BRADYCARDIAS

1. Pulse Bradycardia

   - Assess poor perfusion x (AMR, CP, hyper T), prior Hx?

   - D. Nov: EKG, Peak, Pulse Co, IV access, O2, Night Treadmill Testing
   - Prepare: Drugs, Solution/Analyses, Pacing Machine

   - Confirm poor perfusion x

   - Adequate/Still

      - 2a. Type II or 3a

         - Sino-atrial

         - Escape Rhythm

      - 2b. Underlying Cause

         - Semi-urgent pacing

   - Poor/Marketable

      - 2a. Type II or 3a

         - All other causes

         - Sino-atrial

         - Escape Rhythm

      - 2b. Underlying Cause

         - Semi-urgent pacing

         - Pacing Machine

         - Drugs

         - If fail

         - Cont. Drugs

         - If fail

         - Call cardiologist for transvenous pacing

         - If fail

         - Call cardiologist for transvenous pacing

Turn On, Place Pads, Turn to Pacing Mode

Set Rate to 60 bpm

Set Energy to 2 mA

If electrodes/malposition continue (1)/N

Yes

No

9 Energy by 2 mA — 9 Energy by 10 mA if needed — If capture occur

2a

If NS it’s all right if you hunch up & doing pacing

Dz of SA Node

Drugs

1. ACh

   - Atropine 0.5 mg IV x 1

   - May repeat @ 1-5 min up to total 3 mg or 0.004 mg/kg

Epinephrine

   - 2-10 μg/ min

Epinephrine

   - 2-10 μg/kg/ min
a. Sinus Bradycardia
   i. normal in a cardiovascularly conditioned person
   ii. metabolic: ischemia, sepsis, myxedema, hypothyroidism, hypoTH, infiltrative dz (sarcoid, amyloid), collagen vascular disease, endocarditis, hyper/okalemia, OSA
   iii. meds: excess B8 (remember eye drops), CCB, amiodarone, Li, digoxin, clonidine
   iv. increased vagal tone 2/2 increased ICP, infection, coughing, micturition, defecation, vomiting, etc

b. Sick Sinus Syndrome
   i. Can be (1) Sinus Arrest/Pause when the node just fails to produce a pulse and an escape rhythm kicks in, (2) Chronotropin Incompetence when the node is unable to increase HR appropriately in response to metabolic need, etc (3) Tachy-Brady Syndrome when the pt alternates from brady to tachy (esp AFlb) therefore always ask brady pt if they had tachy Sx and vice versa
   ii. 2/2 idiopathic degeneration aka aging
   iii. Tx w/ pacemaker if symptomatic or tachy-brady syndrome and you are starting a beta blocker

(2) Dz of AV Node
a. Ranges from asymptomatic (1° and 2° Type I) to CHF because of complete heart block and malignant arrhythmias (2° Type II and 3°)
b. depending on where the block is in relation to the AV node (supra-His vs infra-His) QRS complex could be narrow or wide
c. Treatment depends on the likelihood of developing symptoms
d. Always write “NSR w/ 1/2/3 AV Block”
e. 1° Block
   i. PR >200msec = >1 big block
   ii. symptoms might occur when PR >300msec such that pacemaker may be indicated otherwise you do not treat 1° Block
f. 2° Block (note it if it is 2:1, 3:1, 4:1, etc)
   i. Mobitz Type I (Wenckebach) ______ X
      1. PR progressively increases with each beat and then drops
      2. block is usually supra-His thus narrow QRS such that escape rate is going to be >40
      3. rarely symptoms occur such that pacemaker may be indicated otherwise you do not treat 2° Type II Block
      4. often paroxysmal
   ii. Mobitz Type II ______ X
      1. PR stays constant (and normal i.e. <200msec) but after a few beats it suddenly drops
      2. block is usually infra-His thus wide QRS such that escape rate is going to be <40 b/c ventricles will be beating on their own
      3. rarely symptoms occur but regardless this rhythm can easily progress to complete heart block therefore pacemaker is always necessary even in the absence of symptoms
   iii. NB 2:1 2° Block can be Type I or II (impossible to differentiate)

