SYMPTOMATIC BRADYCARDIA

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Outline:

- Example case
- Definition of Symptomatic Bradycardia and associated symptoms
- Causes of Symptomatic Bradycardia
- Review of PALS
- How transcutaneous pacing is administered and how it works

Case:

- 2yo FT/PH F is bib father to the ED for copious NBNB vomiting (>10 times) and diarrhea (~5 very water stools) that started 1 day prior to admission. No fever, had about 2 wet diapers today and is not able to keep fluids and patient started becoming more lethargic and unresponsive so dad brought her in.
- No trauma, no toxic ingestion, no sick contacts/recent travel/animal exposures, ROS otherwise normal
- No PMH or PSH, UTD on immun., no develop. concerns, no medical issues in family, no meds/allergies

Physical Exam

- Vitals: Temp 37 C, RR 32, HR 50, BP 68/30, SaO2 85% and dropping
- PE: pt obtunded and unresponsive to stimuli, pupils reactive b/l, bradycardic but regular rhythm, CTAB but tachypneic, abd s/nd, gag/patellar reflexes intact, no bruises/rashes, pulses +2 b/l in all extremities, cap refill ~4 secs in the peripheries

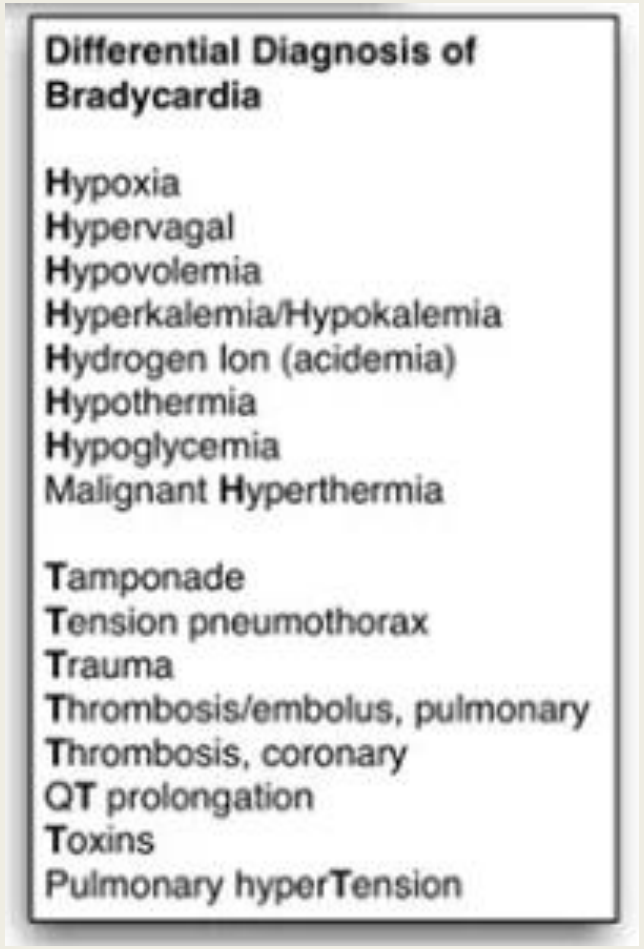
What is your next step?

- Call for help! Need to resuscitate the pt quick
- What are the most worrisome features here?
  - *Pt is unresponsive and vitals show bradycardia, hypotension, low saturations*
- What is going on here?
  - *Pt is bradycardic and has poor perfusion!* = ***Symptomatic bradycardia***

Differential for symptomatic bradycardia

- Primary bradycardia:
  - *Intrinsic to the heart and it's pacemaker cells/conduction system*
  - *Ex: Myocarditis, congenital abnormalities, cardiomyopathy, arrythmias (long QT syndrome, Complete heart block, 1st degree AV block, 2nd degree AV block-Mobitz 1 &2, 3rd degree AV block)*

- Secondary bradycardia:
  - *Caused by non-cardiac problems*
    - Medications (beta-blockers, verapamil, amiodarone, lithium, clonidine, etc.)
    - Hypoxia (number one cause in kids)
    - Acidosis
    - Hypotension/hypovolemia
    - Hypothermia (due to basal metabolic rate dropping)
    - Hypothyroidism
    - Increased intracranial pressure (think Cushing's triad)



