RECAP EM
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# Structured Approach To Assessment Of Bradycardia In ED

1. Identify Critically ill Patients
2. Find The Cause
3. Risk Stratify

## Step 1: Is This Patient Critically ill (Unstable)?

### Unstable Vitals

-MAP < 65? or "SI" > 0.8?
-Extreme Brady (e.g. HR < 30), or Acute Worsening Bradycardia?
-Hypoxia (e.g. $SpO_2$ < 90% despite Supp $O_2$)?
-RR <<12? or RR >>24?
-Temp in extremis? 🥶 Hypothermic?
-BG? 😰💫 Hypoglycemic?

### End-Organ Dysfunction

😴 Altered Mental Status?
😫 Ischemic Chest Pain? 🤦🏾‍♀️Syncope?
😮💨 Acute Dyspnea (from Heart Failure, i.e., Pulmonary Edema 💧)?
😨 Cold, clammy, mottled skin?
🧠⚡ Seizure?
🧠 Focal Neurologic Deficit?

### Assessment

- Focused H&P
- Vitals Monitoring
- POCUS
  - RUSH protocol
    - H- Heart
    - I- IVC
    - M- Morrison's Pouch
    - A- Aorta
    - P- Pulmonary
- i-STAT
  - Electrolytes
  - Lactate
  - Troponin
- 12-Lead ECG

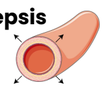
**Shock exists on a spectrum**
Early Signs of Bradycardic Shock Can Be Subtle.
Don't Overlook Occult Bradycardic Shock. Compensatory Mechanisms Can Mask shock with Normal BP.

## Step 2: Is This Brady "Driving" Clinical Presentation?

📝 **History**: Medications, Comorbidities, Permanent Pacemaker (PPM)?

**Severity of Bradycardia?**

- 💡 Bradycardia can directly reduce cardiac output (CO), but a healthy heart can compensate by increasing CO.
- 💡 However, **Extreme Bradycardia** (e.g., <30s bpm) itself can cause Hemodynamic Instability.
- 💡 Mild-Moderate Brady is well-tolerated, unless something else is going on like:

**Systolic Heart Failure** 
**Vasodilation, e.g., Sepsis** 
**Structural Heart Disease** e.g., AR, MR. 

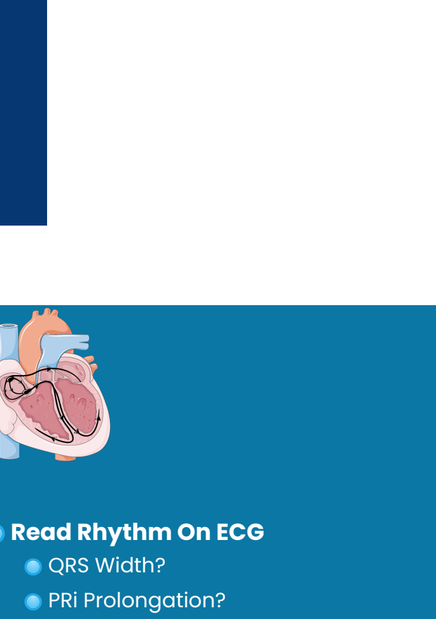

🔗 **Symptom Correlates?**
🔁 **Consider Reversible Causes of Bradycardia?**
💡 Don't Let Bradycardic Patients **DIE**

**Don't DIE**

- **D**rugs 💊 — BBs, CCBs, Digoxin, Clonidine
- **I**nfarction ❤️‍🔥 — ICP Rise 🧠
- **E**lectrolytes (K) ↑↓K,↑↓Ca, ↑Mg, BRASH
  **E**ndo/metabolic ↓Temp.,↓BG, ↓Thyroid, ↓SpO2%

### Assessment

- ECG
  - Signs of ↑**K**?
  - Sign of **Acute MI?**
  - Signs of **Dig Intox**?
  - PPM **Malfunction?**
  - **Cerebral TWI?**
- Labs
  - Drug level
  - Renal/Liver/Thyroid Profile
- Imaging
  - Head CT
  - CXR
- POCUS
  - Optic nerve sheath diameter?
  - Intra-abdominal Free Fluid?



**Interpret Numbers In Clinical Context. For example:**

- Bradycardia → ↓ Cardiac Output → Confusion / Drowsiness
  - ⚪ Bradycardia can ↓ cerebral perfusion, leading to "mild" mental status changes.

‼️But, if mental status is severely depressed (e.g., coma with HR ~40), Think beyond bradycardia itself — consider:

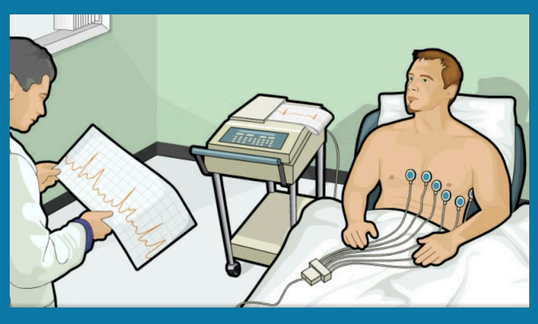
- 💊 Drug overdose, e.g., β-blockers, CCBs.
- 🧠 Neurologic → ↑ ICP (Cushing's reflex).

## Step 3: Risk Stratify

**Where Is The Likely Anatomic Location Causing Bradycardia? Nodal vs Infra-nodal?**



### ECG Analysis



**Read Rhythm On ECG**

- QRS Width?
- PRi Prolongation?
- Rate of Escape Rhythm?

**In Context of AMI ?**

- Inferior MI: More likely Nodal block
- ⚠️ Anterior MI: More likely Infra-nodal block

👩 **Nodal Block: Better prognosis, Good response to atropine.**

- ✔QRS Width < 120 msec
- ✔PRi Prolongation before the block
- ✔Rate of Escape Rhytm 40 -60 bpm

⚠️ **Infra-nodal Block: Worse prognosis, Poor response to atropine.**

- ❗QRS Width > 120 msec
- ❗No/Minimal PRi Prolongation before the block
- ❗Rate of Escape Rhytm< 40 bpm

**Conditions With High Risk of Progression To Asystole**

- ❗Recent asystole.
- ❗Mobitz type II AV block.
- ❗Complete AV block with wide QRS.
- ❗Ventricular pause > 3 seconds