Bradycardia Mini-Course

Recent themes for GJFD:

- Incorrect atropine dosing.
- Prolonged times to atropine in symptomatic cases.
- Long scene times in symptomatic patients.
- Weak/inadequate documentation of EKG findings.
- No pacing used/considered in refractory cases.
- No Dopamine used/considered in refractory cases.

This makes me wonder:

Do we have a strong enough grasp of:

1. The differential diagnosis of bradycardia?
2. What, exactly, should you be looking for on the EKG; i.e. what should you be charting about?
3. What is the current AHA algorithm for bradycardia?
4. What EMS medications/strategies work best in what situations?
5. Review of AV block rules/anatomy/treatment.

What causes bradycardia?:

- Hypoxemia.
- Hyperkalemia.
- AV Blocks:
  - ~ medications
  - ~ electrolyte disturbances
  - ~ ischemia

Why get an EKG?:

1. Sinus brady or an AV block.
2. Hyperkalemia.
3. Ischemia.

Current AHA algorithm:

1. O2 (WOB?, sats?, oxygenate, ventilate).
2. Atropine 0.5mg IV q 3-5 min (max 3mg).
3. Transcutaneous Pacing.
4. Dopamine.

<u>Dery adds:</u>

Bicarb; EKG; ASA; ntg; GO

AV Blocks:

1. 1st degree AVB:
   ~ usually benign and asymptomatic.
2. Mobitz I 2nd degree AVB:
   ~ block is at AV node; usually asympto., benign.
3. Mobitz II 2nd degree AVB:
   ~ block likely below AV node at His/Purkinje.
   ~ often symptomatic, not responsive to Atropine.
4. Third degree AVB:
   ~ can be anatomically anywhere-AV node to BB's
   ~ usually does not respond to Atropine.

Atropine: (I and P)

1. 1st line for symptomatic bradycardia (AHA):
   ~ *temporizing measure* until pacer for sinus brady and blocks at level of AV node.
2. 0.5mg IV every 3-5 minutes; max dose 3mg.
   ~ <0.5mg may cause paradoxical bradycardia.
3. Be careful if ACS/AMI present:
   ~ increased HR may worsen ischemia or infarct size.
4. Avoid *relying* on atropine in Mobitz II/3rd degree AVB:
   ~ these are usually NOT at AV node- atropine doesn't work.
   ~ TCP, Dopamine, GO→ pacer in cath lab.

Transcutaneous Pacing: (I and P)

1. TCP has been compared to drug therapies
   ~ no differences in outcome or survival.
   ~ TCP obtained more consistent heart rates.
2. TCP is, at best, a *temporizing measure*.
   ~ "whether effective or not, the patient should be prepared for TCP" (AHA).
3. Initiate TCP in unstable patients who do not respond to atropine:
   ~ consider immediate TCP in unstable patient with "high-degree AVB; no IV.

Dopamine: (P only)

1. NOT considered 1st line for symptomatic bradycardia (AHA):
2. Good alternative when brady is:
   ~ unresponsive to atropine, or
   ~ inappropriate for atropine therapy.
3. Particularly useful if brady assoaciated with hypotension.
4. "Low dose" Dopamine (<10mcg/kg/min):
   ~ selectively targets heart rate.
5. "High" dose Dopamine (10-20mcg/kg/min):
   ~ adds vasoconstriction as well.
   ~ add volume/assess volume status.

So:

1. Stable/unstable.
2. Get rolling and therapy started ASAP.
3. O2, assess for hypoxemia.
4. IV/IO.
5. Atropine is first line medication:
   ~ consider TCP if high grade AVB; no IV/IO.
6. EKG:
   ~ ischemia; hyperkalemia; AVB's.
7. TCP for non-responders to atropine, "high-grade" AVB's.
8. Non-responders; hypotension ("low" vs. "high" dose).
9. Ischemia?: careful c atropine; ASA; ntg.
10. Hyperkalemia?: consider bicarbonate.