Symptomatic Bradycardia

- Defined as poor circulation/perfusion and shock-like symptoms secondary to low heart rate
- Symptoms include hypotension, decreased level of consciousness/lethargy, fatigue, syncope, weak pulses, possible organ injury, and cardiac arrest
- Various definitions of bradycardia (but here's one from *Journal of Pediatric Cardiology*):
  - *0 to 3 months: <126 bpm*
  - *3 to 6 months: <116 bpm*
  - *6 to 12 months: <106 bpm*
  - *1 to 3 years: <97 bpm*
  - *3 to 5 years: <77 bpm*
  - *5 to 8 years: <64 bpm*
  - *8 to 12 years: <59 bpm*
  - *12 to 16 years: <53 bpm*

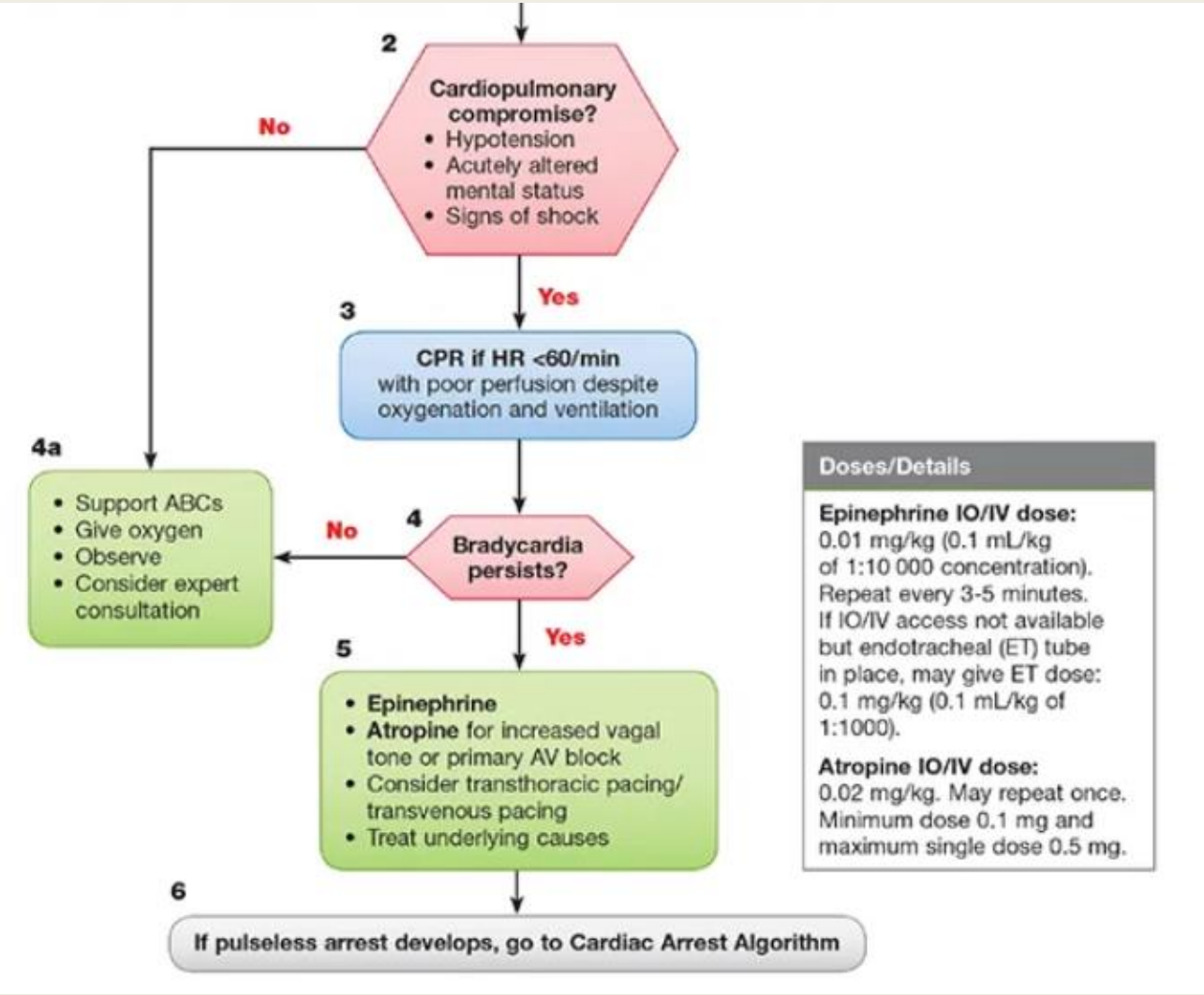
Hypervagotonia

- Bradycardia induced by exaggerated vagal activity (increased parasympathetic tone)
- Can be caused by:
  - *Nasopharyngeal/esophageal stimulation*
  - *Breath holding spells*
  - *OSA*
  - *Reflux and vomiting*
  - *Coughing*
