  - The degree of ventricular filling during diastole (preload)
  - The resistance against which the ventricle must pump (afterload)
  - The contractile state of the myocardium
Cardiovascular Assessment

- Compare strength/quality of central and peripheral pulses
- Evaluate cardiac rhythm—normal, fast, slow, or absent
- Look for visible hemorrhage; control bleeding if present
- Evaluate skin color, temperature, moisture
- Assess skin turgor
- Evaluate capillary refill
- Blood pressure
- Pulse pressure
- Urine output

Normal Heart Rates by Age

<table>
<thead>
<tr>
<th>Age</th>
<th>Beats/Minute*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant (1 to 12 months)</td>
<td>100 to 160</td>
</tr>
<tr>
<td>Toddler (1 to 3 years)</td>
<td>90 to 150</td>
</tr>
<tr>
<td>Preschooler (4 to 5 years)</td>
<td>80 to 140</td>
</tr>
<tr>
<td>School-age (6 to 12 years)</td>
<td>70 to 120</td>
</tr>
<tr>
<td>Adolescent (13 to 18 years)</td>
<td>60 to 100</td>
</tr>
</tbody>
</table>

*Pulse rates for a sleeping child may be 10% lower than the low rate listed in age group.

Lower Limit of Normal Systolic BP by Age

<table>
<thead>
<tr>
<th>Age</th>
<th>Lower Limit of Normal Systolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Term neonate (0 to 28 days)</td>
<td>&gt;60 mm Hg or strong central pulse</td>
</tr>
<tr>
<td>Infant (1 to 12 months)</td>
<td>&gt;70 mm Hg or strong central pulse</td>
</tr>
<tr>
<td>Child 1 to 10 years</td>
<td>&gt;70 mm Hg + (2 x age in years)</td>
</tr>
<tr>
<td>Child 10 years and older</td>
<td>&gt;90 mm Hg</td>
</tr>
</tbody>
</table>
Shock

- Early (compensated) shock
  - Also called reversible shock

- Late (decompensated) shock
  - Also called progressive shock

- Irreversible shock
  - Also called terminal shock

Early Shock

- Shock with a "normal" blood pressure

- Presence of compensated shock can be identified by:
  - Evaluation of heart rate
  - Presence and volume (strength) of peripheral pulses
  - Adequacy of end-organ perfusion
    - Brain—assess mental status
    - Skin—assess capillary refill, skin temperature
    - Kidneys—assess urine output

Late (Decompensated) Shock

- "Classic" signs and symptoms of shock are evident

- Difficult to treat, but still reversible if appropriate aggressive treatment is initiated
Irreversible Shock

- Compensatory mechanisms fail
- Cardiac dysrhythmias may develop
- Cell membranes break down and release harmful enzymes
- Irreversible damage to vital organs occurs

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- Although the amount and type of information gathered will vary depending on the child’s presentation, a history should be obtained as soon as possible from the parent or caregiver.
- The information obtained may help identify the type of shock present, ascertain the child’s previous health, and the onset and duration of symptoms.

Classification of Shock

by Etiology
Hypovolemic Shock

- Inadequate volume
- ↓ intravascular volume → ↓ venous return (preload) → ↓ ventricular filling → ↓ stroke volume → ↓ cardiac output → inadequate tissue perfusion

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- Hypovolemia and sepsis are the most common causes of shock in children.
Hypovolemic Shock—Assessment Findings

Compensated shock
- Normal blood pressure
- Narrowed pulse pressure
- Increased heart rate
- Peripheral vasoconstriction
  - Skin mottling
  - Delayed capillary refill
  - Cool extremities
- Normal or minimally impaired mental status
- Decreased urine output

Decompensated shock
- Hypotension
- Significant tachycardia
- Markedly delayed capillary refill
- Pale, mottled, mild peripheral cyanosis
- Altered mental status—irritability, lethargy
- Minimal urine output
- Weak central pulses

Hypovolemic Shock—Interventions

- Type and cross emergently if the child has severe trauma and life-threatening blood loss
- Administer a bolus of 20 mL/kg of isotonic crystalloid solution (NS or LR)
- Assess response (i.e., mental status, capillary refill, heart rate, ventilatory effort, BP)

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- Signs of shock should be treated with a bolus of 20 mL/kg of isotonic crystalloid even if blood pressure is normal.
**Hypovolemic Shock—Interventions**

- Check glucose; give dextrose if indicated
- Maintain normal body temperature
- Obtain a history as soon as possible
- Insert a urinary catheter
- Obtain appropriate laboratory studies
- Consider vasopressors if poor perfusion persists

