# Cardiac Review

**LETS GET THE BASICS**

**"THE FORMULA"**

**SV + HR= CO**

- Cardiac output = Blood Pressure
- Be mindful of signs of perfusion
- MAP (>60 to perfuse the kidneys)
- 4-8L/min
- Cardiac Index (specific to body surface) (>2.5L/min/m) (2.8-4.2L/min)

FACTORS THAT AFFECT BLOOD PRESSURE

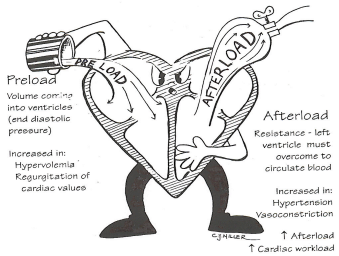
- Circulating Volume
- Diameter of Arterioles
- Strength of contraction
- Heart Rate

PRELOAD:

- "Starling's law" (↑ stretch-↑contractility)
- Volume coming into ventricules (end diastolic pressure)
- ↑'st when you have regurgitative valves and hypervolemia

AFTERLOAD

- Resistance L-ventricle must overcome to circulate volume
- ↑'st when you have HTN & vasoconstriction
- ↑ afterload ↑ cardiac workload (↑ myocardial oxygen demand)




EJECTION FRACTION

- The % of blood that is ejected out of the ventricle during systole (% of diastolic volume ejected during ventricular systole)
- Heart does not completely empty ventricles
- Normal volume of the ventricle is 120mL's
- Normal Ejection fraction is 50-70%
- 120(ventricular volume mL)-60 (Ventricular systolic volume) = 60/120= 50%

FILLING CHAMBERS

- 80% of blood flows passively from the atria to ventricles.
- 20% requires energy
- Ventricular emptying dependent on contraction

ANGINA

- Imbalance between O2 demand and supply.
- Thrombus, Emboli, Spasm, ↓perfusion/hemoglobin, and Hypoxia. Pain due to Lactic Acid >30 minutes= irreversible

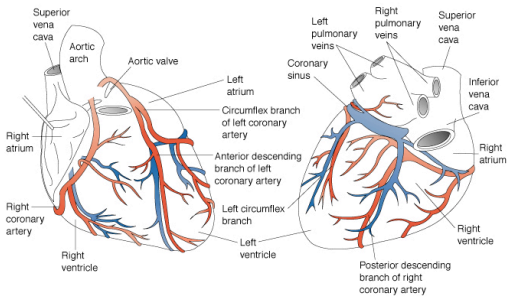
CIGARETTES: (Cardiovascular disease due to)

* Vasoconstriction/Reduced O2 availability ↓CO/ Thrombosis and Platelet activation

**NUR 272**

**CARDIOLOGY**

VESSELS OF THE HEART




- First arterial branches off Aorta
- Very dependent on CO/encircles like a crown
- Exclusive blood supply to cardiac tissue
- Perfusion during Ventricular diastole.

**Left Coronary Artery (LCA)-Splits into two branches**

- **Left Anterior Decending (diagonal small branch)**
- Anterior wall of LV
- Interventricular septum
- Bundle Branches
- Left Circumflex
- L-Atrium
- L-Lateral wall of L-Ventricle

**Right Coronary Artery (RCA)**

- 90% of time, extends to crux, then down towards apex into interventricular grove
- 90% of time, Right side dominant (forms Post. Decending artery)
- Branch anteriorly=marginal artery
- Supplies: R-Atrium, R-Ventricle, Inferior wall of L-Ventricle and Posterior Wall.

R==== Right Coronary Artery Infarct (RCA)
I=====Inferior Wall MI
B====Bradycardia, Nausea, vomiting, abdominal pain
L====Left Coronary Artery Infart (LCA)
A==== Anterior wall MI
T==== Tachycardia, SOB, Rales, Possibly Hypotension

INFERIOR WALL vs. ANTERIOR WALL MI

INFERIOR WALL:

- Conduction issues (RCA blocked ↓ SA node perfusion.
- Hypotensive: Limit NTG but give FLUIDS

ANTERIOR WALL

- Tachycardia secondary to ↓ CO
- Hypotensive due to ↓ CO
- Give +ionotrope (limit fluids)

RIGHT VENTRICULAR INFARCTS

* 10-50% of patients with RCA, occurs with Posterior Wall

**HEMODYNAMIC MONITORING**

- Measures pressures in vessels/chambers of heart
- Waveform conversion.
- Pressure bag to prevent blood from backing up under ↑ pressure and prevent clotting in line.

**A-LINE (Arterial Line)**

- Direct arterial access
- Accurate BP's
- Automatically calculates MAP
- Check cuff pressures once a shift after you take cuff pressures in both arms.
- Zero Q 8hrs & after position changes and Xray
- Access for arterial blood gasses and labs
- Used for "pressor" patients
- Allens test (spasm, occlude both, squeeze hands)
- Transducer
- 4'th ICS, MCL (Phlebostatic axis)

**CVP/RAP (Central Venous Pressure**

- Lumen sits in the superior vena cava. (RA)
- Measures volume and venous return
- RA pressure 2-6mmHg
- Measures fluid volume status (PRELOAD)
- Wet vs. dry (Right sided heart function)
- Complications
- Infection/Line displacement
- Pneumothorax/Thrombosis
- Air Embolism

**Swan Ganz Catheter**

- More direct method of measuring CO
- Catheter into central vein
- Balloon at the tip
- Inflate ➔float until "wedged" into small branches of pulmonary circulation.
- Able to read multiple chamber pressures of the heart.
- Measures overall heart function. (LV emphasis)
- Directly measures CO/CI/Core Body Temperature
- Measures mixed venous saturations
- Measures individual chamber pressures
- WAVEFORMS
- RA= 2-6mmhg
- RV pressure (systolic) 15-25mmHg (Dia 0-8)
- PAP 25/10 (mean 15)
- PAWP=8-12, left ventricular end diastolic pressure

**VENOUS OXYGEN SATURATION**

- SVO2 (balance between O2 supply and demand)-SWAN
- Normal arterial saturation 98-100%
- AT REST: Consume 25% of O2, rest returned to heart. NORMAL SVO2===75%

**NURSING CARE**

- Check calibration of lines at least once per 8hr shift & with position changes.
- Maintain 300mmHg pressure on pressure bags
- Monitor pressure trends
- CXR prior to any new central line insertion
- Manual pressure on the site when discontinued.
- Set alarms and never ignore them
- Assess pulses & distal perfusion distal to arterial line insertion site.
- Check all connections for secure lock
- Change solutions and tubings per hospital policy.

## AUTONOMIC NERVOUS SYSTEM PHARMACOLOGY

### PNS (Peripheral Nervous System)

1) **Somatic Division:** Cranial/Spinal motor nerves that innervate skeletal muscle **(Vol/consc)**
2) **Autonomic Nervous System:** Cranial/Spinal nerves that innervate (cardiac/smooth/glands) **(involuntary/unconscious control)**

Parasympathetic Nervous System

- C III, VII, IX, and X (S2-S4
- Preganglionic=Long_____Post ganglionic short (message to one site cardiac/smooth/gland)
- Cholinergic Nerves (ACH) (act on Muscarinic receptors) if they inhibit called "anticholinergic)
- ONE body system at a time. "REST & DIGEST or FEED & BREED"
- ↓ HR, protects retina, ↓ BP, Empties bowel/bladder, ↑ GI motility, ↑ absorption of nutrients.