- These are usually transient; however increased parasympathetic activity is thought to be the reason how metabolic acidosis, hypoxemia and hypotension leads to bradycardia, although the mechanism is not quite understood
- AV node dysfunction may also occur and contribute to symptoms
- The combination of slow HR and peripheral vascular resistance are often the cause of symptoms such as syncope and leads to pt's becoming symptomatic

Treatment

- Stabilizing the pt is most important, but should run through differential and think about the reversible causes of SB.
- Initial interventions:
  - *provide oxygen*
  - *cardiac monitor for rhythm identification*
  - *monitor blood pressure*
  - *monitor pulse oximetry*
  - *obtain IV or I/O access*
  - *obtain 12-lead ECG if possible*

PALS Algorithm

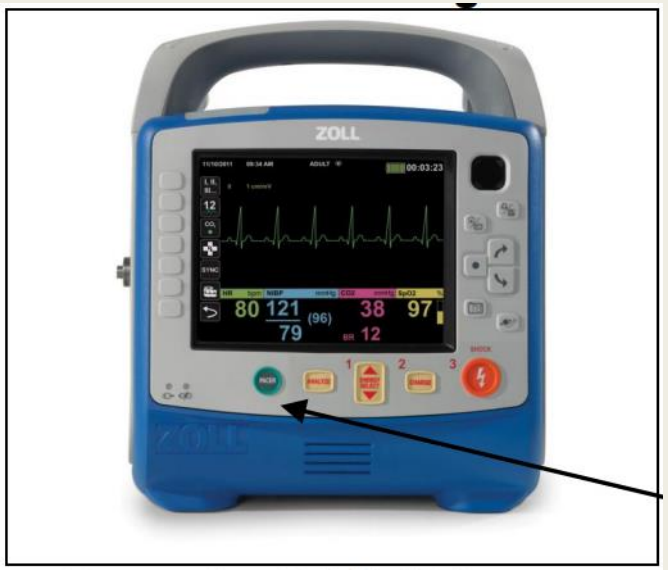


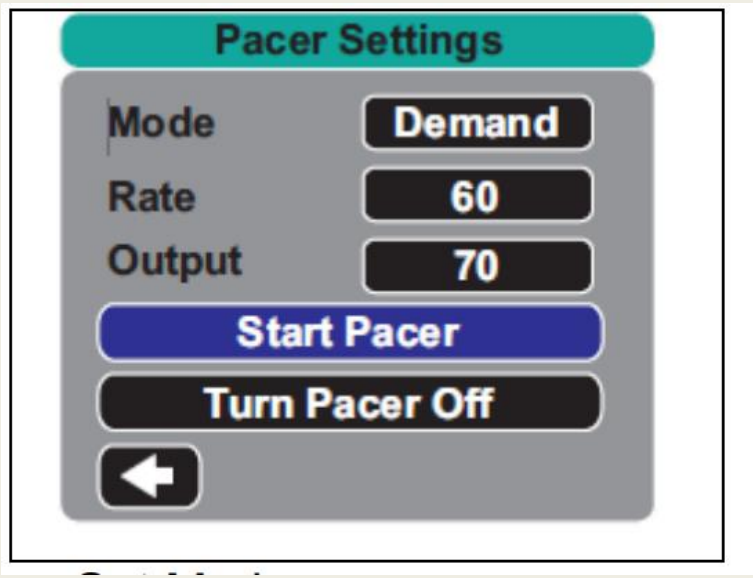
Why Epinephrine is 1st line over Atropine