(3) Escape Rhythm (in sinoatrial block there is recurrent block of sinus for more than one beat which sometimes evokes several escape beats after which SA node eventually takes over OR there is just a long pause with no escape beats after which SA node takes over again)
   i. Atrial Escape Rhythm (60-80bp, different looking P wave, nl QRS)
   ii. AV Junctional Escape Rhythm (40-60bp, no P wave or inverted P wave before/hidden/after QRS b/c of retrograde depolarization, nl QRS)
   iii. Idioventricular Escape Rhythm (20-40bp, no P wave, wide QRS)

TACHYCARDIAS
• Causes of tachycardia
  o (1) Increased Automaticity of Ectopic Rhythms
  o (2) Re-Entry
Monomorphic / Regular RR Interval

- **Paroxysmal Supraventricular Tachycardias (PSVTs)**
  - abrupt onset/termination hence called “paroxysmal” due to the random emergence of PACs
  - all PSVTs occur because there is an accessory pathways that conducts impulses at a different speed than the normal pathway, this difference in conduction velocities allows an impulse traveling down one pathway to travel up the other pathway creating an abrupt, rapid, self-sustaining re-entrant tachycardia
  - AV Nodal Reentry Tachycardia (AVNRT) 70%
    - Mechanism: instead of there being one pathway in the AV node (normal) in AVNRT there are two pathways: (1) fast conduction / long refractory period and (2) slow conduction / short refractory period, in pts with AVNRT most of the time impulses are transmitted down (antegrade) the fast pathway and to ventricles only acting like a normal AV node therefore nl EKG
      - Typical 95%: but when a PAC is generated it tries to travel down (antegrade) the fast pathway but it can’t because the fast pathway is still refractory and thus PAC can only travel down (antegrade) the slow pathway but instead of conducting down to ventricle only it also conducts up (retrograde) fast pathway (b/c now enough time has passed that the fast pathway is no longer refractory) and into atrium causing tachycardia
      - Atypical 5%: sometimes when impulse are transmitted down (antegrade) the fast pathway they not only conduct down to ventricles but also up (retrograde) thru slow pathway to atrium causing tachycardia
    - EKG
      - Typical: tachycardia (~160-180bpm), P wave is buried within QRS so you can’t see it (no RP) or is inverted and right after QRS (short RP)
      - Atypical: tachycardia (~160-180bpm), P wave is inverted and right before QRS (long RP)
  - Epidemiology (CAN OCCUR IN ANYONE!!!)
    - can develop at any age but most common at ~40yo, 70% are women
    - most often occur in pts with NO structural heart disease
  - Treatment
    - When the arrhythmia occurs follow ACLS guidelines
    - If pt has frequent episodes teach them to use ACLS guidelines (vagal maneuvers and drugs)
    - b/c medical therapy is only 40% successful and has a high recurrence rate pts can be offered radiofrequency catheter ablation which is ~90% successful and has a low recurrence rate but has 1% incidence of complete heart block complication
  - AV Reentry Tachycardia (AVRT) 30%
    - Mechanism: instead of there being one pathway from atrium to ventricle via AV node there is an accessory pathway (Kent Bundle) that connects the two chamber bypassing the AV node (similar to the fast and slow pathways in the AV node of AVNRT), either pathway can conduct antegrade and/or retrograde therefore look at the two pathways as a race track
    - NB Wolf Parkinson White is a disease where you have an accessory pathway outside of the AV node, it leads to two different rhythm abnormalities: (1) AVRT (obviously!!!) and (2) Afib (not quite sure but pts w/ WPW have increased risk of Afib)
      - Non-Pre-excited
        - During normal rate the accessory pathway conducts up (retrograde) such that during normal sinus rhythm nothing is seen on EKG
        - Orthodromic AV Reentry Tachycardia: occurs when the impulse is traveling down (antegrade) AV node and up (retrograde) accessory pathway creating a loop, EKG: Tachycardia, Inverted P after QRS, No Delta Wave, Narrow QRS
      - Pre-Excited
        - During normal rate: the accessory pathway conducts down (antegrade) such that during normal sinus rhythm there is premature depolarization of part of ventricle before normal depolarization resulting in a delta wave
        - Antidromic AV Reentry Tachycardia: occurs when the impulse is traveling down (antegrade) accessory pathway and up (retrograde) AV node creating a loop, EKG: Tachycardia, Inverted P after QRS, No Delta Wave, True Wide QRS (looks kind of like Vtach)
        - Wolff Parkinson White (WPW) Syndrome: when during normal sinus rhythm the pt is also tachycardic, EKG: tachycardia, (1) short PR (<120msec), (2) delta wave, (3) widened but not true wide QRS (>120msec) (NB Important b/c in sinus rhythm there is a recognizable pattern hence commonly tested), when the tachycardia develops into atrial fibrillation the combination of pre-excitation and atrial fibrillation is devastating b/c the accessory pathway cannot slow conduction to the ventricle like the AV node can thus the ventricle can reach rates up to 300bpm (EKG: irregular wide complex tachycardia) causing sudden death (“pre-excitation atrial fibrillation”) therefore do not give AV blocking agents typically used in treating Afib b/c promotes conduction down accessory pathway instead treat w/ IV procainamide
• **Treatment**
  
