Bradycardia

January 2, 2017 by Josh Farkas


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why bradycardia is dangerous

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The effect of tachycardia on cardiac output is often over-estimated

- Tachycardia has a mixed impact on cardiac output:
  - Increase in heart rate tends to increase the cardiac output.
  - Decreased filling time tends to decrease the stroke volume, which decreases cardiac output.
- Mild-moderate tachycardia will generally increase cardiac output. This is a normal physiologic response to stress. The effect of increased heart rate predominates here.
- Severe tachycardia (heart rates >>150 b/m) may drop the cardiac output because the heart doesn’t have time to fill with blood during diastole, causing a reduced stroke volume.
- The deleterious effect of heart rate on cardiac output is often over-estimated. For example, if a patient has atrial fibrillation with a heart rate of 150 b/m, it’s unlikely that cardioversion or rate control will improve cardiac output. Usually, slowing down a moderate tachycardia will cause deterioration.

The effect of bradycardia on cardiac output is often under-estimated

- Bradycardia directly pulls down the cardiac output, potentially causing shock.
- Slowing down the heart rate may cause a minimal increase in diastolic filling, thereby increasing the stroke volume. However, this compensatory factor is weak and extremely limited. For example, if the heart rate decreases by a factor of two, the stroke volume cannot possibly double.
- In severe bradycardia, the cardiac output must be low. This is simple math.
- Cardiogenic shock is defined as inadequate cardiac output to support organ function (supply/demand mismatch). Some patients can compensate for low cardiac output without developing shock. However, with increasingly severe bradycardia there should be an increasing concern for cardiogenic shock.

don’t be fooled by normal-pressure bradycardia

Occult shock: normal-pressure bradycardia

#1) Bradycardia with vasoconstrictor response

Blood Pressure = (Cardiac Output)(Systemic Vascular Resistance)

#2) Bradycardia without vasoconstrictor response

Blood Pressure = (Cardiac Output)(Systemic Vascular Resistance)

#3) Bradycardia with massive sympathetic response causing hypertension (rare)

Blood Pressure = (Cardiac Output)(Systemic Vascular Resistance)
"the heart rate is 25 b/m but the blood pressure is fine... I think we can send her to the floor"

Some patients with bradycardia will maintain a normal blood pressure, due to an endogenous sympathetic response causing vasoconstriction. Despite a normal blood pressure, these patients still have a low cardiac output and still may be in shock.

Rare patients can present with severe bradycardia and severe hypertension (#3 in figure above). Hypertension is caused by a massive sympathetic response, as the body struggles to compensate for the bradycardia. This dangerous situation must be managed thoughtfully, because the sympathetic response is actually keeping the patient alive. Aggressive vasodilation to treat the 'hypertensive emergency' will cause hemodynamic collapse. Management should focus on correction of the bradycardia. Once the heart rate normalizes, the endogenous sympathetic response should relax and everything will resolve.

progressive bradycardia is often a harbinger of death

- Progressively worsening bradycardia is often seen immediately preceding death ("the patient is bradying down").
- If the patient's heart rate is consistently dropping in front of your eyes don't just stand there – get some epinephrine. Fast.
- The differential diagnosis of bradycardia here is broader than usual and may include such entities as severe hypoxemia and right ventricular failure from massive PE. Immediate evaluation should focus on the ABCs: airway, breathing, and circulation (bedside echocardiogram).

one more reason to fear bradycardia: torsade de pointes

- Torsade de pointes is a pause-dependent arrhythmia, which is more likely to occur at slower heart rates. Furthermore, bradycardia itself may prolong the QT interval. It's possible that leaving patients in a severely bradycardic state may increase their risk of torsade.