- Atropine works by affecting the vagus nerve and removing parasympathetic inputs to the heart
  - *Works well for bradycardia 2/2 vagal reflexes, cholinergic drugs*
  - *However it fails for bradycardia 2/2 mechanisms beyond the AV node*
  - *According to one study, atropine was effective in only 28% of pts with symptomatic bradycardia (Brady 1999, Journal of Resuscitation - adult study)*
- Epinephrine stimulates the entire myocardium, including the AV node and may even stimulate atropine-responsive bradycardias
- Epinephrine also causes increased HR, myocardial contractility, arterial and veno-constriction which all helps combat the *shock* experienced by pts in symptomatic bradycardia
- Atropine at lower doses can actually cause/worsen bradycardia
  - *Also seen with slower infusion of atropine*
- It is the higher doses of atropine that lead to tachycardia and overcome parasympathetic activity

When medical management fails?

- Next is to set up Transcutaneous pacing
- Pacing applies electrical current to essentially manually pace the heart to the heart rate you set



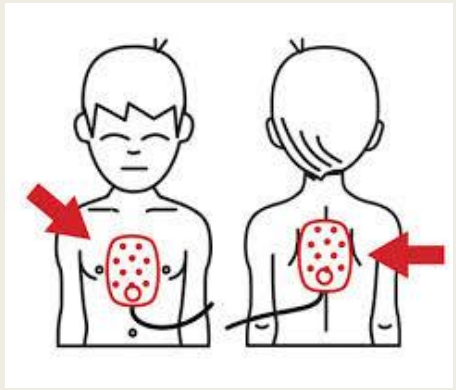


All AEDs are different, be familiar with the ones that are available at your institution

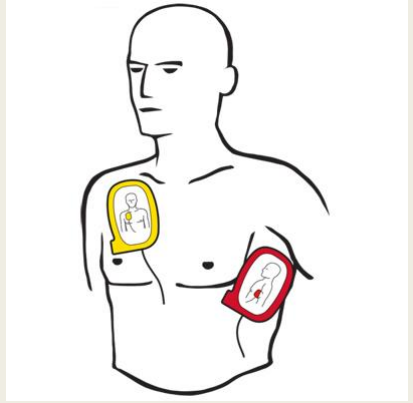
How to set up the Pacer

- 1. Apply pads/paddles (hopefully this is being set up during your resuscitation)

