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EM:RAP C3
Continuous Core Content

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Bradyarrhythmias

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* *Drug doses are a guide only, always check second source and follow local practice guidelines*

Take Home Points:

- **Patients with bradycardias can be divided into groups based on a series of simple questions, leading to a stepwise approach:**
  - **STEP 1: IV, O2, monitor, crash cart with airway equipment, defibrillator and pacemaking equipment to bedside**
  - **STEP 2: Is the patient stable or unstable?**
  - **STEP 3: Are the QRS complexes wide or narrow?**
  - **STEP 4: Are there P waves before each QRS?**
  - **STEP 5: Could this be:**
- **Ischemia? (think ACS)**
- **Electrolytes? (think K+)**
- **Drug Induced? (think, beta blocker)**
- **Hypothermia? (think temp=78 F!)**
- **Endocrine? (think hypothyroid)**
- **With the notable exceptions of hypothermia and hypothyroidism (myxedema coma), the goal of treatment is to increase the heart rate to achieve an adequate cardiac output**
- **Treatment generally begins with atropine and adrenergic drugs (epinephrine, dopamine) and proceeds to electrical pacing (transcutaneous and transvenous)**

Introduction

In last month's C3, we discussed the concept of *primacy of rate*. Simply stated, the more abnormal the heart rate, the more likely that it is responsible for the patient's symptoms. In most cases (but not all - e.g. not in hypothermia and myxedema coma) intervening to correct the rate is a high priority in the resuscitation.

- Managing patients with bradycardia is similar to managing patients with tachycardia. How is that? The more extreme the heart rate - e.g., really fast or really slow, the more aggressive treatment will be. And by aggressive, we generally mean electricity: *cardioversion* in the case of tachyarrhythmias and *electrical pacing* in the case of bradydysrhythmias.
- The other key similarity is that whether the heart rate is 25 or 150, our treatment decisions will be largely based on how the patient looks, not their actual rate.
  - On one end of the spectrum, a patient may be tolerating their crazy slow heart rate just fine with a normal blood pressure.
  - On the other end, they may be unconscious and on the verge of cardiac arrest. A patient with a heart rate of 40 who says they feel just fine may not need any intervention at all. Keep in mind that all interventions, whether drugs or electricity, may do harm as well as good!

The Approach

- **STEP 1: IV, O2, monitor, crash cart with airway equipment, defibrillator and pacemaking equipment to bedside**
  - Pacemaking equipment may include both transcutaneous and transvenous pacing set-up
  - Transvenous pacers are most easily floated into the right ventricle through a right internal vena caval or left subclavian central line
- **STEP 2: Is the patient stable or unstable?**
  - This traditional definition of "unstable" includes chest pain, altered mental status, pulmonary edema or systolic BP < 90 mmHg
    - Even in unstable patients, medical management is likely to occur more quickly and is just as likely to help as electrical pacing!
- **STEP 3: Are the QRS complexes wide or narrow?**
  - In general, if wide complexes are of more concern because they represent a ventricular rhythm and in the case of heart block, a more severe block
- **STEP 4: Are there P waves before each QRS?**
  - In general, if there are QRS complexes *without* P waves in front of them, a severe form of block (2nd degree Mobitz

Type II or 3rd degree AV block) is present
  - If there are P waves in front of most of the QRS complexes, a less severe form of block (2nd degree Mobitz Type I/Wenckebach) is present

- **STEP 5: Could this be:**
  - Ischemia? (think ACS)
  - Electrolytes? (think K+)
  - Drug Induced? (think beta blocker, other drugs)
  - Hypothermia? (think temp = 78 F!)
  - Endocrine? (think hypothyroid emergency)

Universal Treatment Algorithm

- Cardioactive drugs
  - Atropine 0.5mg IV increments
    - May redose every 5 minutes to 2-3g maximum
    - Higher doses necessary in organophosphate poisoning
  - Epinephrine
    - ACLS dose range is 2 to 10 mcg/min
    - May give 0.1mg increments (one-tenth of crash cart epi dose)
    - "Dirty" epi drip
- 1 L bag of NS
- Inject 1 amp of epi (either 1/1,000 or 1/10,000)
- This yields 1 mcg / ml of epinephrine
- Running this through a 18 gauge IV wide open gives them 20-30 mcg/min
  - Dopamine
    - ACLS dose range is 2 to 20 mcg/kg/min
- Pacing
  - Transcutaneous
  - Transvenous