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**Cardiogenic Shock**

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**Cardiogenic Shock—Assessment Findings**

**Compensated shock**
- Anxiety
- Pale skin, cool extremities
- Diaphoresis
- Normal or delayed capillary refill
- Weak, thready peripheral pulses
- Mild tachycardia
- Jugular venous distention
- Narrowed pulse pressure
- Mild basilar crackles
- Normal or mild decrease in urine output
- Orthopnea

**Decompensated shock**
- Lethargy
- Pale, mottled, or cyanotic skin
- Diaphoresis
- Markedly delayed capillary refill
- Weak, thready central pulses; peripheral pulses may be absent
- Hypotension
- Tachypnea with decreased tidal volume
- Increasing pulmonary congestion and crackles
- Oliguria
Cardiogenic Shock—Interventions

- Perform an initial assessment
- If a pulse is absent or ineffective, begin CPR
- Consider giving a small IV/IO fluid bolus of isotonic crystalloid solution (5 to 10 mL/kg of LR or NS)
  - Repeat the primary survey after each fluid bolus
  - An inotrope may be necessary to improve myocardial contractility and increase cardiac output
- Vasodilators may be used to reduce preload and afterload
- Treat dysrhythmias if present and contributing to shock
- Obtain a chest radiograph

Distributive Shock

- Possible causes
  - Severe infection (septic shock)
  - Severe allergic reaction (anaphylactic shock)
  - Spinal cord injury (neurogenic shock)
  - Certain overdoses (e.g., sedatives, narcotics)
Sepsis

- Systemic inflammatory response syndrome (SIRS)
  - Response to infection manifested by derangement in two or more of the following: temperature, heart rate, ventilatory rate, and white blood cell count

- Sepsis
  - Systemic response to an infection

Sepsis

- Severe sepsis
  - Sepsis associated with organ dysfunction

- Septic shock
  - Severe sepsis and the persistence of poor perfusion or hypotension for more than 1 hour despite adequate fluid resuscitation or a requirement for inotropic agents or vasopressors

Septic Shock—Assessment Findings

Early phase (increased cardiac output)
- Warm, dry, flushed skin
- Blood pressure may be normal or possible widened pulse pressure
-Bounding peripheral pulses
-Brisk capillary refill
-Tachycardia
-Tachypnea

Late (decompensated) phase
- Mottled, cool extremities
- Diminished or absent peripheral pulses
- Altered mental status
- Tachycardia
- Delayed capillary refill
- Decreased urine output
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- Late septic shock is usually indistinguishable from other types of shock.
- If you observe a change in mental status in a febrile child (inconsolable, inability to recognize parents, unarousable), immediately consider the possibility of septic shock.

Septic Shock—Interventions

- Perform an initial assessment
- Administer supplemental oxygen if indicated
  - Titrate oxygen administration to maintain an oxygen saturation of 94% or higher
- Begin CPR if indicated
- Obtain vascular access
  - Give an isotonic crystalloid solution (NS or LR) fluid bolus
- Check glucose; treat if serum glucose is less than 60 mg/dL

Anaphylaxis—Assessment Findings

- Stridor, wheezing, coughing, hoarseness, intercostal and suprasternal retractions
- Tachycardia, hypotension, dysrhythmias
- Vomiting, diarrhea
- Anxiety, restlessness
- Angioedema
- Urticaria (hives)
- Abdominal pain, cramping
- Pruritus (itching)
Anaphylaxis—Interventions

- Remove/discontinue the causative agent
- Administer supplemental oxygen if indicated
- Attach cardiac monitor
- Give epinephrine via intramuscular injection
- Obtain vascular access
  - Give an isotonic crystalloid solution (NS or LR) fluid bolus

- Consider inhaled bronchodilator therapy (e.g., albuterol)

- Administer other medications to help stop the inflammatory reaction
  - Consider diphenhydramine
  - Consider methylprednisolone

Diphenhydramine (Benadryl)

- Antihistamine/H₁ receptor antagonist
  - Stimulation of H₁ receptors causes bronchoconstriction
  - Stimulation of H₂ receptors causes peripheral vasodilation and secretion of gastric acids

- Diphenhydramine blocks cellular histamine response, but does not prevent histamine release
Neurogenic Shock

- Caused by a severe injury to the head or spinal cord that results in a loss of sympathetic vascular tone below the level of the spinal cord injury

Loss of peripheral vascular tone results in widespread vasodilation below the level of the injury → ↓ venous return → ↓ stroke volume → ↓ cardiac output → ↓ tissue perfusion