< 10 kg



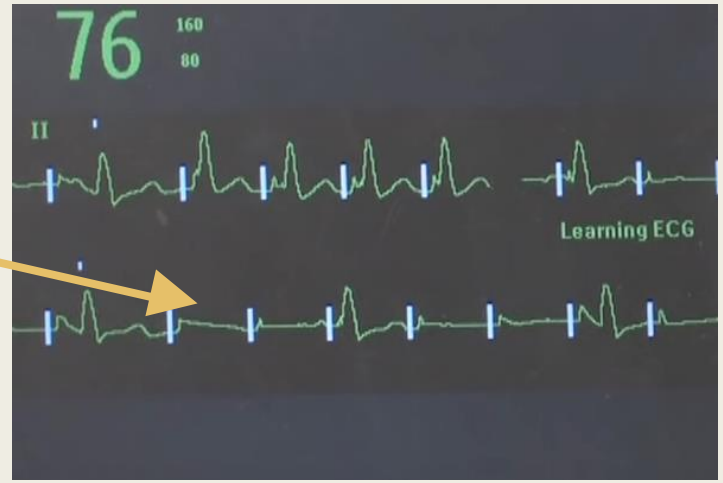
> 10 kg



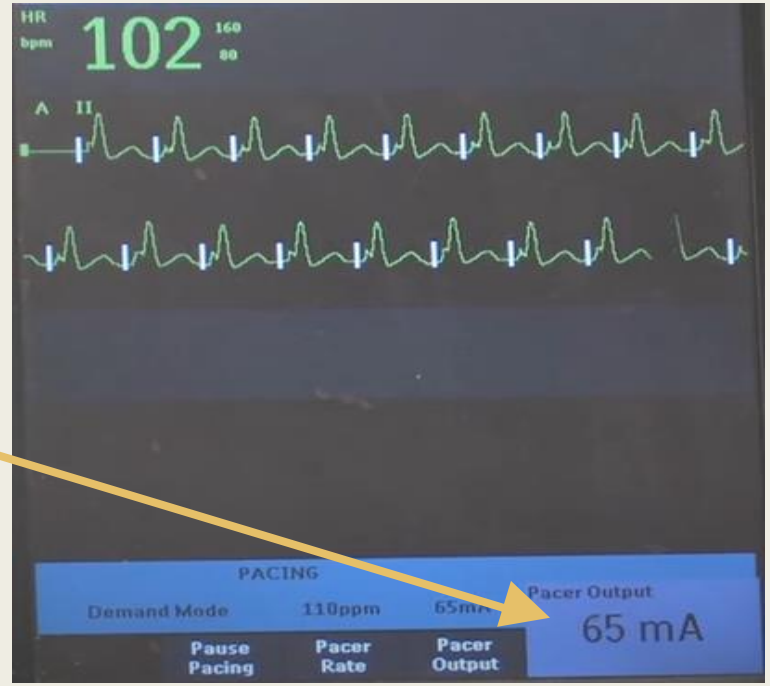
- 2. Set electrical energy output (mA): General rule of thumb- start with 20mA or 40mA and adjust accordingly (*next slide*)
- 3. Set your pacer rate that is appropriate for their age
- 4. Press the “Sync” Button: Synchronize your pacer with ECG of the patient, this allows your pacer to send electrical current while the ventricles are depolarizing (*R wave in the cardiac cycle*)

Assessing Capture

- Here, your pacer is not providing enough electrical energy to adequately depolarize the heart with charge



- In this case, increase the mA (electrical energy) until there is ventricle spike in your EKG
  - *This pt required a minimum of 65mA of output to do this*



Good things to keep in mind

- Typically the minimum amount of energy needed to have a QRS spike is called the “Threshold Level”
- Good rule of thumb is to keep your output 10mA above the threshold
- Important to know that you can continue to assess and touch the patient while your pacer is sending out electrical energy.
  - *This is unlike defibrillation or cardioversion where you must “clear” away from the pt when diffusing a charge*
  - *This is partly because the electrical energy in transcutaneous pacing is set off in a longer amount of time which helps diffuse the energy and is generally a lower degree of output as well*
- Transcutaneous Pacing is a temporary measure to initially stabilize the pt, please continue to evaluate for causes of bradycardia and treat them
- Continue to work-up your patient with labs/imaging during your resuscitation to help diagnose the underlying etiology

4 H's	MANAGEMENT
Hypoxia	➢ Check and maintain airway ➢ Insert Guedel, ETT, LMA, surgical airway if required ➢ Check oxygenation and ventilation
Hypovolaemia	➢ Replace blood or fluid loss Replacement of blood with: - Crystalloid/ Colloid - Blood Products ➢ Anaphylaxis: Management of ABC - Adrenaline (IMI, S/C, or IV) - Hydrocortisone - Correct hypovolaemia
Hypo/Hyperkalaemia	Hypokalaemia ➢ Potassium of less than 3.5mmol/L ➢ Replace Potassium ➢ Hyperkalaemia ➢ IV calcium, 10 mLs 10% CaCl2, up to 3 ampoules, each over 5 minutes ➢ hyperventilation: $CO_2 + H_2O \Leftrightarrow H_2CO_3 \Leftrightarrow H^+ + HCO_3^-$ ➢ 50mls 50 % glucose + 10 units Actrapid over 10-15 minutes. ➢ $NaHCO_3$ to correct acidosis ➢ Nebulised salbutamol
Hypo/Hyperthermia	Hypothermia ➢ Active core re-warming ➢ Warmed humidified oxygen ➢ Warmed intravenous fluids ➢ Peritoneal lavage ➢ Extracorporeal warming ➢ Pleural lavage Hyperthermia ➢ Cooling Blankets ➢ Cooling packs or ice to head, axilla, chest, groin and legs ➢ Cooled IV fluids

4 T's	MANAGEMENT
Tamponade	➢ Pericardiocentesis ➢ open sternotomy wound if post cardiac surgery
Tension Pneumothorax	➢ Thorococentesis -Chest tube insertion if there is time or a large bore needle through the 2nd intercostal space in the mid-clavicular line
Toxins/tablets	➢ Antidote ➢ Charcoal (within 1 hr of ingestion) ➢ Supportive measures ABCDEFG
Thrombus	➢ Thrombolysis, embolectomy or cardiopulmonary bypass to allow operative removal of the clot.