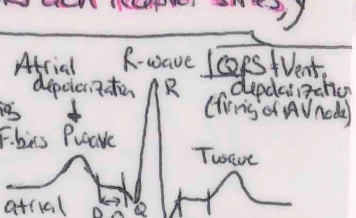
Cardiology

Sinus Bradycardia - Not arrythmia, P-R interval is not elongated (.12-.20 sec)
Slow heart rate → low perfusion
Causes: Excessive vagal tone, carotid sinus pressure, opiates, benzos, Narcan, acute MI, etc...
Tx: EKG, establish IV access, Administer IV Atropine, if refractory initiate Transcutaneous pacing
(Atropine moa - non-selective muscarinic acetylcholinergic antagonist ↑firing of SA node and conduction through AV node, opposes actions of vagus nerve, blocks ach receptor sites, and ↓ bronchial secretions)

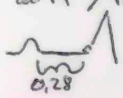
AV nodal Block (4 types)

SSx: Dizziness
conduction disorder b/n SA → AV
(Atrial depolarization)
Atrial Contraction (P-wave)
QRS → Ventricular Contraction
} ↑ distance b/n P-R interval.



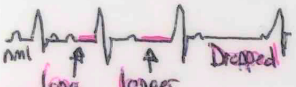


1st Degree only finding is elongated P-R interval.
nml PR interval = 0.20 sec
∴ if > than 1 big box = elongated



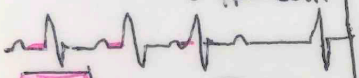
No Tx

2nd Degree - Mobitz Type I
The PR interval becomes more and more elongated.



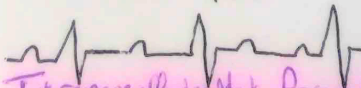
Tx: None (Step I), or Atropine if symptomatic

2nd Degree - Mobitz Type II
Dropped QRS but not prolonged PR
nml PR interval and dropped Beat.



Tx: Internal Pacing

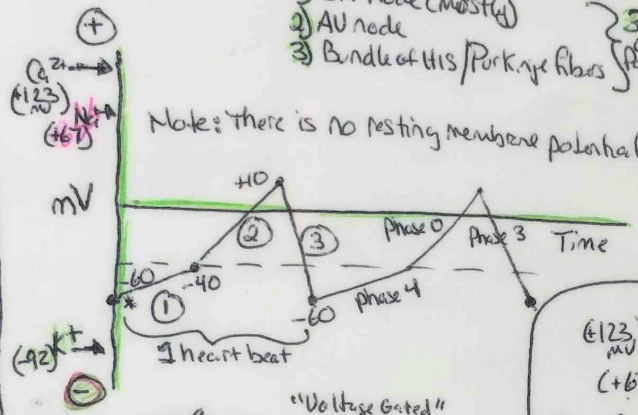
3rd Degree Heart Block
No Correlation b/n P-wave and QRS complex

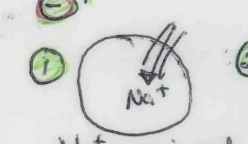


Tx: remove offending Meds, Pacemaker (implantable cardioverter defibrillator)

Pacemaker Cells

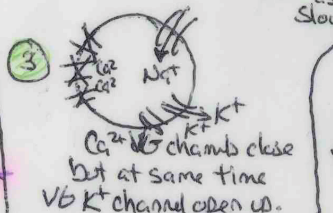
"Automaticity" meaning we don't need a neighboring cell to help activate
1) SA node (mostly)
2) AV node
3) Bundle of His / Purkinje fibers
} 3 locations of Pacemaker Cells.



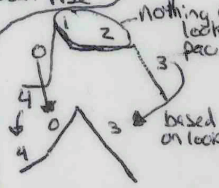


Na+ comes in and brings up to -40 mV. Then a new channel opens

at -40 mV Ca2+ VG channels open. Ca2+ flows in. (Ca2+ isn't as quick as Na+. Slower rise



Ca2+ VG channels close but at same time VG K+ channel open up.
∴ line drops to -60 mV then K+ channels close.
∴ back to step 1



phase 0 in heart myocyte is faster as opposed to pacemaker phase 0 which is slower.