common causes

- Medication/intoxication
  - Beta-blocker or calcium-channel blocker
  - Clonidine, dexmedetomidine
  - Cholinergic medications
  - Digoxin, antiarrhythmics
  - Propofol infusion syndrome
  - Alpha-blockers (e.g. prazosin)
- Metabolic
  - Hypermagnesemia
  - Hypothyroidism, Hypothermia
  - Hypoglycemia
  - Severe hypoxia / hypercapnia / acidemia (sinus bradycardia is a common pathway of impending death from any cause)
- MI
- Neurologic catastrophe
  - Cushing's reflex due to increased ICP
  - Neurogenic shock
Infection
- Lyme disease, syphilis
- Aortic valve endocarditis with ring abscess (conduction block)
- Senile degeneration of sinus node or conduction system
- Failure of a permanent pacemaker

evaluation
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evaluation

physical exam

- Primary focus is adequacy of perfusion.
  - Overt bradycardic shock: altered mental status.
  - Occult bradycardic shock: Blood pressure and mental status intact, but cool extremities & poor urine output.
- Cardiopulmonary exam with ultrasound
  - Volume status?
  - Evidence of myocardial infarction (e.g. inferior wall motion abnormality)?
  - Evidence of pulmonary congestion (e.g. B-lines throughout the lung fields)?
- Neuro/toxicologic exam
  - Evidence of elevated intracranial pressure (e.g. stupor, widened optic nerve sheath [https://emcrit.org/pulmcrit/pulmcrit-algorithm-diagnosing-icp-elevation-ocular-sonography/])?
  - Pinpoint pupils may suggest toxic ingestion (e.g. clonidine or cholinergic agent)

EKG: Focus on three things

- Rhythm diagnosis (e.g. sinus bradycardia vs. heart block)
- Signs of hyperkalemia (e.g. peaked T-waves)
• Signs of ischemia

**medication review**

• Active medication list?
• Recent medication changes, including dose titration?
  • Some medications can unexpectedly cause bradycardia (e.g. donepezil, tizanadine). So if the patient just started a medication, look up whether it can cause bradycardia.
  • Even eye drops with sympatholytic properties may be enough to cause bradycardia in elderly patients.
• Interactions?
• Renally cleared meds plus acute kidney injury?

**Labs**

• Fingerstick glucose if altered mental status
• Chemistries including Ca & Mg
• Troponin, if MI suggested by history/EKG
• Digoxin level, for patients taking digoxin
• Consider checking TSH, Lyme serology

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**resuscitation**

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Terminology used to describe bradycardia

**Progression towards brady/asystolic arrest**

**Usual terminology:**

Symptomatic Bradycardia

**ARREST**

**Better terminology?**

Stable Symptomatic Bradycardia

Bradycardic Peri-arrest

**ARREST**

Bradycardic peri-arrest responds to a very unstable patient with threatened imminent cardiac arrest. This calls for more aggressive management than a patient with stable symptomatic bradycardia for hours or days.


• Bradycardiac peri-arrest may be loosely defined as severe bradycardia with marked shock and concern for immediate cardiac arrest. The algorithm below shows a maximally aggressive strategy designed to prevent further deterioration into cardiac arrest.
  • There are two "arms" of therapy: electrical & medical.
  • It's hard to predict which patients will respond best to medical or electrical therapy.
  • Proceed simultaneously down both arms of therapy as rapidly as possible until the patient is stabilized.
• For patients with mild signs of organ malperfusion (e.g. normal blood pressure but poor urine output), then a more gradual and stepwise approach may be most appropriate. For example, simply starting an epinephrine infusion will often improve heart rate and perfusion.
problems with atropine

- At low doses, atropine may cause paradoxical bradycardia. Atropine works by poisoning the vagus nerve, so it is only effective for bradycardias mediated by excess vagal tone. It will predictably fail in cases of high-degree AV block.
- Contraindicated in patients who have had cardiac transplantation, in whom atropine may precipitate asystole.
- Atropine may stabilize the patient for 30-60 minutes, but then wear off. This may initially make the patient appear stable, only to deteriorate later on (once everyone has stopped paying so much attention).

strategy when using atropine?

