How to Treat Bradycardia’s
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Introduction
The major ECG rhythms classified as bradycardia include:

1. Sinus Bradycardia
2. First-degree AV block
3. Second-degree AV block
4. Type I — Wenckenbach/Mobitz I
5. Type II — Mobitz II
6. Third-degree AV block complete block

Sinus Bradycardia
Bradycardia is defined as any cardiac rhythm with a heart rate less than 60 beats per minute. (The AHA pushes this rate down to <50/min). This could also be called asymptomatic bradycardia. Bradycardia can be a normal non-emergent rhythm. For instance, well trained athletes may have a normal heart rate that is less than 60 bpm.

Symptomatic Bradycardia
Symptomatic bradycardia however is defined as a heart rate less than 60/min that elicits signs and symptoms, but the heart rate will usually be less than 50/min. Symptomatic bradycardia exists when the following 3 criteria are present: 1.) The heart rate is slow; 2.) The patient has symptoms; and 3.) The symptoms are due to the slow heart rate.

Bradycardia Algorithm
When a patient is suffering from Symptomatic Bradycardia; there are 3 medications that may be used in the Bradycardia ACLS Algorithm. These are atropine, dopamine (infusion), and epinephrine (infusion).

- **Atropine:** The first drug of choice for symptomatic bradycardia. Dose in the Bradycardia ACLS algorithm is 0.5mg IV push and may repeat up to a total dose of 3mg.
- **Dopamine:** Second-line drug for symptomatic bradycardia when atropine is not effective. Dosage is 2-10 micrograms/kg/min infusion.
- **Epinephrine:** Can be used as an equal alternative to dopamine when atropine is not effective. Dosage is 2-10 micrograms/min.
Healthcare Providers may also opt to use Transcutaneous Pacing.

**Transcutaneous pacing (TCP)**

Preparation for TCP should be taking place as atropine is being given. If atropine fails to alleviate symptomatic bradycardia, TCP should be initiated. Ideally the patient should receive sedation prior to pacing, but if the patient is deteriorating rapidly, it may be necessary to start TCP prior to sedation.

Do not delay TCP for the patient with symptomatic bradycardia with signs of poor perfusion. TCP rate should use 60/min as a starting rate and adjust up or down based on the patient’s clinical response. The dose for pacing should be set at 2mA (milliamperes) above the dose that produces observed capture.

TCP is contraindicated for the patient with hypothermia and is not a recommended treatment for asystole.

For the patient with symptomatic bradycardia with signs of poor perfusion, transcutaneous pacing is the treatment of choice.

Identification of contributing factors for symptomatic bradycardia should be considered throughout the ACLS protocol since reversing of the cause will likely return the patient to a state of adequate perfusion.

**How Do You Know?**

The healthcare provider may choose an ACLS intervention from the bradycardia algorithm if they determine that the patient does not have adequate perfusion. For the patient with adequate perfusion, you should observe and monitor. If the patient has inadequate cardiac perfusion demonstrated by temperature, heart rate, and blood pressure. The healthcare provider should prepare for transcutaneous pacing or one of the medications in the bradycardia algorithm. The healthcare provider and ACLS team should also think about the patient’s other contributing causes (**H’s and T’s**).
ACLS and PALS H’s and T’s

Introduction

Knowing the H’s and T’s of ACLS and PALS will help prepare the healthcare provider for any ACLS/PALS scenario. The H’s and T’s stand for a mnemonic used to help providers in their secondary assessment of the patient which may be major contributing factors to pulseless arrest including PEA, Asystole, Ventricular Fibrillation, and Ventricular Tachycardia. These H’s and T’s will most commonly be associated with PEA, but they will help the provider direct their care towards a potential underlying causes to any of arrhythmias associated with ACLS/PALS.

The H’s include:

Hypovolemia—Hypoxia

Hydrogen ion (acidosis)—Hyperkalemia—Hypokalemia

Hypoglycemia—Hypothermia.

The T’s include:

Toxins—Tamponade (cardiac)

Tension pneumothorax—Thrombosis (coronary and pulmonary)

Trauma
Hypoxia
Hypoxia is the deprivation of adequate oxygen and can be a significant contributing cause to cardiac arrest. The health care provider must ensure that the patient’s airway is open, and that the patient has chest rise and fall and bilateral breath sounds with ventilation.