"less negative" = Depolarization
"more negative" = repolarization

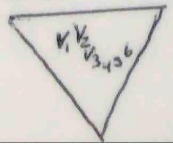
Bundle Branch Blocks

RBBB or LBBB (rarely symptomatic)
Look at Precordial Leads



V1, V2, V3, V4 RBBB

V3-V6 LBBB



Block of Bundle of HIS

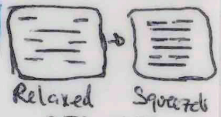
(Interferes w/ Ventricular Contraction)
- Most MI's occur in Ventricular Tissue
? previous MI
ITS a QRS complex issue

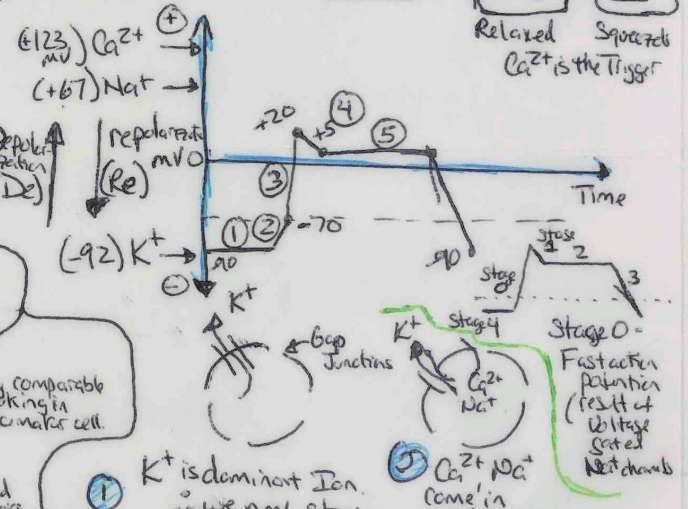
No Tx (Inf. wall MI = RCA (post descending br.) II, III, aVF)

Ant. wall infarct V1-V4
anteroseptal infarct V1-V2
anterolateral V4-V6 (Left circumflex)

Na+-K+ pump (ATPase)
(3 Na+ out, 2 K+ in)

Cardiac Myocytes

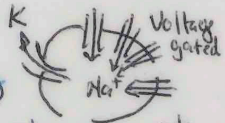




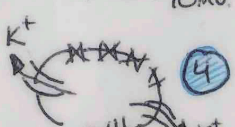
1) K+ is dominant Ion. ∴ we nml stay around -90 mV

5) Ca2+, Na+ come in a bit bring up to -70 mV

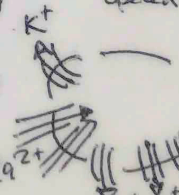
Stage 0 - Fast action potential (result of voltage gated Na+ channels



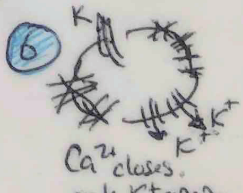
3) when moving up to -70 then a bunch of Sodium channels open up. Na+ rushes in. The voltage gated Na+ channels will close as quickly as they opened.

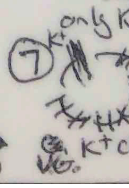


4) K+ always leaks out but now voltage gated K+ open bc time Na+ closed.



5) Voltage gated Ca2+ open since K+ leaves and Ca2+ leaves ∴ we set (straight)





Arrythmias (make assumption that we have a pulse, Pulseless if different)

Brady
Tachy
Flutters, Fibrillations
Premature Contracture and Re-excitation

Sinus Tachycardia
QRS after p-wave.
Tx: underlying cause
narrow QRS (< 0.12 sec) wide QRS. No pharm necessary.