- If atropine is the most immediately available drug, then give it. Alternatively, if you have immediate access to epinephrine, it may be more effective to go straight to epinephrine.
  - Atropine is traditionally the 1st-line medical therapy. However, for very unstable patients, epinephrine is more reliably effective and may be preferable.
  - Start at 1 mg atropine, additional doses can be given to a maximal dose of ~3 mg.
  - Overall only ~25% of patients have a complete response to atropine, so don’t delay other therapies while waiting for atropine to work.
    - Don’t give atropine, sit back, and expect that it will fix everything. Give epinephrine while simultaneously preparing epinephrine and transcutaneous pacing, with the full expectation that the atropine will often fail.

advantages of epinephrine

- Available everywhere, can be obtained quickly.
- Unlike atropine, epinephrine stimulates the entire myocardium. This provides epinephrine with a broader spectrum of efficacy for various mechanisms of bradycardia.
  - Epinephrine is the piperacillin-tazobactam of bradyarrhythmias.
  - Safe for peripheral infusion (no worries about extravasation, you don’t need to place a central line).
**epinephrine strategy**

- Boluses for peri-arrest patient
  - For patient on verge of a cardiac arrest, bolus with doses of ~20-50 mcg epinephrine.
  - Boluses will stabilize the patient for a few minutes, but this is only a temporary bridge to an epinephrine infusion.

- Epinephrine infusion
  - The usual dose is 2-10 mcg/min (but there is no hard upper limit in a crashing patient).
  - Dosing strategy depends on how unstable the patient is. For more unstable patients, start high and down-titrate as the patient responds. For patients who are fairly stable, start low and gradually up-titrate.

- Figure out how to achieve this at your unit:
  - a) If you have immediate access to pre-mixed epinephrine bags, know how to use them (know their concentration and how many ml's are needed to deliver push-dose epinephrine).
  - b) If you don’t have immediate access to pre-mixed epinephrine, then, read on...

**creating & using a “dirty epi drip”**

Mixing a bag of epinephrine is easy. This is conventionally termed a “dirty epi drip,” but if done properly it's a safe and precise way to deliver epinephrine.

**step #1: create the epinephrine reservoir bag**

- Inject 1 mg of epinephrine into a liter bag of normal saline. One milligram of epinephrine can be obtained *either* from an entire syringe of cardiac epinephrine (1:10,000) or an entire vial of IM epinephrine (1:1000).
- Squish around the bag to mix well.
- Label the bag. The most elegant way to do this is using a pre-printed label which includes dosing instructions as shown below. But obviously this isn’t necessary.
step #2: push dose epinephrine

- For a patient in peri-arrest, you will want to deliver small boluses of epinephrine until the patient stabilizes.
- Fill up an empty 20 cc syringe with diluted (1 mcg/ml) epinephrine from your one-liter bag.
- Bolus the patient with 20 ml of this solution, which will deliver a bolus of 20 mcg epinephrine.
- Refill your 20 ml syringe and repeat as needed.
- Push-dose epinephrine is a temporizing solution. As soon as the patient stabilizes, start an epinephrine infusion.

step #3: epinephrine infusion

- Attach your bag of epinephrine to an infusion pump and set the rate. For example:
  - Infuse at 60 ml/hour to achieve 1 mcg/min infusion
  - Infuse at 240 ml/hour to achieve 4 mcg/min infusion
  - Infuse at 600 ml/hour to achieve 10 mcg/min infusion

advantages of the “dirty epi” bolus & drip strategy:

- Relatively idiot-proof. As long as you mix well and label the bag, it should be pretty difficult to make dosing errors:
  - Regardless of what type of epinephrine you use, you will be fine (either 1:1,000 or 1:10,000 will work).
  - It’s physically impossible to bolus a lethal dose of epinephrine after it’s been diluted to 1 mcg/ml (you would need a >100 ml syringe, which doesn’t exist).
  - Even if you run the epinephrine bag in wide open, you would only be delivering about ~30 mcg/min of epinephrine – so again, it’s basically impossible to deliver a lethally high epinephrine dose.
- Encourages a rapid transition from push-dose epinephrine to an epinephrine infusion (which is ultimately a safer and more controlled strategy).
- Viable approach during epinephrine shortages:
  - Easily performed with 1:1000 epinephrine, if your shop runs out of 1:10,000 epinephrine.
  - One vial of epinephrine can be used for both pushes & drip strategy.