- Total blood volume remains the same, but vessel capacity is increased (relative hypovolemia)

Neurogenic Shock—Assessment Findings

- Skin is warm and dry
- Heart rate within normal limits or bradycardic
- Hypotension
- Wide pulse pressure
- Ventilatory rate/effort and breathing pattern may be affected depending on the location of the injury
Neurogenic Shock—Interventions

- Perform an initial assessment
- Spinal motion restriction
- Administer supplemental oxygen if indicated
- Attach cardiac monitor
- Obtain vascular access
  - Administer 20 mL/kg of NS or LR
  - Assess response

Obstructive Shock

- Develops from cardiac tamponade, tension pneumothorax, or a massive pulmonary embolism
- Common pathophysiology in these conditions is obstruction to blood flow from the heart

Tension Pneumothorax—Assessment Findings

<table>
<thead>
<tr>
<th>Early</th>
<th>Late</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Dyspnea</td>
<td>- Decreased level of responsiveness</td>
</tr>
<tr>
<td>- Anxiety</td>
<td>- Tracheal deviation toward the unaffected side</td>
</tr>
<tr>
<td>- Tachypnea</td>
<td>- Hypotension</td>
</tr>
<tr>
<td>- Tachycardia</td>
<td>- Distension of neck veins (may not be present if hypovolemic or in cases of severe hypotension)</td>
</tr>
<tr>
<td>- Hyperresonance of chest wall on affected side</td>
<td>- Cyanosis</td>
</tr>
<tr>
<td>- Diminished or absent breath sounds on affected side</td>
<td></td>
</tr>
</tbody>
</table>
Cardiac Tamponade—
Assessment Findings

- Beck’s triad
  - Increased jugular venous pressure
  - Hypotension
  - Muffled heart sounds
- Dyspnea
- Anxiety, restlessness
- Cold extremities
- Pale, mottled, or cyanotic skin
- Tachycardia
- Weak or absent peripheral pulses
- Narrowed pulse pressure
- Pulsus paradoxus

Obstructive Shock—Interventions

- Perform an initial assessment
- Obtain a history as soon as possible
- Administer supplemental oxygen if indicated
- Obtain vascular access
- Administer 20 mL/kg of NS or LR over 5 to 20 minutes
- Check glucose
- Maintain normal body temperature
- Perform needle decompression of the affected side for tension pneumothorax
- Cardiac tamponade
  - Volume expansion to maintain an adequate circulating blood volume
  - Pericardiocentesis is definitive treatment
- Obtain appropriate laboratory studies
- Insert a urinary catheter if necessary

Cardiopulmonary Failure
Signs of Cardiopulmonary Failure

- Bradypnea with irregular, ineffective ventilations
- Decreasing work of breathing
- Delayed capillary refill time (longer than 5 seconds)
- Bradycardia
- Weak central pulses and absent peripheral pulses
- Cool extremities
- Mottled or cyanotic skin
- Diminished level of responsiveness

Cardiopulmonary Arrest

- In children, cardiac arrests are usually the result of:
  - Respiratory failure (aphyxia precipitated by acute hypoxia or hypercarbia) or
  - Circulatory shock (ischemia from hypovolemia, sepsis, or myocardial dysfunction [cardiogenic shock])

Rhythm Disturbances
Cardiac Dysrhythmias

- Disorders of heart rate and rhythm are uncommon in infants and children
- When they do occur, they are most often a result of hypoxia secondary to respiratory arrest and asphyxia

Cardiac Dysrhythmias—Four Categories

- Dysrhythmias are divided into four broad categories based on heart rate
  - Normal for age
  - Slower than normal for age (bradycardia)
  - Faster than normal for age (tachycardia)
  - Absent/pulseless (cardiac arrest)
ECG Paper

Waveforms and Complexes

Artifact

Loose electrode

Muscle tremor
Analyzing a Rhythm Strip

- Assess the rate
  - Determine if the rate is normal for age, too fast, too slow, or absent
- Assess the width of the QRS complex
- Assess rhythm/regularity
- Evaluate the rhythm’s clinical significance
  - Stable (not sick)
    - Asymptomatic (i.e., normal BP, mental status, and respiratory status)
  - Unstable (sick)
    - Decreased responsiveness, hypotension, or respiratory failure
    - Chest pain due to ischemia may be present in older child and adolescent

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- The initial emergency management of pediatric dysrhythmias requires a response to four important questions
  - Is a pulse (and other signs of circulation) present?
  - Is the rate within normal limits for age, too fast, too slow, or absent?
  - Is the QRS wide (ventricular in origin) or narrow (supraventricular in origin)?
  - Is the patient sick (unstable) or not sick (stable)?