Sympathetic Nervous System

- Thoracolumbar Division
- T1-L3
- Preganglionic=short____Postganglionic=long (Global response Cardiac/Smooth/Glands)
- Adrenergic (ACH from PRE and Norepinephrine from POST)
- ALL systems respond "FIGHT or FLIGHT"
- ↑ HR, ↑ BP, Blood shunted to skeletal muscles, ↑ Blood Glucose, Pupils dilate, ↑ Epineprine

**MOST MAJOR BODY SYS. & GLANDS RECEIVE A NERVE FROM EACH but BLOOD VESSELS ARE NOT PNS INNERVATED**

- One system dominates at a time.
- When one Autonomic Nervous System is blocked, the other will work.
- Medications are made to stimulate or block a system or receptor site

### NEUROTRANSMITTERS

- ACH:
- DOPAMINE: most turns into norepinephrine, (Fine motor movement/emotions)
- EPINEPHRINE: made from norepinephrine in the adrenal medulla adrenaline-adrenergic fibers
- NOREPINEPHRINE: Excitatory
- SEROTONIN: Sleep, Behavior, and Consciousness
- GABA: feeling of panic/anxiety (amygdala releases GABA to inhibit this response) Tranquilizing, calming effect on emotion. (inhibitory) Benzodiazepines for these patients)
- ONE OF THREE THINGS OCCUR
- Neurotransmitter released, attaches, and you have a RESPONSE.
- Passes back to pre-synaptic neuron for RE-UPTAKE
- "Extra" may be destroyed by Monoamine Oxidase MAO
- ACH is released and binds with cholinergic receptor sites→action occurs→ACE inactivates extra→ no further actions occur.

ALPHA 1 Receptors: Smooth Muscles of the arterial system (Vasoconstrict when stimulated) FILL TANK
BETA 1 Receptors: "one heart" ↑ Rate, Conduction, & Contraction of cardiac cells and tissue
BETA 2 Receptors: "two lungs" Smooth muscles specifically at bronchiole/vasc. level. Dilation when stimulated
DOPAMINERGIC Receptors: renal arterioles, vasodilate ↑GFR/Urinary output. →sodium excretion
**Inotrope/contractility--------Chronotrope/rate-------Dromotrope/conductivity**

## REVIEW QUESTIONS

2

- What are the four things that determine cardiac output?
- What specifically is the cardiac index?
- How do you know that you client is perfusing well?
- Are the atria accustomed to very low pressures?
- Does blood flow, with normal valve function, one directional? And does the flow change due to pressure gradient changes?
- When does the coronary arteries receive their blood flow?
- What is collateral blood flow?
- The left coronary artery begins as one main artery. What are the two arteries that it branches into?
- What do the two feed?
- The Right Coronary Artery in 90% of the population becomes the posterior descending coronary artery. What does it feed? What are the branches?
- What does RIBLAT stand for?
- What does it mean to be RIGHT or LEFT sided dominant?
- What does a Swan Ganz catheter measure?
- What does the wedge pressure measure?
- What is an ejection fraction? What is the normal ejection fraction?
- What is SVO2? What is the normal value when your body is at rest?
- What three effector organs does the Sympathetic NS stimulate?
- What system is responsible for involuntary muscle movement?
- The adrenergic nervous system is another name for the SNS
- The cholinergic nervous system is another name for the PSNS
- There are specific neurotransmitters responsible for carrying on the "message" in the SNS
- There are specific neurotransmitters responsible for carrying on the "message" in the PSNS
- What happens to the messengers when they don't all bind to effector organs receptor sites?

## SYMPATHOMIMETICS

- Mimics the SNS
- Produces similar effects to neurotransmitters
- May be Alpha, Beta, or both

DOPAMINE

- Pressor first used for Hypotension
- Titrated to keep SBP >90-100
- Monitor MAP (action dose related
- 5mcg/kg/min (Dopaminergic dose, vasodilates renal arterioles ↑ GFR/urinary output/promotes Na excretion)
- >5mcg/kg/min= ↑ dromotrope/chronotrope ↑ SBP
- >10mcg/kg/min= vasoconstriction, monitor peripheral circulation, and maintain CVP 8-10 for hydration

EPINEPHRINE

- Stimulates BETA 1, ↑ Force of contraction, ↑HR due to increased membrane depolarization of pacemaker cells. ↑ cardiac output
- 1:10,000 Cardiac arrest 1mg IVP
- 1:1,000 0.5ml SQ (anaphylaxis

LEVOPHED (Norepinephrine)

- potent inotrope at </= 2mcg/min OR 2-10mcg/min ALPHA1
- May need low doses of dopamine (renal protectant)
- Monitor for reflex bradycardia
- Potential for ↓ CO due to peripheral vascoconstriction (↓ preload)
- ↑vascular resistance to renal beds by 55%
- Monitor renal function/distal circulation

DOBUTAMINE

- Beta 1 Agonist (facilitates action)
- Ionotrope effect
- ↑ HR with higher doses, can cause dysrhythmias
- Potential cause of ↓ BP due to mild Beta 2 effects on blood vessels.

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ARB (ANGIOTENSIN II BLOCKER) (SARTAN)

- No big difference between the ACE Inhibitors
- Irbesartan (Avapro)
- Losartan (Cozaar)
- Olmesartan (Benicar)
- Valsartan (Diovan)

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ALPHA ONE BLOCKERS

- blocks norepinephrine from vascoconstricting vessels
- Doxazosin (Cardura)
- Prazosin (Minipress)
- Terazosin (Hytrin)
- Alfuzaosin (Uroxatral

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ADENOSINE

- Natural neurotransmitter in your body
- Acts as a sedative
- 6, 12, 12 mg ½ life, 10 seconds give close to the heart AC

## NUR 272 PHARMACOLOGY

CALCIUM CHANNEL BLOCKERS (DIPINE)

- blocks inward movement of Ca into cardiac/smooth cells
- Cardiac Muscle (Negative Inotropes
- Conduction (Negative Chronotropes/Dromotropes for automaticity)
- Vascular Smooth Muscle: vasodilator
- Side Effects: Constipation, headache, edema, hypotension, drowsiness, dizziness, and nausea
- NORVASC (Amlodipine)
- ↓ peripheral vascular resistance
- ↑ Cardiac output (↑ preload)
- PROCARDIA (Nifedipine)
- ↓afterload
- ↓ Myocardial oxygen demand
- CARDIZEM (Diltiazem)
- Effective against fast dysrhythmias (AFIB)
- ↓ ventricular rate with A-FIB/A-FLUTTER
- Dilates coronary arteries w/ Prinzmetals Angina
- ↓ afterload & Myocardial Oxygen Demand

ACE INHIBITORS (PRILS)

- Blocks the Angiotensin I converting enzyme (ACE)
- Angiotensin II is a vasoconstrictor, also causes aldosterone release (↑Na & H2O)
- Blocks conversion from Angiotensin I—II
- Popular with CHF crowd, reducing remodeling
- May cause hyperkalemia w/ ↓ renal function (monitor creatine level) (Angio edema)
- ↓ vascular inflammation and helps with plaque stab.