Specific Scenarios

- Scenario: Bradycardia + **Chest Pain/Dyspnea**
  - Ischemia can cause either **tachycardia** or **bradycardia**
  - Tachycardia in the setting of chest pain/dyspnea occurs as a reflex to low cardiac output due to:
    - CHF as a result of a large MI
    - PE as a result of decreased preload
    - increased adrenergic tone (e.g. with the severe pain of dissection or MI)
- Scenario: Bradycardia + **Acute Coronary Syndrome/MI**
  - *Bradycardia* in ACS may signify ischemia of the conduction system or a vagal response
    - Bradycardia in inferior MI is usually narrow complex, less severe, transient and caused by vagal stimulation
      - Usually responsive to atropine
    - Bradycardia in anterior MI or is often wide complex, severe and caused by infarcted conduction tissue
      - Usually not responsive to atropine
    - **Nitroglycerin** may result in a reflex tachycardia (due to a drop in preload and afterload) but may also slow heart rate and is contraindicated in severe bradycardia
- Scenario: Bradycardia + **Syncope**
  - 3rd degree AV block (Stokes-Adams attacks)
  - Sick sinus syndrome (SSS)
    - Patients with SSS may have both tachycardias *and* bradycardias (sinus arrest)
- Scenario: Bradycardia + **Overdose**
  - The most common agents to result in bradycardia are:
- The "Brady Bunch"
  - Digoxin
  - Beta-blockers
  - Calcium-channel blockers
  - Clonidine
- All of these will result in profound bradycardia but other clues may identify which one is involved - these include:
  - Digoxin: ventricular arrhythmias, systemic symptoms
  - Beta-blocker: hypoglycemia
  - Calcium-channel blocker: hyperglycemia
  - Clonidine: opioid-like toxidrome (like a heroin OD)
- Scenario: Bradycardia + **Altered Mental Status**
  - Patients with bradycardia can be drowsy or confused solely on the basis of having low cardiac output from a slow heart rate
  - However, when mental status is disproportionately depressed for a given heart rate (e.g. coma with a heart rate of 40), causes of the bradycardia such as overdose and increased ICP (Cushing's response) should be considered
- Scenario: Bradycardia + **Renal Failure**
  - Always think of high **potassium** as a cause of bradycardia!
  - Initial treatment is with IV calcium (chloride or gluconate) to stabilize myocytes
  - Other treatments include: Albuterol, HCO3, Insulin/D50, Furosemide, Kayexalate
- Scenario: Bradycardia + **Accidental Hypothermia**
  - Rhythm may be a sinus bradycardia or atrial fibrillation with a very slow ventricular response
  - In severe cases (temp<32C), Osborne J waves (a terminal notch in the QRS complex) may be seen
  - In less severe cases (e.g. temp >32C) patients may still be shivering which can cause an artifact on the ECG (may look like a course Afib or flutter)
  - **Treatment focuses on warming the patient, not the heart rate**

- Scenario: Bradycardia + **Myxedema Coma**
  - Rhythm may be a sinus bradycardia or atrial fibrillation with a very slow ventricular response
  - **Treatment focuses on thyroid replacement, not the heart rate or temperature**

Disposition

- Patients with symptoms (e.g. repeated loss of consciousness) or severe blocks resulting in low heart rates (usually <45) should be admitted to a monitored setting, typically a cardiac intensive care unit, in preparation for pacemaker placement
- Patients who are asymptomatic or suffer only from a mild block may be managed as an outpatient
  - The presence of a chronotropic response (e.g. the patient's heart rate increases appropriately as they get up from bed) is reassuring
  - Event monitors (e.g. 24 hour EKG monitoring) may be indicated

References

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