Rhythm Recognition
Sinus Rhythm

<table>
<thead>
<tr>
<th>Rate</th>
<th>Usually within normal limits for age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Uniform in appearance, positive (upright) in lead II, one precedes each QRS complex</td>
</tr>
<tr>
<td>PR interval</td>
<td>Within normal limits for age; constant from beat to beat</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.09 second or less</td>
</tr>
</tbody>
</table>

Sinus Arrhythmia

<table>
<thead>
<tr>
<th>Rate</th>
<th>Usually within normal limits for age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Irregular, phasic with respiration</td>
</tr>
<tr>
<td>P waves</td>
<td>Uniform in appearance, positive (upright) in lead II, one precedes each QRS complex</td>
</tr>
<tr>
<td>PR interval</td>
<td>Within normal limits for age; constant from beat to beat</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.09 second or less</td>
</tr>
<tr>
<td>Clinical Significance</td>
<td>Normal phenomenon that occurs with respiration and changes in intrathoracic pressure. Rate increases with inspiration (R-R intervals shorten) and decreases with expiration (R-R intervals lengthen). Common in infants and children.</td>
</tr>
</tbody>
</table>

Tachydysrhythmias:

Too Fast Rhythms
**Sinus Tachycardia**

<table>
<thead>
<tr>
<th>Rate</th>
<th>Faster than the upper limit of normal for age; rate usually slower than 220 beats/min in infants and slower than 180 beats/min in children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Uniform in appearance, positive (upright) in lead II, one precedes each QRS complex</td>
</tr>
<tr>
<td>PR interval</td>
<td>Within normal limits for age; constant from beat to beat</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.09 second or less</td>
</tr>
<tr>
<td>Cause</td>
<td>Anxiety, fear, fever, crying, hypovolemia, hypoxemia, pain, congestive heart failure, respiratory distress, toxins/poisonings/drugs, myocardial disease</td>
</tr>
<tr>
<td>Clinical significance</td>
<td>Compensatory response to the body’s need for increased cardiac output or O₂ delivery. Increased myocardial workload is usually well tolerated by the infant or child with a healthy heart.</td>
</tr>
</tbody>
</table>

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**Supraventricular Tachycardia**

<table>
<thead>
<tr>
<th>Rate</th>
<th>240 ± 40 beats/min, may be as high as 300 beats/min in infants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Often indiscernible due to rapid rate; may be lost in the T wave of the preceding beat. If P waves are visible, they differ in appearance from P waves that originate in the SA node and there is a 1 to 1 relationship to the QRS.</td>
</tr>
<tr>
<td>PR interval</td>
<td>Usually not measurable because P waves are not visible</td>
</tr>
<tr>
<td>QRS duration</td>
<td>0.09 second or less unless an intraventricular conduction delay exists</td>
</tr>
<tr>
<td>Cause</td>
<td>Most often due to a reentrant mechanism that involves AV junction or an accessory pathway</td>
</tr>
<tr>
<td>Clinical significance</td>
<td>Onset and termination of the rhythm are often abrupt (paroxysmal); supraventricular tachyarrhythmias may result in decreased cardiac output (↑ heart rate → ↓ ventricular filling time → ↓ stroke volume → ↓ cardiac output)</td>
</tr>
<tr>
<td>Treatment</td>
<td>Vagal maneuvers, antiarrhythmics, or synchronized cardioversion depending on stability of patient</td>
</tr>
</tbody>
</table>
Supraventricular Tachycardia

SVT in a child complaining of chest pain

Same child after one IV dose of adenosine

Differentiation of Sinus Tachycardia and SVT

<table>
<thead>
<tr>
<th>Sinus Tachycardia</th>
<th>SVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>Usually slower than 220 bpm in infants and 180 in children</td>
</tr>
<tr>
<td>Ventricular rate and regularity</td>
<td>Varies with activity/stimulation</td>
</tr>
<tr>
<td>Onset/termination</td>
<td>Gradual</td>
</tr>
<tr>
<td>P waves</td>
<td>Visible, normal appearance</td>
</tr>
<tr>
<td>History</td>
<td>History given explains rapid heart rate; pain, fever, volume loss due to trauma, vomiting, or diarrhea</td>
</tr>
<tr>
<td>Physical examination</td>
<td>May be consistent with volume loss, possible fever, clear lungs, liver normal size</td>
</tr>
</tbody>
</table>