  • **NON-Pre-Excited:** when the tachycardia occurs follow ACLS guidelines and use AV nodal blocking agents (adenosine and Class III and IV anti-arrhythmics) to slow down the loop, Long Term Therapy: same drugs
  
  • **Pre-Excited:** when the tachycardia occurs follow ACLS guidelines and use Pathway blocking agents (Class I and III anti-arrhythmics) to slow down the loop, NB do not use AV nodal blocking agents b/c they promote conduction down accessory pathway, Long Term Therapy: same drugs except if pt has WPW then do radiofrequency catheter ablation
  
  o NB there are many other PSVTs

- **Regular Sinus Tachycardia**
  
  o 100-130 bpm (upper limit of nl is 220-age)
  
  o Key feature is that it is gradual in onset
  
  o usually well tolerated (cardiac filling is not compromised until HR is >180bpm) and thus does not require primary treatment rather just manage the underlying illness (unless it occurs in the presence of myocardial ischemia then slow it down with a BB)
  
  o **Mechanism**
    
    ▪ Normal: pt is scared, in pain, exercising, anxious
    
    ▪ Abnormal: hyperTH, hypovolemic, infection/fever, anemia/hypoxic, pulmonary embolism, adrenergic meds

- **Atrial Tachycardia**
  
  o **Mechanism**
    
    ▪ Underlying cardiac disease that increases the excitability of atrial tissue esp at crista terminalis, atrial septum, atrial appendages, tricuspid annulus, pulmonary veins
    
    ▪ **Digitalis Toxicity (AT + Z** AV Block)**
Polymorphic / Irregular RR Interval

- Premature Atrial Complexes (PACs)
- Irregular Atrial Tachycardia (many atrial foci)
  - Wandering Pacemaker
  - Multifocal Atrial Tachycardia (MAT)
    - A handful of foci in atrium such that the P wave that is generated has only about a handful of different shapes and the PR interval has only three different lengths but unlike atrial flutter there are isoelectric periods between P waves
    - RFs: >70yo, pulmonary disease (probably due to a theophylline side effect), hypomg, hypok, CAD (in general pts are sick and in ICU)
    - Tx:
      - d/c theophylline if present
      - replete Mg even if normal and then replete K only if low
  - Atrial Flutter w/ Variable Block
    - A: 250-350 bpm ("saw-tooth pattern") V: variable beat (although usually conducts 2:1 many times it changes from 2:1 to 4:1 to 3:1 to 2:1, etc) usually atrial rate of 300 (saw tooth, best seen in inferior leads and V1) and vent rate of 150 b/c of 2:1 block
    - Never "honed" hence always associated w/ some systemic disease
    - similar to Afib in causes, Tx, even AC, etc except Aflutter requires less cardioversion energy (10-50 Joules) but higher antiarrhythmic drug concentration (mg/dl) and ablation is especially successful if done b/c Aflutter is almost always caused by a single macro-re-entrant circuit around the tricuspid valve, therefore ablation b/t ICV and TV is first line Tx

Atrial Fibrillation (AFib)