Hydrogen ion (acidosis)
To determine if the patient is in acidosis, an arterial blood gas must be performed. To prevent respiratory acidosis the provider must ensure to provide adequate ventilation and oxygenation. Metabolic acidosis can be treated by giving the patient sodium bicarbonate.

Hyperkalemia
High potassium levels can be a significant contributor to cardiac arrest. The major sign of hyperkalemia or high serum potassium is taller and peaked T-waves. Also, a widening of the QRS-wave may be seen. Hyperkalemia can be treated in several ways such as; sodium bicarbonate (IV), glucose and insulin, calcium chloride (IV), Kayexalate, and dialysis.

Hypokalemia
Low potassium levels can also cause significant issues. The major signs of hypokalemia are flattened T-waves, prominent U-waves, and possibly a widened QRS complex. Treatment of hypokalemia involves administration of IV or oral potassium. Administering IV potassium has risks and should be administered at 10 meq/hr via a peripheral IV or 20 meq/hr via a central line. Healthcare providers should always follow the appropriate infusion standards.

Hypoglycemia
Hypoglycemia can have many serious negative effects on the body, and it can be associated with cardiac arrest as well as altered mental status. Treatment of hypoglycemia with IV dextrose to reverse a low blood glucose is typically the treatment of choice for patients with a blood sugar of 50 or less. (Hypoglycemia is no longer a part of the ACLS/PALS H’s and T’s)

Hypothermia
If a patient has been exposed to the cold, warming measures should be taken. A hypothermic patient may be unresponsive to drug therapy as well as to defibrillation and/or pacing. Core temperature should be raised in order to have an effective outcome for these patients.

Toxins
Accidental and incidental overdose of several types of medications can cause pulseless arrest. Some of the most common include: tricyclics, digoxin, beta blockers, and calcium channel blockers). Other various types of
illegal drugs and chemicals can also bring about cardiac arrest. Cocaine is the most common illegal drug that increases incidence of pulseless arrest. ECG signs of toxicity include prolongation of the QT interval. Physical signs include bradycardia, pupil symptoms, and other neurological changes. Support of circulation while an antidote or reversing agent is obtained is of primary importance. Healthcare providers should contact Poison control at 1-800-222-1212.

**Tamponade**
Cardiac tamponade is a medical emergency in which fluid accumulates in the pericardial sac. The buildup of fluid results in the ineffective pumping of the blood which can lead to pulseless arrest. ECG symptoms include narrow QRS complex and rapid heart rate. Physical signs include jugular vein distention (JVD), no pulse or difficulty palpating a pulse, and muffled heart sounds due to fluid inside the pericardium. The recommended treatment for cardiac tamponade is pericardiocentesis.

**Tension Pneumothorax**
Tension pneumothorax occurs when air is allowed to enter the plural space and is prevented from escaping naturally. This leads to a buildup of tension that causes shifts in the intrathoracic structure that can rapidly lead to cardiovascular collapse and death. ECG signs include narrow QRS complexes and slow heart rate. Physical signs include JVD, tracheal deviation, unequal breath sounds, difficulty with ventilation, and no pulse felt with CPR. Treatment of tension pneumothorax is needle decompression.

**Thrombosis (Coronary)**
Thrombosis can come in the form of a Coronary thrombosis or Pulmonary Thrombosis. A Coronary thrombosis is an occlusion or blockage of blood flow within a coronary artery caused by blood that has clotted within the vessel. The clotted blood causes an acute myocardial infarction which destroys heart muscle and can lead to sudden death depending on the location of the blockage.

ECG signs during PEA indicating coronary thrombosis include ST-segment changes, T-wave inversions, and/or Q waves. Physical signs include: elevated cardiac markers on lab test.

**Thrombosis (pulmonary embolism)**
Thrombosis can come in the form of a Coronary thrombosis or Pulmonary Thrombosis. A Pulmonary thrombus or pulmonary embolism (PE) is a blockage of the main artery of the lung which can rapidly lead to respiratory collapse and sudden death. ECG signs of PE include narrow QRS Complex and rapid heart rate. Physical signs include distended neck veins, positive d-dimer test, and prior positive test for DVT or PE. Treatment includes fibrinolytic therapy.

**Trauma**
Trauma may also be a cause of pulseless arrest. Proper assessment of the patient’s physical condition and history should reveal any traumatic injuries. Treat trauma injuries individually as needed to correct any reversible cause or contributing factor to the pulseless arrest. Trauma also has been removed from the T’s but is still to be considered important during the assessment of any person in cardiac arrest.