BETA BLOCKERS (LOL-Little Old Lady) (typically not 1'st for HTN)

- Role with ischemic heart disease to ↓ myocardial oxygen demand.
- Role with certain dysrhythmias due to ↓ stimulation of beta 1 receptors
- Used to treat HTN
- There are non-specific beta blockers
- NORMODYNE (Labetalol)
- Beta1/Beta2 blockade
- Alpha 1 blockade as well
- Used for hypertensive crises
- **BETA 1 SELECTIVE (cardioselective)**

LOPRESSOR (Metoprolol)

- Lower dose, primarily B1 antagonist
- Higher doses aslo B2 receptor blocker
- Toprol XL-extended release
- Atenolol (Tenormin)
- Esmolol (Brevibloc)
- **NON-SELECTIVE**
- Nadolol (Corgard)
- Propranolol (Inderal)
- Sotalol (Betapace)
- Timolol (Timoptic)
- Coreg (with alpha blocker)

Two problems with Beta Blockers: Diabetics (signs of hypoglycemia were blocked and reactive airway disease due to bronchoconstriction)

## VASODILATORS (LETS BRING IT DOWN)

NITROGLYCERIN

- Dilates venous beds in low doses ↓preload=↑HR
- Dilates arteriole beds high doses ↓ afterload
- Coronary artery vasodilator/cerebral (↑ HA)
- Can develop tolerance with long term use
- 10-20mcg/min starting dose, up to 200mcg/min
- Vital signs both arms, higher of the two pressures
- Remove old patches, not over bone

NITROPRUSSIDE (nipride, nitropress)

- binds to vascular smooth muscle causing vasodilation by interfering with Ca influx
- ↓ preload/afterload
- Immediate onset, 2 minute 1/2life
- Used in Hypertensive crisis
- Creates cyanide ion when binded to HgB, can create toxic levels of cyanide. (lower rate, shorter period of time for excretion) (use extreme caution in renal failure)
- 0.5-4mcg/min (protect from light)
- Side effects: Hypotension, seizures, bradycardia, flushing, blurred vision, agitation, ↑ ICP

NICARDIPINE (Cardene)

- anti-hypertensive
- Usually given for patients not responsible to labetalol.

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CARDIAC GLYCOSIDES

DIGOXIN

- + inotrope, -chronotrope/dromotrope
- Check apical heart rate
- CHF/Atrial Fibrillation/other tachycardias
- Can be VERY TOXIC 0.5-2.0ng/ml
- Can cause hypokalemia
- Hold for HR < 60BPM
- Adverse affects: Nausea/loss of appetite, vomiting/diarrhea, dizziness, blurred vision, yellow or green halos, difficult breathing.

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LASIX (Last six hrs)
HCTX (last up to 12hrs)
ZAROXOLYN (thiazide diuretic)

- lasts up to 24hrs
- works better with patients with ↑ creatinine levels
- Can diurese patients with GFR <20ml/min

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CARE AND COMFORT

- Administer through CVAD, use pump
- If placed peripherally (Phentolamine/Regitine) can be given for infiltrate (physician can give)
- Perform double checks with another RN
- Add current weights to pumps
- LABEL LABEL AND LABEL

## THE ELECTROCARDIOGRAM

- Recording of "Electrical Activity" in the heart.
- Gives only the ELECTRICAL activity within the heart
- Does not reflect MECHANICAL activity.

Isoelectric Line: Flat line denoting no electrical activity, heart at rest.

**P Wave:**

- Atrial Depolarization
- From the SA node
- Typically upright

**PR Interval:**

- Onset of P wave ends onset of QRS complex
- 0.12-0.20 seconds
- Conduction through the atria>AV Junction
- Can potentially be wide (medications, conditions)

**QRS Complex**

- Q wave: 1'st negative deflection following P
- R wave: 1'st positive after the P wave
- S wave: 1'st negative deflection after the R wave
- Ventricular Depolarization

**Q-T Interval**

- Beginning of QRS complex to end of T wave
- Denotes ventricular activity
- 0.36-0.44
- varies with HR

**ST Segment**

- between end of QRS and beginning of T wave
- Point where QRS complex meets the ST segment is J point
- Early repolarization of ventricles

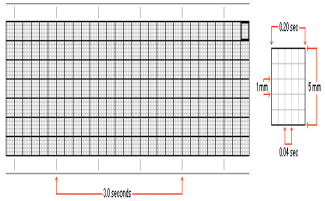
**T Wave**

- Rounded and upright
- Ventricular Repolarization
- Typically follows direction of QRS complex

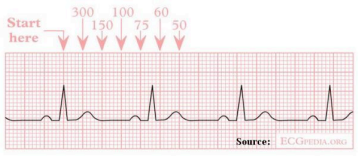
**U Wave:**

- follows T wave when seen
- May represent repolarization of Purkinje fibers
- Easier to see with slower rates, < 2 mm

COUNT THE RATE




Calculating the Heart Rate




## BASIC CARDIAC ELECTROPHYSIOLOGY

- Special pathways exist w/I the myocardium
- *Automaticity* (Pacemaker cells only)
- *Excitability:* ability to charge
- *Conductivity:* Ability to transmit the electrical charge
- *Refractoriness:* Inability to prematurely depolarize (Toilet bowl theory)
- *Contractility:* It either does or does not.
- *Polarization: (Gear Up)* Resting membrane potential outside is +, and inside it is –
- *Depolarization: (Trigger):* Ions move across the membrane changing the apposing charge===action potential
- *Repolarization (Re*-charge): Ions move back in to position, cell becomes negative again.
- *Refractory Period:* (absolute)Myocardial cell will not respond. (toilet bowel) QRS complex>>>>>peak of T wave (relative) some cells have repolarized and have the potential to be stimulated. (downslope of T wave

### *CONDUCTION SYSTEM*

PACEMAKER SITES (From 1 of 3 sites)

- Altered by: MI, Acidosis, Electrolytes Abnormalities, Hypoxia, Age, Drugs, Altered pathways, Pericarditis, Electrocution
- **Atria (SA Node 60-100)**
- Posterior wall, right atrium
- Natural Pacemaker
- Right Coronary Artery supplies blood
- **AV Junction (AV node 40-60)**
- AV node & Bundle of His
- Gatekeeper, electrical link between atria/ventricles
- Right Coronary Artery supplies blood
- **Right and Left Bundle Branches**
- Right-R Ventricle
- Left-L Ventricle
- Conducts the electrical impulses down to ventricles
- **Ventricles (Purkinje Fibers (20-40)**
- Spreads impulses received from bundles to ventricular walls.
- 0.08-0.12 seconds to occur
- **Ectopic Pacemaker (Ectopic beat)**
- Impulses created outside the normal conductive pathway.
- Creates ectopic beat (not from SA node)

**** LOOK AT THE QRS COMPLEX**********

Narrow: Atria or AV Junction

Wide: Artificial Pacemaker, Bundle or Ventricular in nature

Does it have "P" waves?

## FIVE RULES FOR BASIC INTERPRETATION

- Is there a "P" wave for every "QRS" complex?
- What is the "PR" interval?
- What is the "QRS" interval?
- Is the rhythm regular or irregular?
- What is the ventricular and atrial rate?

**USE OF COUMADIN:**

- Low risk emboli= ASA
- Moderate risk emboli= ASA or COUMADIN
- Great risk of emboli=COUMADIN
- INR 2-3
- Risks: Cardiac Failure, DM, Age, HTN, and Stroke.
- Risk of Fall vs. benefits.

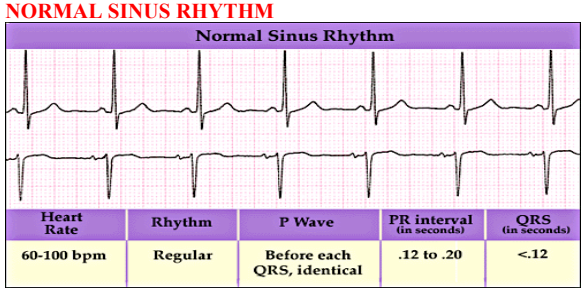
**APICAL-RADIAL PULSE DEFICIT**

- Apical pulse might differ from radial rate.
- What is conducted might not be felt in the periphery.