Paroxysmal Supraventricular Tachycardia (PSVT)
Intermittent Tachyarrythmia that originated above the AV node.
- can see electrical alterans

Significant Tachycardia, QRS w/o any P-waves (no full Atrial depolarization)
- Can be triggered by stress, caffeine, alcohol, stimulants

Tx: get cardiac enzymes
Stable → start w/ Vagal maneuvers
unstable or non-responsive to Vagal: IV adenosine or IV CCB (Verapamil, Diltiazem)
If chronic → Beta-blocker, CCB, Digoxin

Ventricular Tachycardia
can degenerate to V-fib. MEDICAL EMERGENCY
Very wide and bizarre QRS complexes w/ no distinctly discernable P or T waves (b/c they are hidden)
Ventricles aren't filling
SSx: Syncope, chest pain, Palpitations
Rapid Ventricular depolarization w/o atrial depolarization = drastic ↓CO = Cardiac Shock
self-stim. w/ ventricles
wide QRS

Tx: IV amiodarone (Lidocaine and procainamide can be used if refractory)
Stable
unstable → Immediate Cardioversion w/ or w/o Sedation.

Tachycardia
HR > 100
SSx: Palpitation
lots of causes: Fever, Pain, panic, dehydration, cocaine, hyperthyroid, pheochromocytoma.

Stable vs. non-stable
non-stable → sedate and cardiovert (Benzos) (Synchronized cardiovert)

SVT
"any arrythmia that causes electrical activity to go to AV node and not from SA node. (a competing input not from SA node)
Includes: Multi-focal Atrial Tachycardia, A.fib w/ rapid ventricular response, WPW, PSVT

WPW
Wolf-Parkinson-White
(From Ventricle Back to Atria)
Accessory conduction site b/n Atria and Ventricle
- Multiple sites possible

CONGENITAL
Delta Wave

Tx: acute Symptomatic: Vagal Maneuvers, IV adenosine, also IV procainamide (adenosine will linger) long Term ablation.
atria → ventricle → atria

Multi-Atrial Tachycardia (MAT)
multiple Pacemaker sites. Unlike other SVTs there will be P-waves present here. Tachycardia at least 3 distinct Morphologies of P-waves with QRS complex. Cause is generally COPD, other respiratory D.
SSx: palpitations, dizziness SOB.
Dx: on EKG
Tx: Oxygen by face mask.

* "all p-waves look different"

Torsade De Pointes (TdP)
Specific Type of V-tach.
SSx: Palpitation, lightheaded
V-tach of oscillatory amplitude.
causes: hypomagnesia, congenital QT syndrome, Drugs
may require cardioversion.
Acute Stable = IV Magnesium
Acute unstable = Cardioversion
long Term = Beta-Blocker, Implantable Cardiac defibrillator.
(Don't use Beta Blocker in pts with congenital long QT syndrome)

Drugs: Methadone, Lithium, TCA, antipsychotics, Macrolids, Zofran.

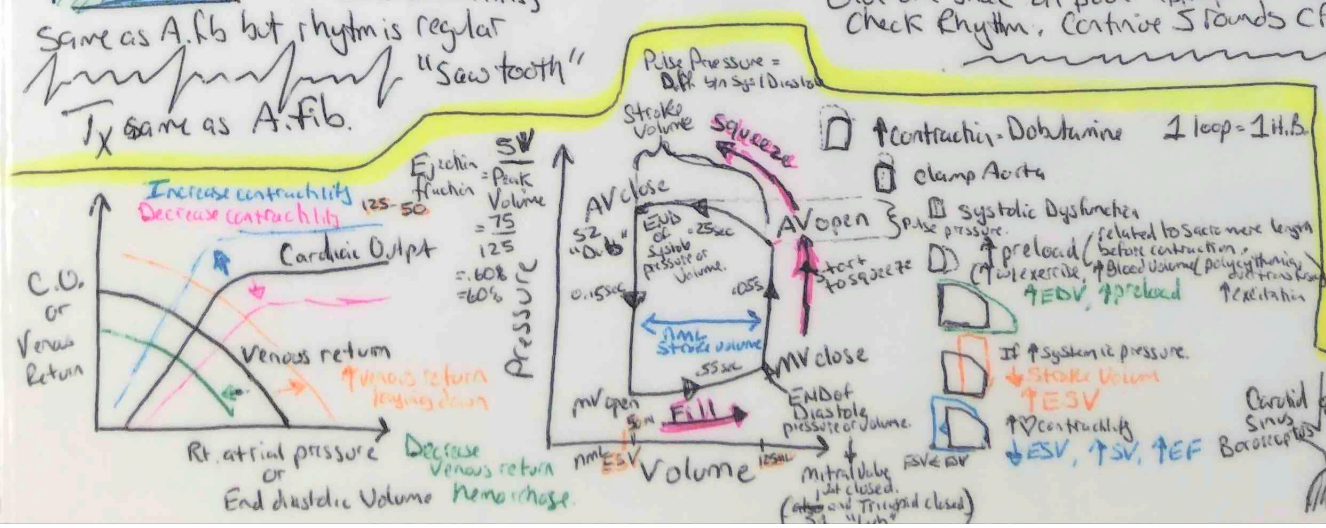
A.fib.
CHADS
C = CHF
H = HTN
A = Age > 75
D = DM
S = Stroke (2 pts)
0-1 = ASA
2+ = Warfarin