Tachycardia with Adequate Perfusion

- Narrow QRS
  - Administer oxygen if indicated
  - Obtain 12-lead ECG
  - Attempt vagal maneuver
  - Give adenosine IV
Adenosine

- Slows the rate of the SA node
- Slows conduction time through the AV node
- Can interrupt reentry pathways that involve the AV node
- Can restore sinus rhythm in SVT
- Half-life less than 10 seconds
- Onset of action of 10 to 40 seconds
- Duration of action: 1 to 2 minutes

Tachycardia with Poor Perfusion

- Narrow QRS
  - Administer oxygen if indicated
  - Obtain 12-lead ECG
  - Consider vagal maneuver if no delays
  - Give adenosine if IV/IO access available
  - Perform synchronized cardioversion if vascular access not available or adenosine ineffective

Ventricular Tachycardia
### Monomorphic Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Feature</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rate</strong></td>
<td>120 to 250 beats/minute</td>
</tr>
<tr>
<td><strong>Rhythm</strong></td>
<td>Essentially regular</td>
</tr>
<tr>
<td><strong>P waves</strong></td>
<td>Usually not seen; if present, they have no set relationship to the QRS complexes appearing between them at a rate different from that of the VT</td>
</tr>
<tr>
<td><strong>PR interval</strong></td>
<td>None</td>
</tr>
<tr>
<td><strong>QRS duration</strong></td>
<td>Greater than 0.09 second; may be difficult to differentiate between the QRS and T wave</td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>May be caused by acute hypoxemia, acidosis, electrolyte imbalance, reactions to medications, toxins/poisons/drugs, myocarditis</td>
</tr>
<tr>
<td><strong>Clinical significance</strong></td>
<td>Slower rates may be well tolerated. Rapid rates often result in decreased ventricular filling time and decreased cardiac output; may degenerate into ventricular fibrillation</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>If no pulse, CPR and defibrillation. Pulse present—antarrhythmics or synchronized cardioversion depending on stability of patient</td>
</tr>
</tbody>
</table>

### Polymorphic Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Feature</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rate</strong></td>
<td>150 to 300 beats/min, typically 200-250 beats/min</td>
</tr>
<tr>
<td><strong>Rhythm</strong></td>
<td>May be regular or irregular</td>
</tr>
<tr>
<td><strong>P waves</strong></td>
<td>None</td>
</tr>
<tr>
<td><strong>PR interval</strong></td>
<td>None</td>
</tr>
<tr>
<td><strong>QRS duration</strong></td>
<td>Greater than 0.09 second; gradual alteration in amplitude and direction of the QRS complexes</td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>May be precipitated by slow heart rates; associated with medications or electrolyte disturbances that prolong the QT interval; a prolonged QT interval may be congenital or acquired; lengthening of the QT interval may be the only warning sign suggesting impending TdP</td>
</tr>
<tr>
<td><strong>Clinical significance</strong></td>
<td>Symptoms are usually related to the decreased cardiac output that occurs because of the fast ventricular rate; signs of shock are often present; patient may experience a syncopal episode or seizures; may occasionally terminate spontaneously and recur after several seconds or minutes; may deteriorate to ventricular fibrillation</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>If unstable, defibrillation. Pulse present—magnesium sulfate is drug of choice</td>
</tr>
</tbody>
</table>
Ventricular Tachycardia

- Monomorphic VT
- Polymorphic VT

Tachycardia with Poor Perfusion

- Wide QRS
  - Administer oxygen if indicated
  - Obtain 12-lead ECG
  - Consider adenosine if IV/IO access available and:
    - Rhythm is regular
    - QRS is monomorphic
  - Seek expert consultation before giving amiodarone or procainamide
  - If unstable, perform synchronized cardioversion

Amiodarone

- Slows conduction through the AV node
- Used for a wide range of atrial and ventricular dysrhythmias
- Prolongs the PR, QRS, and QT intervals
- Seek expert consultation before giving to an infant or child with a perfusing rhythm
- Adverse effects include hypotension, bradycardia, and AV block
Procainamide

- Used for both atrial and ventricular dysrhythmias
- Suppresses automaticity in the atria and ventricles
- Observe ECG closely for increasing PR and QT intervals, widening QRS complex, heart block, and/or onset of Torsades de Pointes
- If the QRS widens to more than 50% of its original width or hypotension occurs, slow or discontinue the infusion

Bradydysrhythmias:

Too Slow Rhythms

Bradycardias

- Heart rate slower than lower limit of normal for patient’s age
- In children, most bradycardias occur secondary to hypoxia and acidosis
Bradycardias