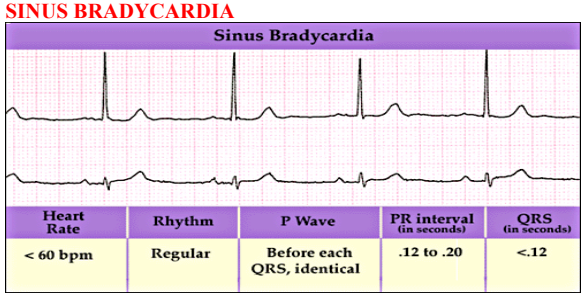
SEE BELOW CARDIAC RHYTHM STRIPS

## NORMAL SINUS RHYTHM




| Heart Rate | Rhythm | P Wave | PR interval (in seconds) | QRS (in seconds) |
|---|---|---|---|---|
| 60-100 bpm | Regular | Before each QRS, identical | .12 to .20 | <.12 |

## SINUS BRADYCARDIA




| Heart Rate | Rhythm | P Wave | PR interval (in seconds) | QRS (in seconds) |
|---|---|---|---|---|
| < 60 bpm | Regular | Before each QRS, identical | .12 to .20 | <.12 |

## SINUS TACHYCARDIA

PREMATURE VENTRICULAR CONTRACTIONS & VENTRICULAR TACHYCARDIA (MI, valve disease, RHD, etc)

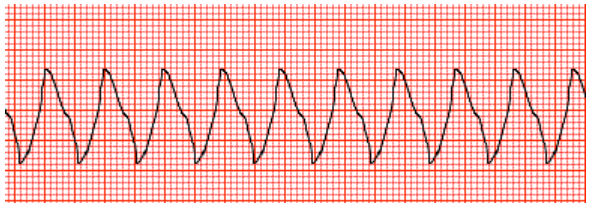
PVC: Ectopic beat orginating from ventricle before next sinus beat

Sinus Rhythm w/ bigeminy, trigeminy, quageminy & Couplets.

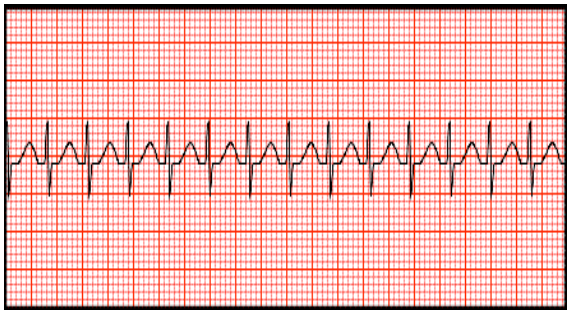
Normal beat>PVC====Bigeminy

Normal beat, normal beat>PVC====Trigeminy

Normal beat, normal beat, normal beat>PVC===Quadgeminy.

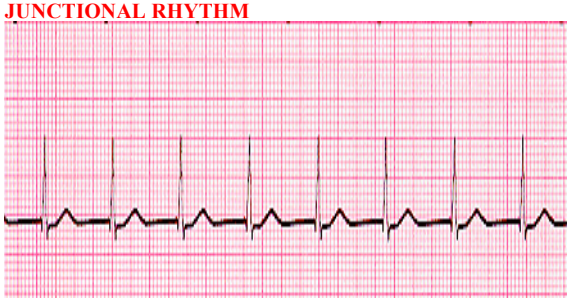


## SUPRAVENTRICULAR TACYCARDIA

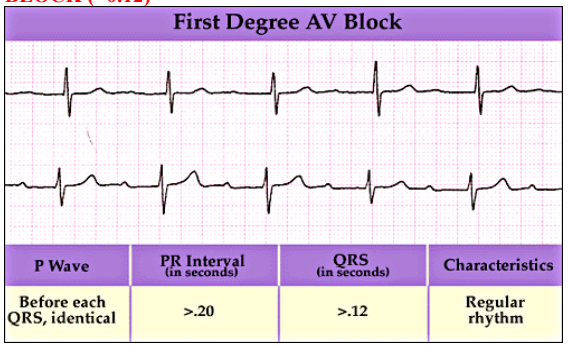


"DO YOU SEE A P WAVE"???????

## JUNCTIONAL RHYTHM

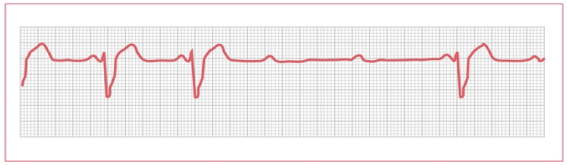


## NORMAL SINUS RHYTHM w/ FIRST DEGREE AV BLOCK (<0.12)

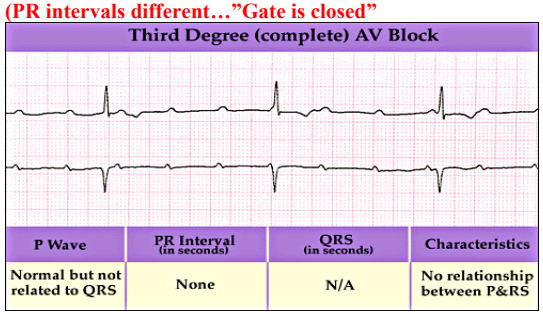



| P Wave | PR Interval (in seconds) | QRS (in seconds) | Characteristics |
|---|---|---|---|
| Before each QRS, identical | >.20 | >.12 | Regular rhythm |

## SINUS RHYTHM w/ SECOND DEGREE AV BLOCK (PR interval the same)



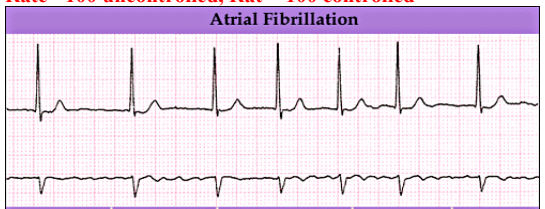
## SINUS RHYTHM w/ THIRD DEGREE AV BLOCK (PR intervals different…"Gate is closed"




| P Wave | PR Interval (in seconds) | QRS (in seconds) | Characteristics |
|---|---|---|---|
| Normal but not related to QRS | None | N/A | No relationship between P&RS |

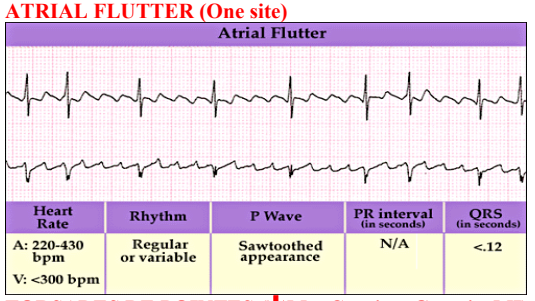
## ATRIAL FIBRILLATION (multiple sites)

Rate >100 uncontrolled, Rat < 100 controlled



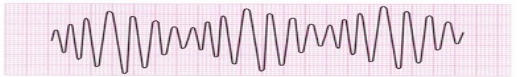

| Heart Rate | Rhythm | P Wave | PR interval (in seconds) | QRS (in seconds) |
|---|---|---|---|---|
| A: 350-650 bpm<br>V: Slow to rapid | Irregular | Fibrillatory (fine to course) | N/A | <.12 |

## ATRIAL FLUTTER (One site)



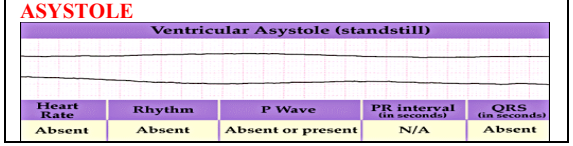

| Heart Rate | Rhythm | P Wave | PR interval (in seconds) | QRS (in seconds) |
|---|---|---|---|---|
| A: 220-430 bpm<br>V: <300 bpm | Regular or variable | Sawtoothed appearance | N/A | <.12 |