- A: 350-500 bpm V: unlike atrial flutter which is regular in afib ventricular response is truly variable anywhere between 75-175 bpm (if tachy QRS then called Afib w/ RVR = Rapid Ventricular Rate which is bad) EKG: quivering appearance b/c of many irritable foci
- Epidemiology: 1% of population making it the most common arrhythmia (8% of elderly)
- Classification (always do this):
  - New Onset and thus Acute vs Recurrent and thus Chronic and if so then determine if...
    - Paroxysmal: self-terminates
    - Persistent: does NOT self terminate
    - Permanent: does NOT terminate despite attempted cardioversion
- Why is AFib bad? (Afib alone does not increase mortality rather it’s the associations noted below that cause problems)
  - (1) Potential for Reduced Cardiac Output (contraction of atria is responsible for 1/4 of ventricular end diastolic volume (preload) and in afib this is lost (there is no atrial "kick"), in a normal heart this is of little consequence but in pts with impaired diastolic dysfunction afib can be very bad especially if there is a rapid ventricular response (RVR) b/c ventricular filling is additionally reduced)
  - (2) Potential for Thrombus Formation and Embolization
  - (3) chronic ventricular tachycardia can cause tachy induced dilated-CM that is why chronic rate control is important
- Pathophysiology
  - Originates from ectopic foci in atrial "sleeves" in pulmonary veins
  - NB if pt is >40yo always screen for various types of heart disease
  - Acute Causes (50% unknown)
    - Heart Dz: Ischemia, Myocarditis/Pericarditis, Valve Dz, CHF, HTN, CM, Congenital Heart disease, Atrial Myxoma
    - Pulmonary Dz: Pneumonia, PE, lung cancer, chronic lung disease
    - Systemic Dz: hyperTH, sepsis/infection/fever, stress/anxiety
    - Meds: excess ETOH and then withdrawal ("Holiday Heart"), Cocaine, Theophylline
    - Familial (10%!!!, mutation at 1p36-p35 resulting in increased but defective ANP)
- Post-Op (35%/60% of pts undergoing CABG/valve surgery) occurs during first 4d and self-limited with most converting to sinus in 6-8wks
- Lone Afib: pt is <60yo and has no predisposing conditions and no heart dz in echo (15% of afib)

  Three Fundamental Strategies for Management
  - If Acute
  - (1) Rate Control
    - Goal is to reduce ventricular rate to b/t 60-80bpm and if art line is in place monitoring SBP can be a more physiologic end-point for rate control b/c SBP reflects SV and SV reflects VEDV and EDV reflects HR

  - IV AV Blocking Agents
    - BB (especially if pt is in a hyperadrenergic state such as acute MI and post-cardiac surgery, if hypotensive then treat w/ glucagon) metoprolol/esmolol
    - CCB (especially if pt has obstructive lung dz, if hypotensive then treat w/ CaGluconate) diltiazem/verapamil
    - Digoxin (esp if pt has CHF or LV Dysfunction or is already hypotensive, not good at all for acute Afib but if can be helpful as an adjunct)
    - NB try not to give BB and CCB at same time b/c both act at AV node
    - NB some give amio as a rate controlling agent (150mg over 10min then 0.5-1 mg/min)

  - Chronic
    - PO AV Blocking Agents (same ones as above)
    - Invasive Procedure (if failure to above)
      - Catheter AV Node Ablation w/ Permanent PPM Implantation (NB pt is still in Afib but no longer in RVR)

  - (2) Anticoagulation
    - General Risk: 1%/yr vs Risk in Pt w/ Afib: >2%/yr
• paroxysmal vs persistent vs permanent is the same in terms of stroke risk therefore it doesn’t matter which kind they have
• "CHADS2" System in structurally normal AF (if pt has structural disease like valve dz, hypertrophy, low EF<35%, atrial thrombus, etc then Coumadin)
  o C = CHF (1 point)
  o H = HTN (1 point)
  o A = Age >75 (1 point)
  o D = Diabetes (1 point)
  o S = Stroke (2 points)
  o 0 (Lone Afib) = 1.9% yearly stroke rate = ASA 81-325mg PO QD
  o 1 = 2.8% yearly stroke rate = ASA 81-325 or Coumadin INR 2-3
  o 2/3/4/5/6= 4.0/5.9/8.5/12.5/18.2% yearly stroke rate = Coumadin INR 2-3
  