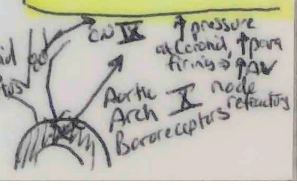
In ER = Hypotension w/ hemodynamic instability
Tx: immediate progressive cardioversion (start at 100J)
If A.fib and stable w/ rapid ventricular Response. use CCB (↑ refractory period of AV node) - BEST
Beta blocker, Digoxin
LT: Anticoagulate - warfarin (INR 2-3)

A.flutter
caused by atrial reentrant circuit (self stimulating)
Same as A.fib but rhythm is regular
"Saw tooth"
Tx same as A.fib.

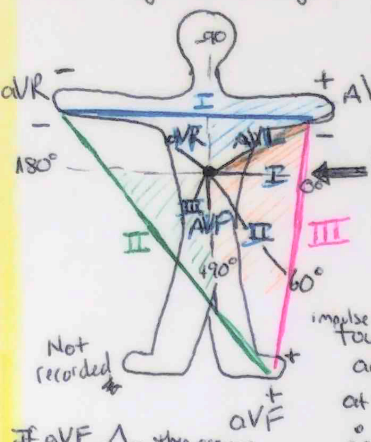
Flutters / Fibrillations

Ventricular Fibrillation | Medical Emergency
100 compressions/min
always hemodynamically unstable.
* EKG will show erratic rhythm w/ no discernable features w/ fib / V.Tach
Tx: IV access, begin CPR, prepare defibrillator.
Give one shock at power appropriate for device.
Check Rhythm. Continue 5 rounds CPR
CPR, O_2
Defibrillate
IV access
resume CPR
Epinephrine 1mg q3-5 min (or Vasopressin 40 units 1st or 2nd dose of Epi)
No Asystole? ← shock → Yes CPR
Amiodarone 300mg IV push
Non-shockable Asystole? Yes shock





"aVR" = augmented VoltageRt. I, II, III = Bipolar (takes info. from 2 Leads)



Lead I = combined info from aVL, aVR
looks at Heart from Top.
Lead II = aVR, aVF
Lead III = aVL, aVF

looks at ♡ from the following vectors.
II travels in best general direction

Nml axis +90° - -30°
Lt. Axis dev -30 - -90°
Rt. axis dev +90 - +180°

impulse traveling towards lead = ↑
away from lead = ↓
at 90° to lead = equal
∴ aVL should be 90° to II

If aVF ↑, then assume Rt. axis deviation abnl.
So look at ~~QRS~~ and ask is it ↑ equiphasic (ie. lead I)

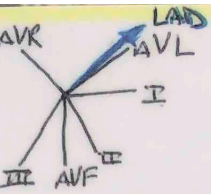
↑ equiphasic (then axis is close to 90°)
↑ more positive (then shift axis ≈ 15° towards equiphasic lead)
↑ more negative (then shift axis ≈ 15° away from equiphasic lead to find true axis)

Note: we are trying to find true axis not correct it.