## TORSADES DE POINTES (↓Mg, Cocaine, Genetic, MI)



## VENTRICULAR FIBRILLATION

## ASYSTOLE

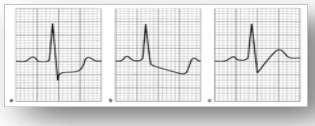



| Heart Rate | Rhythm | P Wave | PR interval (in seconds) | QRS (in seconds) |
|---|---|---|---|---|
| Absent | Absent | Absent or present | N/A | Absent |

## ISCHEMIA vs. INJURY & INFARCTION

- unstable lesions lead to infarction
- Result from blockages in coronary artery
- Rupture of lesions release substances causing platelet aggregation, thrombin generation & vasoconstriction.
- Acidosis develops and depresses myocardial contractility.
- Collateral circulation??? Is it present (age of patient)
- **Cellular acidosis**
- Decreased myocardial contraction (↓ CO)
- Electrolyte imbalances ( K, Mg, lead to Torsades)
- Impulse conduction complications (BBB)
- Dysrhythmias (biggest complication)

## ISCHEMIA

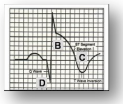


- ST segment depression >/= 1mm below the isoelectric line in 2 leads
- Can lead to Infarction
- Also note K levels

## INJURY/INFARCTION

- ST segment elevation >/= 1mm above the isoelectric line in 2 leads.
- May see T-wave inversion
- May see Q-waves

## PATHOLOGICAL Q-WAVES



- < 20 minutes, subendocardial injury (small not through the muscle completely, NO Q wave present)
- > 1-6hrs, transmural infarction noted (large completely through the muscle Q WAVE PRESENT, possibly inverted T wave) (re-modeling of the ventricle, HF, Aneurysm, rupture)

## EMERGENT PACEMAKER YOU SEE (BRADYCARDIA)

- Hemodynamically significant bradycardia
- Bradycardia unresponsive to drugs.
- I ISUPREL
- D DOPAMINE
- E EPINEPHRINE
- A ATROPINE

## ACUTE CORONARY SYNDROMES

Coronary Perfusion:

- Heart Rate (too fast or too slow can ↓ CO)
- Aortic Pressure (Can be too high or low>>>>>CO
- Metabolic Activity (Metabolic Acidosis affects, contractility.
- Collateral Circulation: If it isn't present, suddent cardiac death. Hearts way to "preserving itself"
- Diameter of the vessel: "↓ leads to ↑pressure.
- ↓ CO can lead to an MI

Oxygen to the tissues:

- Oxygen has to get in
- Got to have Hemoglobin to carry it.
- Pump
- Ability to unload O2

STABLE vs. UNSTABLE ANGINA

STABLE

- Predictable.
- Usually activity related
- Cold/stressed
- Rest and Nitroglycerin
- Pain is easily relieved

UNSTABLE

- Take a nitro and it continues
- Patient rests and the pain continues
- The pain is unpredictable or becomes more frequent

PRINZMETALS (Variant)

- Spasm or lesion
- Does not usually occur with activity or stress
- Usually occurs with some degree of stenosis or fibrous plaque.
- Can be secondary to recreational drugs.

### ACUTE CORONARY SYNDROMES CAUSED BY

- Rupture of atherosclerotic plaque (activation of SNS/Catecholamine release)
- Vasospasm
- Obstruction of plaque or re-stenosis
- Inflammation of coronary artery
- Increased oxygen demand.

INFLAMMATORY CHANGES

- Lipid Core (fibrous cap) seperates the clotting factors in the blood. (thicker it is the better)
- Inflammatory cells and macrophages erode protective cap and it ruptures.
- Contact with blood in the lumen
- Platelet aggregation and thrombin then generated
- THROMBOSIS-----then ACS
- Lactic acid causes the pain ↓ CO

## WHATS IT FEELS LIKE?

- Pressure sensation, squeezing, substernal or epigastric pain.
- Dull ache, does not increase with deep breath
- Elephant sitting on my chest.
- Nausea, indigestion, weakness, upper back pain, and fatigue

## SIGNS AND SYMPTOMS

- Diaphoresis, Cool/mottled skin, palpitations
- Nausea/vomiting, dysrhythmias, L/R HF
- Altered mental status.
- RIGHT SIDED HEART FAILURE
- JVD, Peripheral edema, Hepatomegaly
- Abdominal pain, loss of appetite, ↑ CVP
- Increased abdominal girth

## ANGINAL EQUIVALENTS

- **Shortness of breath**
- **Fatigue**
- Pallor/Palpitations
- Anxiety, Nausea and vomiting
- Diaphoresis, lightheadedness
- Impending doom, weakness
- Indigestion/heartburn

## DIAGNOSTIC TESTING AND LAB RESULTS

- EKG (but be careful with it……..)
- Electrolytes, CBC w/ diff, CPK w/ isoenzymes
- CXR to rule out aortic dissection prior to lytics
- **CPK w/ Isoenzymes (MB%)**
- MB% > 5% indicative of MI
- Levels q8hrs X 3
- Elevated up to 72hrs post MI
- (elevate with, injections, cardioversion, trauma, rhabdomyolysis, CPR, debrillated)
- **Troponin I (T)** (proteins of actin/myosin unit released with cardiac damage/measured in blood)
- Should be "O"
- Peaks in 24hrs (elevated 7-14 days post MI
- ↑ renal failure, cardiac surgery, shock, myocarditis, heart failure(↑BNP, normal EKG and no chest pain), cardiac contusion, cardioversion
- **Myoglobin**
- Unreliable (muscle damage, peaks early)
- Stress Testing (on or off treadmill)
- ECHO (Detect wall abnormalities/valvular problems) EF %
- Computed Tomography Angiography (X-rays taken following dye injection, takes about 20 minutes, overestimate size of occlusion)
- PCI (Percutaneous Coronary Intervention)
- BMS: Bare Metal Stent (no medication, 20% reocclusion rate)
- DES: Drug eluded Stent (with medication Sirolimus-RAPAMUNE immunosupressant)
- CRP (C-reactive protein) released by the liver due to inflammation
- <1.0 Low RISK
- 1.0-3.0mg/L= Average RISK
- >3.0mg/L= high risk for MI

↑elevated in: aged, increased body mass, HTN, and Tobacco smoke.

**REVIEW QUESTIONS GO AHEAD QUIZ YOURSELF!**

- **Differentiate the anginas**
- **Typically, what does an inverted T wave mean?**
- **Typically, what does ST segment depression mean?**
- **Typically, what does ST segment elevation mean?**
- **With an MI, what could be your EKG changes?**
- **Differentiate between a non-Q wave and a Q wave MI**
- **What does acidosis do to heart muscle?**
- **What is an anginal equivalent?**
- **Name some common ones**
- **What is different about women?**
- **Differentiate between the cardiac markers**
- **What does "IDEA" stand for?**
- **Name the drugs…**
- **What are the drugs used acutely for a patient with unstable angina or MI?**
- **What is the FIRST one used?**
- **Name the categories of drugs used for maintenance post MI – understand their mechanism of action for their use**
- **What are some complications post MI?**
- **How would we manage them?**
- **Be able to discuss the labs that need to be closely monitored post MI**

## MEDICAL MANAGEMENT OF THE MI PATIENT

### ACUTE MANAGEMENT

- MONA (morphine, oxygen, nitro, and asprin) ** O2 FIRST
- Beta blockers
- Fibrinolytics
- Arrhythmics
- Diuretics********monitor renal status*******
- ACE Inhibitors
- ↓ Heart failure risks and overall mortality
- Anticoagulants
- LMW Heparin (lovenox)
- IV Heparin
- Coumadin (LV impairment or A-fib)
- Statins (works in liver)
- Zetia (decreased amount of absorbtion in the GI tract specifically the small bowel)
- Vytorin(statin/zetia together)
- Antiplatelet aggregates
- Stool Softners

### PLAVIX

- Synergistic w/ ASA
- ↑ risk of bleeding
- Avoid use of PPI's (Protonix) (different metabolism and difficult to deal with side effects.
- Altered effects in some, doesn't always work

### NITROGLYCERIN

- Need IV for Nitro virgin (give Nitro if VS ok
- Give 3Sl 5 minutes apart
- Take BP's both arms and use higher of two
- Repeat B/P in between each NTG
- EKG before and after pain relief
- NTG drip for unrelieved pain and consider morphine.