**calcium**

Along with epinephrine, calcium is a drug which is often under-utilized in bradycardia. IV calcium is potentially effective for various etiologies listed below. Calcium is pretty safe (unless it extravasates), so when other therapies fail it makes sense to try to some calcium.

**calcium-responsive bradycardias:**

- Hyperkalemia
- Hypocalcemia
- Hypermagnesemia
- Calcium-channel blocker
- Beta-blocker (maybe effective)

**dosing**

- **Bradycardia of unknown etiology**: Try one round of calcium (1 gram calcium chloride or 3 grams calcium gluconate).
- **Known or suspected hyperkalemia**: Start with 1 gram of calcium chloride or 3 grams of calcium gluconate. If ineffective and patient is dangerously unstable, consider additional calcium. The max dose of calcium is unknown in this situation. Bedside chemistry monitoring with an iSTAT might be helpful here,
shooting for moderate hypercalcemia (e.g. ionized calcium level of 2-3 mM).

### Other Medications

**Dobutamine**

- Dobutamine is mostly a beta-agonist, with very weak alpha-adrenergic activity. Unlike epinephrine, dobutamine tends to cause systemic vasodilation:
  - Dobutamine might be perfect for a patient with bradycardia and normal/elevated blood pressure, where you're trying to increase cardiac output (without increasing the blood pressure).
  - Dobutamine isn't a good choice for the crashing, hypotensive patient. If the dobutamine fails to accelerate the heart rate then it could act solely as a vasodilator, and thereby cause worsening hypotension.
  - Dobutamine might not be quite as safe for peripheral infusion as epinephrine. If you're giving dobutamine for prolonged peripheral infusion, monitor the site carefully and avoid any IVs in the hand/wrist.

**Isoproterenol**

- This is an excellent drug for bradycardia if you can get ahold of it.
- Isoproterenol is a pure beta-agonist, which is safe for peripheral infusion. Isoproterenol does seem to be a bit more powerful than epinephrine (there seem to be some patients who don't respond to epinephrine yet will respond to isoproterenol).
- The main drawbacks to isoproterenol are logistic: Isoproterenol is insanely expensive in the United States (an infusion may cost several thousand dollars). Many hospitals don't have it. Even if your hospital does have it, it will usually take time getting it from pharmacy. More on isoproterenol from Pharmacist Scott Dietrich here [http://empharmd.blogspot.com/2016/08/is-it-time-to-ditch-isoproterenol-for.html](http://empharmd.blogspot.com/2016/08/is-it-time-to-ditch-isoproterenol-for.html).

**Dopamine**

- Dopamine has a long track record of use in symptomatic bradycardia. The main advantage of dopamine is that it's stable at room temperature, so it may be more widely available in pre-mixed bags (e.g. in ambulances).
- Disadvantages of dopamine compared to epinephrine:
  1) Dopamine can cause skin necrosis with prolonged infusion.
  2) At high doses, dopamine may act predominantly as a vasoconstrictor. This can be undesirable if you're mostly looking for chronotropy.
- If dopamine is the most readily available agent, then use it. When you have time, consider switching over to an epinephrine infusion.

### Advanced Toxicologic Therapies

- Local Anesthesia Systemic Toxicity (LAST)
  - Suspect in any bradycardic patient on lidocaine infusion or recently treated with nerve block.
  - Front-line therapy is intralipid.
- Beta-blocker and/or calcium-channel blocker toxicity
  - Advanced toxicologic treatments are primarily useful for patients who present with massive overdose. However, these therapies can also be considered for patients with bradycardia due to therapeutic misadventures.
  - Treatment may involve high-dose insulin, glucagon, or intralipid.

### Transcutaneous Pacing

Transcutaneous pacing is often the fastest strategy to increase the heart rate. Even if it doesn't capture, the discomfort may be enough to trigger a sympathetic response that keeps the patient alive. Either way, this is a temporary measure until more definitive stabilization is possible (e.g. transvenous pacing).
Air is a poor conductor of electricity, so placing pads that overlie the lungs is a poor strategy. Anterior-posterior pad placement may be preferred (image above)\textsuperscript{12,13}

- Anterior pad is on the left side of the lower part of the sternum, covering the “left parasternal window” of the heart. Based on experience with echocardiography, this is the most reliable site of contact between the heart and the soft tissue of the chest.