Most equiphasic lead is?
Lead I ↕
So the true axis must be ~90° to lead I.
Traveling
If aVF is mostly ⊕, then the true axis is most +90. (Near Rt axis dev)

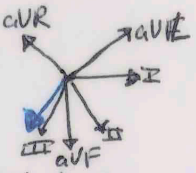
PR = <.2 sec (or one big box)
QRS = <.12 (or 3 small boxes)
QT = < ½ the RR interval

LAD - I ↕, II ↑, III ↓
left axis Deviation



MCC: Left anterior fascicular Block

RAD - I ↓, II ↑, III ↑



RCC Lt. posterior fascicular block

Inf. leads → Right Coronary Artery
II, III, aVF

lateral leads → Circumflex artery
I, aVL, V5, V6

Anteroseptal leads → left ant. descending
V2, V3
V3, V4 - anterior
V5, V6 = left lateral Ventricle
V1 V2 = Septal

Ischemia (low Oxygen) - ST depression or T wave inversion (except T wave inversions are OK in leads V1 and aVR)

old infarct/dead myocardium = pathologic Qwaves. (basically the Qwave of the QRS complex should never be bigger than one tiny box)

Pleuritic Pain (changes w/ respiration)

- Pulmonary embolism = sharp, stabbing chest pain, worse w/ deep breath (pleuritic pain)
- Pneumonia = Fever
- Pleuritis - Pleuritic pain is also pulmonary infarct, pulmonary TB or pulmonary tumor.
- Pericarditis - Pain better when sitting up and leaning fwd. Started after viral Inf., Fever, cardiac hx
- Pneumothorax - unilateral chest pain, sudden onset, SOB ↑HR, ↓unilateral breath sounds, ↓tactile fremitus

(changes w/ body position)

Pericarditis → ST segment elevation everywhere. (Tender) ⊕friction rub could have PR segment depression. Tx: NSAIDs
Costochondritis - sharp pain. Pain/Tenderness.

Acute Coronary Syndrome

BF Tx: Aspirin (↓ mortality of MI by 25% and unstable angina (non-stemi) by 50%)

↓ mortality
Aspirin
Thrombolytics
if Angioplasty
Metoprolol (Not time sensitive)
Statins
Plavix, Prasugrel
ACE inh (if EF is low)

What ↓ mortality in STEMI?
Thrombolytics and primary angioplasty (w/in 90 min of onset.)
Percutaneous Coronary Intervention (PCI) (angioplasty is a form of PCI)

When use prasugrel or clopidogrel?
- aspirin allergy, pt. undergoes angioplasty or stenting, acute MI (prasugrel has greater efficacy than plavix but causes more bleeding)

Cocaine induced chest pain? CCB

Non-Stemi

- No thrombolytic use
- Heparin used → lowers mortality in Non-Stemi (↓ MI & EKG) (Lovenox is superior)
- GPIIb/IIIa inh. ers (abciximab)

Note: Thrombolytics Do Not lower mortality unless STEMI or New LBBB

Indications for CABG:

- 3 coronary vessels >70% stenosis
- Lt. main Coronary artery stenosis >70%

Goal of LDL < 70 if CAD and DM.

Post MI:

Aspirin
Clopidogrel
β-blockers
Statin
ACE

Congestive Heart Failure (CHF)

S3 gallop + ↑ JVP + peripheral edema (↑ fluid overload)

Used ECHO

Systolic Dysfunction ↓ EF
Tx: ACE, ARB
Metoprolol, Carvedilol
Spironolactone
Diuretics
Digoxin (↓ Symptoms but does Not ↓ mortality)

Diastolic dysfunction ↓ or nml EF
Tx: Beta blockers (Metoprolol (Carvedilol)
Diuretic

AAA repaired if >5cm

S3 gallop (mnemonic: CHF) = Dilated left Ventricle
- causes: Idiopathic, infections, alcohol, HTN, MI