• (3) Rhythm Control
  o NB remember when you cardiovert you must rule thrombus
  o Acute
    • DC Electrical Cardioversion
      • More effective but requires anesthesia and pt to be fasting
      • Should be synchronized
      • Monophasic: 200J for Afib and 50J for Aflutter (increase by 100J to max of 400J) vs Biphasic (half the amount of joules)
    • Pharmacologic Cardioversion
      • Less effective but no need for anesthesia and pt to be fasting
      • For first episodes <48hrs that are distressing (NB 50% of recent-onset Afib converts spontaneously) and stable with no evidence of cardiac ischemia
        • <7/8: 1° dofetilide/lecainide/ibutilide/propafenone 2° amio
        • >7/8: 1° dofetilide 2° amio/ibutilide
  • Chronic Maintenance of NSR
    o NB AFFIRM trial showed that converting to NSR has not been shown to reduce mortality and morbidity and anti-arrhythmic agents have their own risk for life-threatening proarrhythmias therefore maintenance of NSR should only be reserved for symptomatic pts who are already rate controlled and anticoagulated, also most pts convert on their own
    • DC Electrical Cardioversion
    • Pharmacologic Cardioversion
      • Exact Anti-Arrhythmic depends on exact comorbidity
        • First Check EF and if low then Amio but if normal then check CAD and if + then Sotalol but if – then check LVH and if + then Sotalol but if – then you can use any antiarrhythmic including the above and also the Ic
      • If pt is symptomatic and has paroxysmal Afib try pill in pocket anti-arrhythmic above but if pt has persistent or permanent then consider scheduled anti-arrhythmic or procedure below
        • Percutaneous Catheter Circumferential Pulmonary Vein Radiofrequency Ablation
          • Used in young healthy pts w/ symptomatic lone paroxysmal Afib that have high Hz of Sx, failed prior anti-arrhythmic meds, have nl heart anatomy, and no lung dz
          • b/c it has been found that ectopic beats originate from the sleeves of muscles within pulmonary veins or in the LA appendage
          • 80% success rate though repeat procedures are often necessary
          • Risk of perforation, pulmonary vein stenosis, atrial esophageal fistula formation, etc
        • Surgical MAZE Procedure w/ LA Appendage Obliteration
          • Multiple incisions are made into the R/LA to create narrow corridors of atrial tissue which have been shown to not support atrial fibrillary waves
          • 90% success rate
          • Consider if pt is undergoing cardiac surgery for other reasons or if pt has persistent Afib
Wide Complex aka of Ventricular Origin Tachycardias

Monomorphic aka Regular RR Interval

- **Regular SVT w/ Aberrancy** (refer above)
  - Aberrancy (some other pathology exists which widens QRS making it look like a wide complex tachycardia when in fact it is just SVT + something else... usually BBB)
- **Regular VT** (100-250 bpm) NB Ventricular Flutter w/ Regular Block (250-350 bpm) is never really used
  - Anatomic Division
    - Structurally Abnormal Heart: examples include scar from prior MI, CMP?, arrhythmogenic RV dysplasia, etc
    - Structurally Normal Heart: examples include RVOT VT, etc
  - Rhythmic Division
    - Non-Sustained (lasting <30sec)
      - Asymptomatic and No CAD or LV Dysfunction: nothing
      - Asymptomatic and CAD or LV Dysfunction: consider ICD
      - Symptomatic: beta-blocker → amiodarone (w/o structural heart disease) or Class I antiarrhythmics (w/ structural heart disease)
    - Sustained (lasting >30sec)
      - Pts are usually symptomatic and can easily progress to Vfib thus they usually require treatment: ICD with Class III antiarrhythmics or beta-blockers to prevent repetitive shock, Radiofrequency Ablation if isolated VT focus or if recurrent VT triggering ICD firing
      - AICD Indications

  - Primary Prevention
    - Any CM pt w/ EF<35% >9mo despite optimal medical Tx or Ischemic CM pt w/ EF<35% >1.5mo despite optimal medical Tx (MADIT, MUSTT, MADIT II, COMPANION, DEFINITE, SCD-HeFT Trials)
    - H-CM w/ FHx of sudden death, syncope, NS-VT, hypoTN after exercise, massive hypertrophy (>3cm)
    - Congenital Long QT Syndrome
    - Channelopathies like Brugada Syndrome
    - Arrhythmogenic RV CM