**current**

- If patient is crashing, start at maximal current and work your way down after the patient has stabilized.
- If patient is doing OK, then start low and titrate up.
  - If the patient is doing OK, then you probably wouldn’t really want to do transcutaneous pacing at all. However, it may be useful to determine if the patient responds to transcutaneous pacing. Proving that transcutaneous pacing will capture the heart may help you decide whether placing a transvenous pacemaker is necessary in a borderline patient.
- Continue pacing at 10-20 mA above the minimum energy required for capture.
- Usually ~40-80 mA required to achieve capture (possibly more in obesity or obstructive lung disease).\textsuperscript{14}

**beware of pseudo-pacing**

- Pseudo-pacing is when the pacemaker isn’t capturing the myocardium, but the monitor displays a heart rate equal to the transcutaneous pacemaker. This provides a false sense of security, because the monitor looks great.
- Always **confirm that the pacemaker is capturing** via one of the following methods:
  - Pulse oximetry waveform shows a pulse matching the pacemaker (image above)
  - Bedside echocardiogram confirms myocardial contraction with pacing
  - Pulse, preferably far away from the chest (e.g. femoral pulse or dorsalis pedis, to avoid being fooled by twitching of the chest musculature)

**analgesia/sedation?**

- Can be limited by patient’s instability. Low-dose fentanyl and/or ketamine might be reasonable.
- Deep sedation & intubation to allow for tolerance of transcutaneous pacing is a popular approach, but probably not the best. The instability induced by sedation and intubation may outweigh benefits from transcutaneous pacing. Also, if the patient becomes hyperinflated on the ventilator, this could theoretically lead to loss of capture by the transcutaneous pacer.

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**transvenous pacing**

Transvenous pacing is the most invasive strategy, but also the most effective (with success rates >95%).\textsuperscript{15} Indications are roughly as follows:

- Unstable bradycardia which doesn't respond to other interventions (e.g. epinephrine).
- High-degree AV blocks that leave the patient at ongoing risk of deterioration (e.g. Mobitz II, third-degree heart block with wide-complex escape rhythm).
have a kit, know your kit, love your kit

- The unit should have everything needed for a transvenous pacer in one specific location (e.g. a large box or drawer in a resuscitation cart). This will include the transvenous pacemaker itself, the venous sheath, the pacing generator, wires, and adapter pins.
- Use the appropriate size venous sheath for the pacemaker.
• If you ask for a random venous sheath, you’re likely to be given an 8.5 French sheath which is designed to accommodate a Swan-Ganz catheter. This sheath cannot be used for placement of a pacemaker wire, it will be too large (leading to leakage of blood out of the sheath or air embolization into the sheath). The pacer sheath will be smaller.

• The crux of this procedure is familiarity with the pacer kit stocked in your unit. Ideally the unit should have a non-sterile kit available for practice purposes. In an emergency, the muscle memory for how all the parts get assembled will be invaluable.

• Also know how to work the pacing generator. Newer digital pacing generators are designed for electrophysiologists, so they can be confusing. Make sure you’re familiar with your hospital’s device.

![idiot's guide to Medtronic dual chamber pacer generator](https://i0.wp.com/emcrit.org/wp-content/uploads/2017/01/medtronic2.jpg)

- In general, the sites which allow for most facile floating of the pacemaker are:
  - 1st choice: Right internal jugular (straight shot into the RV)
  - 2nd choice: Left subclavian (smooth arc through the larger vessels into the heart)

**appropriate current while floating the wire**

• Depending on how unstable the patient is, there are roughly two strategies for floating a temporary pacemaker:

  - **Honey Badger Mode.** As you are floating the wire, increase the current to 20 mA. This will capture the heart as rapidly as possible, which is preferable if the patient is actively dying. The problem is that capture may occur while the wire is in the atrium, so this approach doesn’t always result in ideal placement of the temporary pacemaker. The goal here is to stabilize the patient as soon as possible, you can fiddle with the pacemaker later.