### THROMBOLYTIC ENZYMES (ASE)

- Break up the clotting mechanisms
- alteplase recombinant (Activase)
- anistreplase (Eminase)
- reteplase recombinant (Retavase)
- streptokinase (Streptase)
- tenecteplase recombinant (TNKase)
- urokinase (Abbokinase)

### TISSUE PLASMINOGEN ACTIVATOR (TPa)

- Most frequently used
- Bleeding, anaphylaxis
- Used for all clots.

### GLYCOPROTEIN IIb/IIIa Inhibitors

**Inhibitors of platelet aggregation**

- for acute episodes of unstable angina or MI/ patient going for angioplasty with or w/o stenting
- ReoPro, Integrilin, Aggrastat
- Given IVP or through infusion
- Monitor for bleeding/thrombocytopenia

### ANTI-PLATELET AGGREGATES

* Aspirin and Plavix

### BETA BLOCKERS

- Be CAREFUL
- Contraindications
- Heart failure
- Elderly
- Hypotension
- HR > 110, <60
- May be added a few days post MI

### VENTRICULAR DYSRHYTHMIAS

Short Term (No treatment needed)

Long Term (Persist 48 post MI, consider ICD)

### MEDICATIONS THAT INDUCE HYPOTENSION POST MI

- Thrombolytics (bleeding site)
- Beta Blockers
- Ace Inhibitors (monitor creatinine)
- Nitrates
- Calcium Channel Blockers
- Digoxin (PRE-MI)

### CORONARY ARTERY BYPASS GRAFTING (CABG)

- LIMA, Mammary artery more durable
- Saphenous vein
- Secondary prevention (STOP smoking and CONTROL diabetes)
- Mediastinal chest tubes
- Epicardial pacemaker placed.

### COCAINE

- Premature Atherosclerosis
- LVH due to Hypertension
- Coronary artery vasoconstriction
- ↑ O2 demand, thrombus formation

### BASIC CARE AND COMFORT

- RELIEVE THE PAIN, THIS IS PRIORITY
- Oxygen and pulse ox
- Monitor for dysrhythmias
- Monitor Neurological status (lytic therapy-signs of bleeding)
- Monitor labs, vital signs and lung sounds.
- Emotional support.
- Cold patient (bradycardia, a-fib, pressure fluctuations)
- Re-warming (↑ o2 demand) thorazine, morphine, and valium
- CHEST TUBE (No more than 70ml/hr, monitor for TAMPONADE (chest tube drainage STOPS)
- Disturbed Thought Process (clots, sleep disruption or ↓ CO????)
- Monitor for infection, FEED patient, anemia, depression

## ACUTE DECREASE IN CARDIAC OUTPUT

**(Mechanisms that would typically HELP in a ↓CO situation can COMPLICATE things.**

- Chronic heart failure patients have no reserve, this quickly tips the scale for them and they decompensate quickly.
- Activation of SNS can complication the situation (SEE BELOW---THINK ABOUT WHAT IT HAPPENING WITH THE PATIENT)

***PHYSIOLOGICAL RESPONSES TO ↓ CARDIAC OUTPUT***

**↓ CO (due to infection, hypovolemia, dysrhythmias)**

1. Sympathetic→ ALPHA 1 ↑ Vascoconstriction
   BETA 1 ↑ Heart Rate
   BETA 2 → Bronchodilation
2. Chemoreceptors ↑ Respiratory Rate
3. Renin Aldosterone System--- Vasoconstriction/ Aldosterone (H2o-Na)
4. Antidiuretic Hormone (ADH)--- Keeps H2O only

**CARDIOGENIC SHOCK OCCURS WHEN THE BP DROPS BELOW 90 and THE BODY CAN NO LONGER COMPENSATE**

***LETS REVIEW SOME SHALL WE??????***

- ✖ *** There are very specific signs/symptoms of right sided heart failure.**
- ✖ **There are very specific signs/symptoms of acute left sided heart failure.**
- ✖ **Need to know those compensatory mechanisms and how it causes decompensation in acute heart failure**
- ✖ **What does a BNP tell you about your patient's ventricles? Do we always pay attention to the exact number?**
- ✖ **Identify, by labs and diagnostics, that a client is in acute heart failure.**
- ✖ **What are the treatment goals for a patient in acute heart failure?**
- ✖ **What classes of medications would be most likely ordered for a patient admitted with acute heart failure?**
- ✖ **Why is Natrecor ordered for a patient?**
- ✖ **List the responsibilities of the nurse during an acute episode of heart failure.**
- ✖ **Basic care and comfort!**

## ACUTE HEART FAILURE

**OXYGEN GETS TO THE TISSUES:**

- Have Oxygen?
- Hemoglobin to carry it.
- Pump to get oxygen to the tissues.
- Hemoglobin to unload the oxygen to the tissues.

**HEART FAILURE**

- Hearts inability to generate an adequate cardiac output.
- Cardiac Output is compromised:
- Afterload: HTN
- Preload: Lasix (fluid is held)
- Heart rate: too fast vs. too slow (a-fib ↓ 20%)
- Contractility: ability to pump AMI
- **LEFT SIDED** (pulmonary edema)
- Left Ventricular issue (MI, HTN A-Fib)
- **RIGHT SIDED** (systemic edema/periphery)
- Acute pulmonary Diseases
- Effect of Left sided failure
- Right Ventricular infarct.
- **SYSTOLIC HEART FAILURE**
- Low ejection fraction (<50%)
- Think decrease CO
- Weakness, fatigue, ↓ exercise tolerance
- **DIASTOLIC HEART FAILURE**
- Ejection fraction is normal

**CAUSES OF HEART FAILURE**

Cardiogenic: MI, Acute Heart Failure, Acute HTN, New A-fib or change in rate (GOAL IS TO ↑ HEART FUNCTION)
Non Cardiogenic: Heroin, ARDS, or Sepsis (GOAL IS TO TREAT CAUSE)

**PATHOPHYSIOLOGY**

**YOU HAVE TO THINK CELLULAR LEVEL……**

1. HYDROSTATIC PRESSURE ↑↑↑↑↑↑↑↑, which passes the ONCOTIC pressure. (fluids start to shift)
2. VASOCONSTRICTION secondary to hypoxia.
3. FLUID shifts from capillary beds in to the ALVEOLI in the lungs. (red blood cells too)
4. ↓ GAS exchange.