- Primary bradycardia
  - Usually caused by structural heart disease

- Secondary bradycardia
  - Due to a non-cardiac cause
    - Increased vagal tone
    - Hypothermia
    - Hyperkalemia
    - Medications (e.g., calcium channel blockers, digoxin)

Sinus Bradycardia

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>Slower than lower range of normal for age</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Essentially regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Uniform in appearance, positive (upright) in lead II, one precedes each QRS complex</td>
</tr>
<tr>
<td>PR interval</td>
<td>Within normal limits for age, constant from beat to beat</td>
</tr>
<tr>
<td>QRS</td>
<td>0.09 second or less</td>
</tr>
<tr>
<td>Cause</td>
<td>Hypoxemia, acidosis, increased vagal tone</td>
</tr>
<tr>
<td>Clinical significance</td>
<td>May be normal in conditioned adolescent athletes and in some children during sleep. In other patients, decreased cardiac output may occur because of slow rate, despite normal stroke volume.</td>
</tr>
<tr>
<td>Treatment</td>
<td>Search for treatable cause. Ensure good oxygenation and ventilation. Begin CPR if heart rate slower than 60 beats/min in an infant or child with poor systemic perfusion despite oxygenation and ventilation. Establish vascular access. Epinephrine, atropine, possible pacing.</td>
</tr>
</tbody>
</table>
Bradycardia—Interventions

- If an infant or child is symptomatic because of a bradycardia:
  - Initial interventions focus on airway and ventilation
  - Administer supplemental oxygen if indicated

- Begin CPR
  - If heart rate is slower than 60 beats/min despite adequate oxygenation and ventilation and accompanied by:
    - Abnormal skin color
    - Decreased level of responsiveness
    - Capillary refill longer than 2 seconds
    - Hypotension
  - If bradycardia persists:
    - Epinephrine
    - Atropine for increased vagal tone or primary AV block
    - Consider pacing

Atrioventricular (AV) Blocks

- First-Degree AV Block
- Second-Degree AV Block
  - Mobitz Type I (Wenckebach Phenomenon)
  - Mobitz Type II
- 2:1 AV Block
- Complete (Third-Degree) AV Block
Epinephrine
- Direct-acting endogenous catecholamine
  - Moderate beta-2 (bronchodilation) properties
  - Potent alpha (vasoconstriction) properties
  - Potent beta-1 (↑ heart rate, ↑ force of contraction) properties

Epinephrine
- Although beta-1 effects ↑ myocardial oxygen consumption, it is generally well tolerated in pediatric patient
- In cardiac arrest, epinephrine produces beneficial effects primarily because of its alpha-adrenergic stimulating effects
  - ↑ peripheral vascular resistance (vasoconstriction)
  - → ↑ diastolic pressure → ↑ myocardial and cerebral blood flow during CPR

Atropine
- Enhances AV conduction
- Increases heart rate by accelerating SA node discharge rate and blocking vagus nerve
- Has little or no effect on force of contraction
- Do not give atropine slowly or in smaller than recommended doses (0.1 mg)
  - Paradoxical slowing of heart rate can occur; may last 2 minutes
Atropine

- Epinephrine is drug of choice if bradycardia is due to hypoxia and oxygenation and ventilation do not correct the bradycardia
- Give atropine before epinephrine if bradycardia is due to increased vagal tone or primary AV block

Dopamine

- Endogenous catecholamine with dose-related actions
  - Low doses (0.5 to 5 mcg/kg/min)
    - Acts on dopaminergic receptors located mainly in mesenteric, renal, and coronary vessels, causing vasodilation
  - Moderate doses (5 to 10 mcg/kg/min)
    - Stimulates beta-1 adrenergic receptors, increasing myocardial contractility and stroke volume, thereby increasing cardiac output
  - High doses (10 to 20 mcg/kg/min)
    - Acts on vascular alpha-adrenergic receptors, producing systemic vasoconstriction

Absent/Pulseless Rhythms
Absent/Pulseless Rhythms

Absent/pulseless rhythms include:

- Pulseless VT
  - ECG displays a wide QRS complex at a rate faster than 120 beats/minute
- VF
  - Irregular chaotic deflections that vary in shape and amplitude are observed on the ECG, but there is no coordinated ventricular contraction
- Asystole
  - No cardiac electrical activity is present
- Pulseless electrical activity (PEA)
  - Electrical activity is visible on ECG but central pulses are absent