  - Secondary Prevention
    - Survivor of Sudden Death if not 2/2 a reversible cause like hyperK (CIDS, AVID, CASH Trial)
    - Some pts have hemodynamically stable Idiopathic VT can be Tx w/ various forms of radiofrequency catheter ablation

- NB How do you distinguish SVT w/ Aberrancy from VT?
  - NB assume WCT is VT until proven otherwise (b/c VT deadly and the pharmacologic agents used in SVT can severe hemodynamic instability if used erroneously in VT), thus SVT is a DOE (NB hemodynamics and rate are NEVER predictive)
  - (1) NB WCT of wide complex tachycardias in pts with heart disease are VT therefore if pt has heart disease assume VT
  - (2) EKG: features suggesting VT (Brugada Criteria)

<table>
<thead>
<tr>
<th>VT</th>
<th>SVT w/ Aberrancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete AV Dissociation</td>
<td>AV Dissociation</td>
</tr>
<tr>
<td>There is no fixed relationship between atria (P waves) and ventricles (QRS waves) such that as the SA node continues to pace sometimes the depolarization conducts to the ventricles creating a normal QRS (Capture Beats) or a blend between normal QRS and wide QRS (Fusion Beats)</td>
<td></td>
</tr>
<tr>
<td>Concordance (QRS in all precordial leads have the same pattern and direction aka all the R-waves V1-6 point up or down)</td>
<td></td>
</tr>
<tr>
<td>Before Arrhythmia</td>
<td>PVCs</td>
</tr>
<tr>
<td>Rate</td>
<td>200bpm</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Irregular w/ Beat-to-Beat</td>
</tr>
<tr>
<td>variability of QRS morphology</td>
<td>QRS Width</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-----------</td>
</tr>
<tr>
<td>Response to AV Blockade (Vagal Maneuvers, Adenosine, etc.)</td>
<td>None</td>
</tr>
</tbody>
</table>

- NB you also need to rule out ventricular paced pacemaker induced tachycardia (check for PM on PEx, CXR, or pacing spikes on ECG)
- NB you also need to rule out antidromic AVRT

Polymorphic aka Irregular RR Interval

- Irregular SVT w/ Averbancy (refer above)
- Pre-Excited Atrial Fibrillation (AF+WPP) (refer above)
- Premature Ventricular Complexes (PVCs) (one irritable ventricular focus)
  - Singlet (one) NNNNNNNN vs Couple (two) NNNNNNNNN (NB never triplet b/c then considered VT/F)
  - Bigeminy (every other nl beat) NNXNNXNN vs Trigeminy (every other two nl beats) NNXNNXNN
  - These beats are usually followed by a compensatory pause
  - In healthy pts PVCs are of no concern, in pts with structural heart disease PVCs have been shown to increase M&M but no therapy currently exists which has been shown to change outcome (these findings are more significant if the PVCs occur during exercising or stress testing)
  - If PVCs are severe and disabling then give beta-blocker

- Irregular Ventricular Tachycardia (many irritable ventricular foci) (100-250 bpm) NB Ventricular Flutter w/ Variable Block (250-350 bpm) is never really used
  - Irregular/Long QT w/ U wave aka Torsades de Pointes ("twisting around the points") Irregular Ventricular Tachycardia w/ QRS that change like a twisted ribbon + Prolonged QT
    - If acquired then MgSO4 2g iv over 10min (not hours!!!) and repeat x1 in 10min if needed then follow with a continuous infusion of 1g/hr for 6hrs, correct electrolyte abnormalities, and d/c any potentially causative drugs
    - If congenital then ventricular pac or use isoproterenol to raise HR to >100bpm so that the QT interval is shortened reducing tendency for VT
  - Regular/Normal-Length QT aka Polymorphic VT

- Ventricular Fibrillation (Vfib) (many irritable ventricular foci that are suffering from entrance block) (>350 bpm)
  - Never has a pulse, hypotensive, no appreciable heart sounds, quickly leads to asystole
Staying Alive by the Begees = 100beats/min