**HEMODYNAMICS**

- ↑ PAWP >25mmHg)
- ↑ CVP
- Dysrhythmias
- EKG Changes (is this why their in Acute CHF)

**SIGNS AND SYMPTOMS**

- Dyspnea and Tachypnea
- Hypertention →→→ hypotention/cardiogenic shock
- Crackles/wheezes
- Fatigue
- Restlessness, CP, Tachycardia
- Pale, Cool, Clammy Skin

**LABS**

**BNP**

- B-Type (brain) natriuretic peptide
- Hormone secreted by the ventricles in response to TOO much stretch
- Higher it is the worse it is
- Can decrease with proper management
- Helps differentiate COPD vs. CHF
- FOLLOW TRENDS
- < 100 no heart failure
- 100-300 heart failure present
- >300= mild heart failure
- >600= moderate heart failure
- > 900= severe heart failure

**ABG's**

- Respiratory alkalosis (early) followed by respiratory acidosis
- CXR, electrolytes, LFT's, Thyroid function, 12EKG



## *MANAGEMENT GOALS, BASIC CARE AND COMFORT*

- Improve oxygenation status, ↑ contractility, ↓ preload/afterload, diurese, and ↓ workload on the heart (NORMALIZE RATE)
- ACUTE MANAGEMENT: CPAP (possible intubation) → Diuretics (Lasix/Bumex ↓ preload)→ ↓ afterload (NTG-Natrecor) → ↑ Contractility (Dobutamine) → Maybe Morphine??
- MAINTENANCE THERAPY: ACE inhibitors (ARBS) → Beta Blockers → + Ionotropes (Digoxin) →Aldactone (get rid of the H2O/Na)→ Nitrates → Antidysrhythmics → Anticoagulants → ASA
- LONG TERM/CONTINUED THERAPY: Biventricular Pacemaker, Implantable Defibrillator (INOTROPIC agents) MONITOR RENAL FUNCTION
- BASIC CARE AND COMFORT
- 1) Monitor Oxygen Saturation (wean down to nasal cannula) 2) Elevate legs if feasible 3) Assess Lung sounds 4) Cough up secretions 5) Assess Urinary Output (30ml/hr or 0.5ml/kg/hr 6) bedrest
- 7) Monitor fluid intake 8) Group activities 9) Passive ROM (clots form easily) 10) Watch diet 11) Admitting weigh asap 12) emotional support
- NATRECOR
- Binds to receptor sites in smooth muscle===relaxation/vasodilation (reducing the preload and afterload)
- Promotes diuresis and natriuresis, reduces PCWP in CHF patients
- Short half life
- 2mcg/kg bolus over 60 seconds, DRIP 0.01mcg/kg/min for up to 48hrs. (DRUG NOT TITRATED) Can increase to 0/03 mcg/kg/min for desired effect
- Plasma BNP levels will be affected (falsely elevated) due to recombinant form.

The label on a bag of Natrecor states that there is 6mcg/1mL in the bag.
You have a patient that weighs 185 pounds
The order says to infuse 0.01mcg/kg/min
What should your infusion rate be in mL/hr??

LIDOCAINE
- Antidysrhythmic used for VENTRICULAR heart muscle
- Used for Ventricular Tachycardia/Fibrillation
- 1.0mg/kg IVP

AMIODARONE
- Acts on ALL cardiac tissue
- Affects smooth muscle by ↓ peripheral vascular resistance
- ↑ perfusion to the coronary arteries
- Blocks effects of sympathetic nervous system stimulation
- Pulsed (beating heart) 150 mg/IVP
- Ventricular Fibrillation 300mg/IVP
- ADVERSE EFFECTS
- Sinus Arrest
- Bradycardia
- Hypotension
- Hepatotoxicity
- Pulmonary Toxicity

**CARDIAC ARREST DRUGS**

********EPINEPHRINE**********
- 1 mg 1:10,000 IVP Q 3-5 minutes

********AMIODARONE**********
- Ventricular dysrhythmias as noted above
- Or consider Lidocaine

********VASOPRESSIN**********

********MAGNESIUM**********
- Torsades De' Pointes

********SODIUM BICARBONATE*******
* 1meq/kg

**DRUGS ET vs. LINE**

L Lidocaine
E Epinephrine
A Atropine
N Narcan

**SPECIAL CIRCUMSTANCES**
- Patient with a pacemaker ( attempt to put it around the device, to secure pad to the patient)
- ICP (around the device, device could potentially be firing)
- Transdermal patches (attempt to remove)
- Patient in water ( remove the patient)

**CARDIOVERSION AND DEFIBRILLATION**

SYNCOPE: Is a symptom

NON-ARRHYTHMIA CAUSES OF PALPITATIONS
- Stimulants (caffeine, nicotine, cold remedies, bronchodilators)
- Anemia
- Hyperthyroidism
- Hypertension
- Anxiety
- Fear
- Hypoxia

ARRYTHMIA CAUSES OF PALPITATIONS
- PVCs and PAC's
- SVT, Atrial Flutter and Fib
- Ventricular Tachycardia w/ structural disease
- Use of antidysrhythmics can cause arrhythmias

IN HOSPITAL TELEMETRY vs. HOME MONITORING

TELEMETRY:
- Explain what you are doing
- Good contact with skin
- Monitor for irritation & set alarms
- ASSESS YOUR PATIENT

HOME:
- Holter monitoring (24-48hrs)
- Event Recorder (patient activated)
- Continuous Output Monitoring (2wks)

EPS (Electrophysiology Studies)
- Catheters guided in to the heart
- Electrical stimulation used to "induce" a dysrhythmia.
- Ventricular Fibrillation, Cardiac Perforation are complications.

**ASYSTOLE**
- You cannot defibrillate this, there is no electrical current
- CPR, Epinephrine, and maybe Atropine 1mg max of 3mg

**CARDIOVERSION**

TOO MANY PVC's
- IS YOUR PATIENT HYPOXIC???
- K, Ca, Mg, ARE????????????
- Is it "re-perfusion ectopy?

**"SYNCHRONIZED"**
- Treats certain dysrhythmias other than V-Fib or pulseless Ventricular Tachycardia.
- Interrupt the ectopic focus HOPING the sinus nose picks up.
- "SHOCKS" on the "R" wave
- Obtain a TEE FIRST
- STABLE RHYTHMS (Atrial fib/flutter)
- UNSTABLE RHYTHMS (SVT/V-TACH w/ pulse)
- BASIC CARE AND COMFORT
- Airway assessment/vital signs
- Monitor rhythm
- Assess the skin

---

**SUDDEN CARDIAC DEATH**
- Unexpected death occurs one hr after the onset of cardiovascular symptoms.
- MOST COMMONLY ASSOCIATED WITH VENTRICULAR FIBRILLATION
- MI, Cardiomyopathy, electrocution, PE, Drug Toxicity, Cerebral Hemorrhage
- Ventricular Fibrillation
- Abrupt event
- Unresponsive
- A= Airway
- B= Breathing
- C= Circulation/CPR
- D= Defibrillation/AED

**CHECK YOUR "H" and "T"**

| | |
|---|---|
| Hypovolemia | Toxins |
| Hypoxia | Tamponade |
| Hydrogen Ion | Tension Pneumothorax |
| Hypo/Hyperkalemia | Thrombosis |
| Hypothermia | Trauma |

---

**DEFIBRILLATION (joules takes 5-15 seconds to charge)**
- Delivery of electric current to the heart muscle through an open or closed chest to terminate a life threatening dysrhythmia so that the normal conduction system can take over.
- ONLY TREATMENT FOR VENTRICULAR FIBRILLATION
- CPR started and defibrillation applied w/i 4 minutes
- SUCCESS
- Length of time the patient was in V-fibrillation
- Function status of the heart
- acid/base balance
- Oxygenation status of the patient
- categories of drugs the patient takes.