  - **Usual technique.** For a patient who isn’t actively dying, float the pacemaker with a lower amplitude (e.g. 5 mA). This usually won’t capture the myocardium until you’re close to the right ventricular myocardium. This strategy is better for optimizing the ideal placement of the pacemaker. After you gain capture, advance the pacer a couple of millimeters further and deflate the balloon – this will often position it optimally, lying against the right ventricle.

**insertion site**

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ultrasound guidance

- Not necessary, but can be helpful. Requires a second operator to reach under the sterile drape and position the ultrasound. Ideally the second operator should be skilled in ultrasonography.
- A four-chamber view (e.g. subcostal 4-chamber) is generally best, this can allow visualization of the wire entering the right atrium & ventricle.
- Potential value:
  1. If you advance the pacer wire over ~30 cm and don’t see it in the right atrium, then the wire has probably passed straight into the inferior vena cava. Deflate the balloon, pull back to ~15 cm, and try floating again.
  2. Ultrasound allows fine-tuning of the placement procedure. For example, once you’re through the tricuspid valve you can slow down – you only have a few more centimeters left to go.
  3. If you visualize the wire in the right ventricle but you’re not getting capture then there might be a problem with the pacer box. Make sure all the wires are connected correctly and the settings are correct.
complications

- Most of the complications relate to placement of the pacemaker sheath in the vein (e.g. pneumothorax or bleeding).
- There is a very small, yet finite risk of hemopericardium (<0.6%).
  - This might possibly be reduced by careful placement of the wire with ultrasound guidance.
  - If a patient deteriorates following placement of a temporary transvenous pacemaker, ultrasonography should be performed to exclude hemopericardium.

**dual pacing as a backup strategy**

Some patients will be encountered who are completely pacemaker-dependent (they have no intrinsic rate at all). This is pretty scary, because if electric pacemaking fails for even a minute the patient will have a cardiac arrest. Temporary transvenous pacemakers do occasionally become dislodged, so this can be a real problem. To prevent the patient from dying if their temporary transvenous pacemaker falls out of place, dual pacing can be used:

- The patient is simultaneously attached to both a transvenous pacemaker and a transcutaneous pacemaker.
- The transvenous pacemaker is adjusted the way it normally would be:
  - Set the rate to something reasonable (e.g. 60-80 b/m, or possibly higher if the patient is otherwise shocky).
  - This should be the pacemaker which is driving the patient's heart rate.
- The transcutaneous pacemaker is attached and turned on, with the following settings:
  - Set the rate 20 b/m below the transvenous pacemaker (e.g. 40 b/m).
  - Set the current to a high enough level to capture the myocardium.
  - Optimize pad placement.
  - This pacemaker should ideally do nothing.

The transcutaneous pacemaker is used here purely as a backup device. If the transvenous pacer malfunctions, the transcutaneous pacemaker will pick up without losing a beat. Of course, this will be painful for the patient (because suddenly they will be getting shocked). However, transcutaneous pacing is preferable to sudden cardiac arrest.
approach to bradycardic peri-arrest

Bradycardic Peri-Arrest

- Transcutaneous Pacing (temporizing solution only)
  - Simultaneous initiation of medical & electrical tx
  - Transvenous Pacing
  - Epinephrine OR atropine
    - Epinephrine push-dose & gtt preferred
    - If atropine is more readily available, give it with delay (full 1 mg).
    - NO atropine s/p heart transplant
  - Epinephrine OR atropine (whichever one you haven’t given yet)
  - Patient is stabilized
    - Evaluate etiology of bradycardia
    - Further treatment depending on cause
  - Calcium
  - Other options: Isoproterenol? Intralipid? Glucagon?