Ventricular Fibrillation

<table>
<thead>
<tr>
<th>Rate</th>
<th>Cannot be determined because there are no discernible waves or complexes to measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Rapid and chaotic with no pattern or regularity</td>
</tr>
<tr>
<td>P waves</td>
<td>Not discernible</td>
</tr>
<tr>
<td>PR interval</td>
<td>Not discernible</td>
</tr>
<tr>
<td>QRS</td>
<td>Not discernible</td>
</tr>
<tr>
<td>Causes</td>
<td>Hypoxia, acidosis, hypo-/hyperkalemia, hypoglycemia, hypothermia, hypovolemia, tablets/toxins (drug overdose), cardiac tamponade, tension pneumothorax, thrombosis (coronary or pulmonary), and trauma (among other causes)</td>
</tr>
<tr>
<td>Significance</td>
<td>Terminal rhythm</td>
</tr>
<tr>
<td>Treatment</td>
<td>Confirm patient is unresponsive, has absent (or only gasping) breathing, and no pulse. Begin CPR until a defibrillator is available.</td>
</tr>
</tbody>
</table>
Asystole (Ventricular Standstill)

<table>
<thead>
<tr>
<th>Rate</th>
<th>Ventricular activity not discernible but atrial activity may be observed (&quot;P-wave&quot; asystole)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Ventricular not discernible, atrial may be discernible</td>
</tr>
<tr>
<td>P waves</td>
<td>Usually not discernible</td>
</tr>
<tr>
<td>PR interval</td>
<td>Not measurable</td>
</tr>
<tr>
<td>QRS</td>
<td>Absent</td>
</tr>
<tr>
<td>Causes</td>
<td>Hypoxia, acidosis, hypo-/hyperkalemia, hypoglycemia, hypothermia, hypovolemia, tablets/toxins (drug overdose), cardiac tamponade, tension pneumothorax, thrombus (coronary or pulmonary), and trauma (among other causes)</td>
</tr>
<tr>
<td>Clinical significance</td>
<td>Absence of cardiac output; terminal rhythm. Patient is unresponsive, apneic, and pulseless.</td>
</tr>
<tr>
<td>Treatment</td>
<td>See Pulseless Arrest algorithm</td>
</tr>
</tbody>
</table>

Pulseless Electrical Activity

- Pulseless electrical activity (PEA) is a clinical situation, not a specific dysrhythmia
- PEA exists when organized electrical activity (other than VT) is observed on the cardiac monitor, but the patient is pulseless
Syncope (fainting)
- Brief loss of consciousness caused by transient cerebral hypoxia
- Loss of consciousness typically occurs within a few seconds of symptom onset
- Complete recovery shortly after patient assumes a supine position
- Causes no residual neurological problems

Non-life-threatening causes
- Increased vagal tone
- Psychogenic reactions
- Prolonged standing, fatigue, dehydration
Syncope

- Potentially life-threatening causes
  - Dyshytmias including SVT, bradycardia, prolonged QT syndrome
  - Cardiac abnormalities that decrease blood flow to the heart, lungs, brain, and body
  - Myocardial ischemia
  - Certain drug intoxications
  - Hypoglycemia, anemia, hypoxia, head trauma

- Circulatory causes
  - Vasovagal syncope
  - Orthostatic hypotension
  - Cardiac syncope
  - Extremely fast or slow heart rates
  - Prolonged QT syndrome

- Metabolic causes
- Respiratory causes

- Uncommon before age 10 to 12 years but is quite prevalent in adolescent girls

- If recurrent, may have a major effect on lifestyle and/or quality of life

- Family history positive for similar episodes in 90% of patients
Syncope—History

- Frequently preceded by lightheadedness, nausea, “gray-out,” sweating, and pallor (presyncope)
- May occur while sitting, standing, walking, and occasionally during exercise

Syncope—Common Prodromal Symptoms

- Lightheadedness 89%
- Visual disturbances 71%
- Sensation of warmth 39%
- Nausea 35%
- Diaphoresis 33%
- Altered hearing 25%
- Sharp frontal headache 15%
- Mild tachycardia 13%

Syncope—Interventions

- Treatment is directed toward underlying cause
- If assessment reveals a potentially life-threatening cause of syncope:
  - Secure the airway
  - Initiate pulse oximetry and cardiac monitoring
    - Administer supplemental oxygen if indicated
  - Establish vascular access if possible
  - Check blood glucose levels and treat for hypoglycemia as indicated
**Syncope—Interventions**

- If the child appears stable and there are no findings to indicate a potentially life-threatening cause of syncope:
  - Allow the child to maintain a position of comfort
  - Keep the child warm
  - Provide reassurance