HANDS FREE
- ↑ Speed of delivery
- ↓variability of electrode placement and pressure
- Improves operator safety
- Victim benefits
- I'm CLEAR, You're CLEAR, We're ALL CLEAR!

**OK, LETS REVIEW……ASK SOME QUESTIONS**
- **What is the difference between defibrillation and cardioversion?**
- **What type of drug is Amiodarone? Is there a different dose between a beating heart and a dead heart?**
- **What could cause a patient to have non-arrhythmia and arrhythmia PVC's?**
  - **Which ones do we become immediately concerned with?**
- **Where do you place the paddles or hands-off pads for defibrillation or cardioversion?**
- **What are the specific drugs for a cardiac arrest? How do they work?**
- **What are the ABCD'S of emergency care, both basic and advanced?**

## PERICARDITIS

- Inflammation of the pericardium
- Primary disorder, infectious process, uremia, radiation, cancer, congestive heart failure, cirrhosis, MI, Trauma, Post cardiac surgery
- Inflammatory Response is triggered
- Chest pain, usually abrupt
- Pain usually sharp, increases with respirations. SITTING UPRIGHT AND LEANING FORWARD reduces the discomfort.
- Low grade fever /9below 100 or 38.4)
- Dyspnea and Tachycardia
- Classic EKG changes include s-t elevation

## PERICARDIAL EFFUSION

- SLOW accumulation of fluid in the pericardial sac
- 30-50mls normal can accumulate up to 2000ml before symptoms.
- ↑ rate in patients with CHF, valve disease, and MI
- Transudate fluid: passing through membranes (capillary walls), caused by heart failure, overhydration, and hypoproteinemia.
- Exudate fluid: Substance to it (blood and pus)
- Enough fluid---can cause cardiac tamponade.
- RAPID accumulation causes to the heart to be compressed. Could be fast and as little as 100mL's.

## CARDIAC TAMPONADE

- Pericardial Effusion
- Trauma
- Cardiac Rupture
- Hemorrhage
- Systemic congestion (JVD, Edema, Hepatomegaly)
- ↓ diastolic filling (↓SV, ↓ CO)
- Profound cardiogenic shock
- Pulses Paradoxus (pulse that decreases with inspiration), B/P also decreases by 10mmHg during inspiration.
- Muffled heart tones, JVD and hypotension (Becks triad)
- Poorly palpated apical pulse, dyspnea, anxiousness, narrowed pulse pressure
- DIAGNOSIS
- Cardiac Enzymes elevated due to inflammatory process
- ECHO/EKG/ CXR
- Hemodynamical monitoring (left vs. right pressure)
- MANAGEMENT
- NSAIDS
- Pericardiocentesis (EKG, monitor for elevation, evidence of PVC, add amount on to I/O, assess heart/lung sounds, send sample for C&S, Possible surgery
- Analgesics.

## PERICARDIAL WINDOW

- Small piece pericardium is excised
- Drains into the pleural space
- Improvement quick
- Discharged home in a few days

## BASIC CARE AND COMFORT

- NSAIDS around the clock. (monitor for GI upset/bleeding, take with food, monitor renal function and weight.
- Assess chest pain
- Deep breath (use incentive spirometer to prevent splinting
- Administer O2 and maintain pulse ox.

## OTHER CARDIAC/VESSEL ABNORMALITIES

## CONSIDER A SHORT VERBAL QUIZ

- **List the causes of pericardial effusions.**
- **Differentiate between a pericardial effusion and a cardiac tamponade.**
- **How would these patients present differently?**
- **Compare and contrast your assessment for patients with effusion versus tamponade.**
- **Become familiar with pericarditis!**
- **Explain the treatment modalities for each.**
- **What is an aortic dissection?**
- **What is the goal of therapy?**
- **Why would these patients present with neurological signs/symptoms, decreased pulses to the extremities?**
- **What groups of medications would you use to reduce the blood pressure until the patient can get to surgery?**
- **Differentiate between open and closed repairs for aortic aneurysms.**
- **List the signs/symptoms of graft leakage.**
  - **What kinds of labs should you pay attention to?**
- **Why is it so important to monitor renal function post open aortic aneurysm repair?**
- **What are the basic nursing responsibilities pre and post-operatively for patients with aneurysm repair**

## ACUTE ARTERIAL OCCLUSION

- S/S from acute tissue ischemia
- Typically originate in the left side of heart. "lucky to make the turn"
- Pallor (color), Pulses, Paralysis, Paresthesia, Pain
- A/B=I Like to be 1 <0.9 occulsion
- Heparin (weaned to Coumadin later on, Fibrinolytic therapy. Embolectomy (4-6hrs or irreversible limb ischemia occurs. (fasciotomy to treat compartment syndrome. Renal failure do to dye.
- BASIC C/C: Monitor 5 Ps, IV fluids, Protect the limb. Bleeding precautions, lifestyle modifications. Monitor lab work.

## ENDOVASCULAR STENT-GRAFT REPAIR

- Placed percutaneously- femoral artery -fluoroscopy
- Few complications, short hosp. stays, long term unknown.
- LEAKAGE: Ecchymotic areas, ↑ abd. girth, ↓ motor function lower extremities, ↓u/o ↓h/h
- BASIC C/C: EBL, H&H, Vital signs, pulmonary care, get them out of bed, monitor for decreased blood flow. (Cord/bowel)

- Vital signs (early signs of shock), skin color/temperature/condition.
- Watch for dysrhythmias
- Keep at least one IV patent
- Look for signs of JVD
- Progressive improvement

## THORACIC AORTIC ANEURYSM

- Weakness of the aortic wall
- HTN, Trauma, Marfans syndrome, Tertiary syphilis
- Asymptomatic until rupture
- Vague back or neck pain
- Difficult to swallow, hoarse voice or angina.

## THORACIC AORTIC DISSECTION

- Blood penetrates the aortic wall through the intimal tear and then creates a false channel by dissection of the media

## AORTIC DISSECTION

- Involves the aortic
- Dissection typically occludes blood flow
- Chest pain sudden/unremitting
- Migration of pain to back
- Ripping or tearing
- Diminished or absent pulses in one extremity
- Different blood pressures in both arms.
- May be hypertensive
- Shock type signs and symptoms
- Cardiac Tamponade
- Aortic Regurgitation
- Blood loss
- May present with neurological deficits
- Blowel/kidney ischemia or infarction
- Sudden death, usueally within 15 minutes of onset.
- **MANAGEMENT**
- Rapid Diagnosis
- medically attempt to bring down the pressure (Nipride or beta blockers)
- Surgical procedures

## ABDOMINAL AORTIC ANEURYSM (AAA)

- Dilation of the infra-renal aorta (below renal artery) to more than 3cm
- Elective repairs thoracic-6cm or abdominal 5-cm
- HTN, HTN, HTN, Common sudden cardiac death
- Usually asymptomatic until rupture.
- Elective screening Men 60-85 women 60-85
- Early symptoms (GI tract compression satiety, nausea, weight loss. Pulsating mass in abdomen, pain in back/abdomen. Thrombi in distant vessels due to sluggish flow. Tenderness on palpation)
- **RUPTURED ANEURYSM**
- Severe pain in back
- Flank pain only if contained retroperitoneum
- signs of acute hemorrhage internally.
- **FISTULA FORMATION**
- Aortoenteric fistula causes an upper/lower GI bleed.
- **MANAGEMENT**
- High flow O2, monitor, bedrest, calm enviorvment, gentle handling of patient, Surgery
- 2 large bore IVs, Beta Blockers, Nipride, CCB
- SURGERY (monitor for CLOTS post surgery)
- Open procedure less common. Renal/iliac clamp (Assess renal function post surgery.