Electrical treatment arm

Medical treatment arm

pacemaker generator cheat sheet:

idiot’s guide to Medtronic dual chamber pacer generator

Panic button:
Try to leave this alone.
If you screw up the settings or can’t figure them out,
initiating this will cause pacer to deliver high-energy asynchronous pacing
(Which is great for a bradycardic emergency)

Atrial connector: DO NOT use this
Ventricular connector: attach temporary pacer wires here

1. DOO/Emergency key
2. On/Off key
3. Pacing and sensing status bar
4. Rate dial
5. A (Atrial) Output dial
6. V (Ventricular) Output dial
7. Lock/Unlock key
8. Enter key
9. Selection indicator
10. Up/Down arrow keys
11. Menu Parameter dial
12. Pause key
13. Lower screen
14. Lock indicator
15. Pacing Mode indicator
16. Battery Indicator
17. V (Ventricular) Output scale
18. A (Atrial) Output scale
19. Rate scale

Power button
Rate dial: adjust −60–120 b/m
Atrial currents set to ZERO
Ventricular currents: adjust 2–20 mA
Unlock required to change settings
Leave this alone.

https://i0.wp.com/emcrit.org/wp-content/uploads/2017/01/medtronic2.jpg
Don’t assume that because the blood pressure is normal, the patient is adequately perfusing and doing fine. Some patients vasoconstrict and maintain normal blood pressure, despite organ malperfusion. For an unstable patient, don’t get fixated on any specific intervention. Continue working through a series of electrical and mechanical therapies until something works (figure below). Don’t be afraid to use push-dose epinephrine and peripheral epinephrine infusions for an unstable patient. Don’t forget to get a good medication history, focusing on recent medication changes and drugs which can accumulate in renal dysfunction (e.g. digoxin, atenolol). Don’t be fooled by transcutaneous pacemaker pseudocapture. The fact that the chest is twitching and the monitor shows a normal heart rate means nothing – it’s still possible that the myocardium isn’t being captured. Remember that bradycardia can be caused by myocardial infarction and various intoxications – so fixing the heart rate may not be enough to fix the patient. Try to imagine every piece of your transvenous pacemaker kit and how it is assembled. If you can’t do this, you need practice with the kit. The most common procedural hang-up is being unfamiliar with the kit and pacemaker generator.

**Going further:**

- Bradycardia
  - **Bradycardia** ([https://lifeinthefastlane.com/resources/bradycardia-ddx](https://lifeinthefastlane.com/resources/bradycardia-ddx))(Chris Nickson, LITFL)
  - **Managing unstable bradycardia** ([https://first10em.com/bradycardia/](https://first10em.com/bradycardia/))(First 10 EM, Justin Morgenstern)
  - **Symptomatic Bradycardia** ([http://www.emdocs.net/em3am-symptomatic-bradycardia/](http://www.emdocs.net/em3am-symptomatic-bradycardia/))(Erica Simon, emDocs)
  - **An approach to bradycardia in the emergency department** ([http://www.emdocs.net/approach-bradycardia-ed](http://www.emdocs.net/approach-bradycardia-ed))(Patrick Ng, emDocs)

- **Dirty Epi Drip & Push Dose Epi**
  - **Push dose pressors** ([https://emcrit.org/podcasts/bolus-dose-pressors](https://emcrit.org/podcasts/bolus-dose-pressors))(EMCrit)
  - **Dirty Epi Drip** ([https://www.aliem.com/2013/06/dirtyepi](https://www.aliem.com/2013/06/dirtyepi))(Zlatan Coralic, ALIEM)

- **Transcutaneous pacing**


9. For bradycardia due to cholinergic poisoning, much higher doses of atropine may be needed. [PubMed](https://www.ncbi.nlm.nih.gov/pubmed/15974204)


12. There doesn't seem to be solid clear data on this. This arrangement is consistent with most references and makes a reasonable amount of sense. Based on echocardiography and anatomy, the location of the left parasternal window is fairly uniform. In contrast, the apex of the heart is highly variable. Therefore, trying to place a pad on the apex of the heart is error-prone. [PubMed](https://www.ncbi.nlm.nih.gov/pubmed/18701603)


The Internet Book of Critical Care is an online textbook written by Josh Farkas (@PulmCrit), an associate professor of Pulmonary and Critical Care Medicine at the University of Vermont.

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https://emcrit.org/ibcc/bradycardia/