S4 gallop = left ventricular Hypertrophy

If to test new infarct "ie" = CK-MB

Ischemic Heart Dx:
MA: CK-MB rise 3-6hrs, elevated 1-2 days
Troponin rise 3-6hrs, elevated 1-2 weeks
Myoglobin - 1st to rise 1-4hrs

Cardiomyopathy

Dilated - MCC - alcohol, adriamycin, radiation, chagas' Dx
Tx: ACE, ARBs, β-blockers, spironolactone

Hypertrophic (Cardiomyopathy) - SOB on exertion, and S4 gallop
Tx: β-blockers, Diuretic

Restrictive Cardiomyopathy - MCC sarcoidosis, amyloidosis, hemochromatosis, cancer, Glycogen storage
⊕ Kussmaul's sign (↑ JVP on inspiration)
Tx: treat underlying

CHF w/ EF < 35% are candidates for implantable defibrillator (MCC death in CHF is arrythmia)

If CHF and QRS > 120 msec - use Biventricular Pacemaker

Valvular Heart Dx

Young female = MVP
Healthy athlete = HOCM
Immigrant, Pregnant, > Mitral stenosis (Dysphagia, Hoarseness, AFib)
Diastolic rumble after opening snap
Turners, Coarctation of Aorta = Bicuspid Aortic Valve
Palpitations } MVP
Atypical chest pain } MVP
not w/ exertion

Pericardial Tamponade - SOB, hypotension, JVD
⊕ Pulsus Paradoxus - ↓ BP on inhalation
Electrical Alternans on ECHO
Dx w/ ECHO (Reside in pericardium)
Tx: Pericardiocentesis

Murmurs

Squatting, lifting ↑ Less in Air ↑ venous return to Heart
Valsalva, standing up ↓ venous return to Heart

Inspiration RIGHT
Expiration LEFT

Most all Murmurs ↑ w/ ↑ venous return
except HOCM and MVP

Mitral regurgitation = Heard at APEX and radiates to axilla

AS (gets crescendo-decrescendo systolic murmur. MA = Left Heart Catheterization. nml = <30 mmHg, severe stenosis if >70 mmHg)

If Valsalva increases murmur = Diuretics indicated

Amyl nitrate improves murmur = ACE inhibitor indicated

vasoDilator ↓ Amyl Nitrate ↑ (afterload) ↓ (preload)

Valvular lesion	Handgrip (afterload)	Amyl Nitrate (preload)
AS	↓	↑
AR	↑	↓
MS	negligible	negligible
MR	↑	↓
VSD	↑	↓
HOCM	↓	↑
MVP	↓	↑

When to answer exercise thallium testing or stress echocardiography:

- EKG is unreadable
- Digoxin use
- Pacemaker in place
- Left Ventricular Hypertrophy
- Any baseline abnormality of the ST segment of EKG

When to answer Dipyridamole or adenosine thallium stress test or dobutamine echo?

- COPD
- Amputation
- Deconditioning
- lower extremity ulcer
- obesity
- Weakness / Previous Stroke
- Demented

Dipyridamole: inhibits Phosphodiesterase enzyme
cAMP → (PDE) → cGMP
Blocks platelet aggregation, smooth muscle relaxation
Note: absorption is pH dependent so PPI's will inhibit
Tx overdose: Aminophylline

Adenosine: vasodilator - creates flow into lesions based on relative coronary flow reserves
∴ echo stress is not suitable to use w/ vasodilators
use with Ventricular Tachycardia (avoid in Asthma and sinus Tachy)

If tests show reversible ischemia = Angiography

If ↑ Synchronized Cardioversion = V. Tach w/o pulse, acute pulmonary edema, afib, aflutter, SVT
Unsynchronized Cardioversion = V. Fib, or V. Tach w/o pulse.

Acute Pulmonary edema = Tx: O2, furosemide

Clinical (SOB, rales, S3, cyanosis) Dx
↑ nitrates, morphine.