**Chest Pain**

- Most common causes of pediatric chest pain:
  - A pathologic condition of the chest wall
    - Trauma or muscle strain
  - Costochondritis
  - Respiratory disease
Chest Pain

- Chest pain
  - Can occur in a child of any age
  - Rarely has a life-threatening cause
  - Relatively infrequent chief complaint in the young child
    - Increases in frequency as the child ages
- No cause for chest pain can be found in 12% to 45% of patients

Chest Pain

- Cardiac dysrhythmias
- Marfan syndrome
- Pericarditis
- Myocarditis

Chest Pain—Interventions

- If a cardiac cause for chest pain is suspected:
  - Apply a pulse oximeter and administer supplemental oxygen if indicated
  - Initiate cardiac monitoring
  - Establish IV access if possible
  - Consult a pediatric cardiologist
- Reassess frequently
Congenital Heart Disease

Acyanotic Heart Defects

- Classified according to their hemodynamic effects
  - Increased pulmonary blood flow
    - Atrial septal defect (ASD)
    - Ventricular septal defect (VSD)
    - Patent ductus arteriosus (PDA)
  - Obstruction to blood flow from the ventricles
    - Coarctation of the aorta (COA)
    - Aortic stenosis (AS)
    - Pulmonary stenosis (PS)

Atrial Septal Defect (ASD)
Cyanotic Heart Defects

- May be classified according to their hemodynamic effects
  - Decreased pulmonary blood flow
    - Tetralogy of Fallot (TOF)
    - Tricuspid atresia
  - Mixed blood flow
    - Transposition of great vessels (TGV)
    - Total anomalous pulmonary venous return or communication
    - Truncus arteriosus
    - Hypoplastic heart syndrome
Tetralogy of Fallot

- Four (tetra) elements of TOF:
  - A large ventricular septal defect
  - Narrowing (stenosis) at or just below the pulmonary valve (pulmonary stenosis)
  - A right ventricle that is more muscular than normal (right ventricular hypertrophy)
  - The aorta lies directly over the ventricular septal defect (overriding aorta)

Tetralogy of Fallot

- Severe cyanosis of the lips, tongue, and mucous membranes associated with marked clubbing and cyanosis of the nails

Tetralogy of Fallot—“Tet” Spell
Heart Failure—History

- Poor feeding of recent onset due to fatigue and shortness of breath
- Tachypnea that worsens during feeding
- Diaphoresis on the forehead and/or back of the neck during sleep and feeding
- Poor weight gain
- Increased fatigue, long naps, easy fatigability
- Shortness of breath with activity
- Peripheral edema appearing first around the face and eyes, later in the hands and feet
Heart Failure—Physical Examination

- Cyanosis that worsens with crying
- Tachypnea, often above 50 breaths per min
- Tachycardia: resting heart rate above 150 beats/min in infants or above 100 beats/min in children
- Crackles
- Wheezes
- Increased work of breathing, retractions
- Diaphoresis

Heart Failure—Physical Examination

- Peripheral pulses may be diminished
- Third heart sound
- Blood pressure may be elevated or low depending on the cause of heart failure
- Hepatomegaly

Heart Failure—Interventions

- Semi-Fowler position
- Apply a pulse oximeter
  - Administer supplemental oxygen if indicated
- Initiate cardiac monitoring
- Minimize stress and energy output
- Monitor intake and output, electrolytes, hematocrit
- Daily weight measurement
- Administer a rapid-acting diuretic
Cardiomyopathy

- Disease of the heart muscle itself

  - Primary types
    - Dilated
    - Hypertrophic
    - Restrictive

Kawasaki Disease

- Inflammation of the walls of small and medium-sized arteries throughout the body

- Leading cause of acquired heart disease in children

- Possible causes
  - Bacteria, viruses, and environmental chemicals or pollutants
  - None has proven to be the cause of the disease
Kawasaki Disease

- Usually occurs in children 6 months to 5 years of age
- Occurs year round but is more common in the winter and spring
- In North America, the highest incidence rates are in children of Asian ethnicity
  - Especially those of Japanese or Korean background
- Associated with coronary artery aneurysms in approximately 25% of cases

Kawasaki Disease

- Fever (usually >103°F) for 5 or more days and presence of at least four of following five principal features:
  - Skin rash
  - Swollen, dry, cracked lips or a red tongue with small, raised bumps (papillae)
  - Red (“bloodshot”) eyes
  - Swollen lymph nodes in the neck
  - Swelling and redness of the hands and feet

Kawasaki Disease—Interventions

- Cardiac monitoring
- IV immunoglobulin
- Aspirin
- Diagnostic studies
Questions?