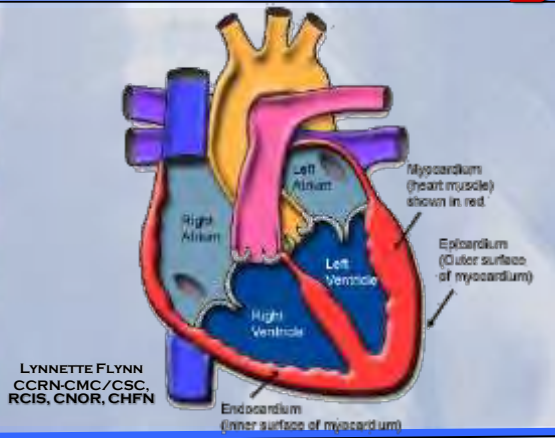
Cardiac Anatomy



Lynnette Flynn
CCRN-CMC/CSC, RCIS, CNOR, CHFN

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Objectives

- Review of **basic anatomy**, cardiac cycle and conduction system
- Explore the various elements that control the CV system including intrinsic and extrinsic mechanisms.
  - Autonomic Nervous system
    - Sympathetic
    - Parasympathetic
  - Renin Angiotensin Aldosterone System

- Analysis of the **action potential** to understand the action of antiarrhythmics by class and Calcium effects
- Relate the mechanics of **myocardial contraction** to explain various medications effects

2

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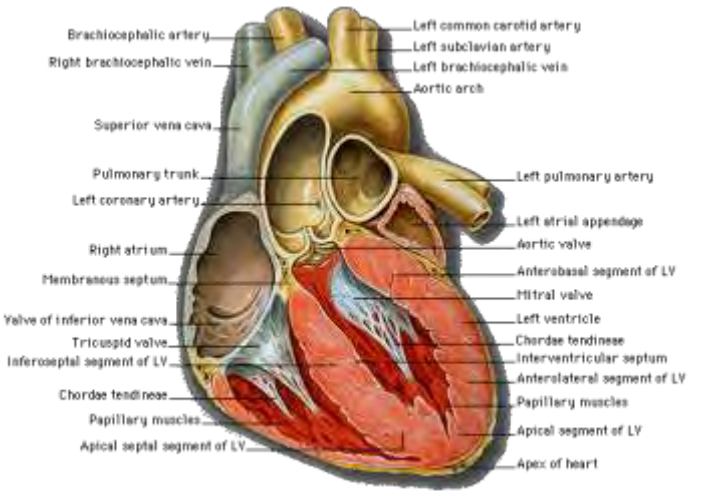
STRUCTURE

Pericardium
Myocardium
Conduction system
Coronaries
Systemic circulation

3



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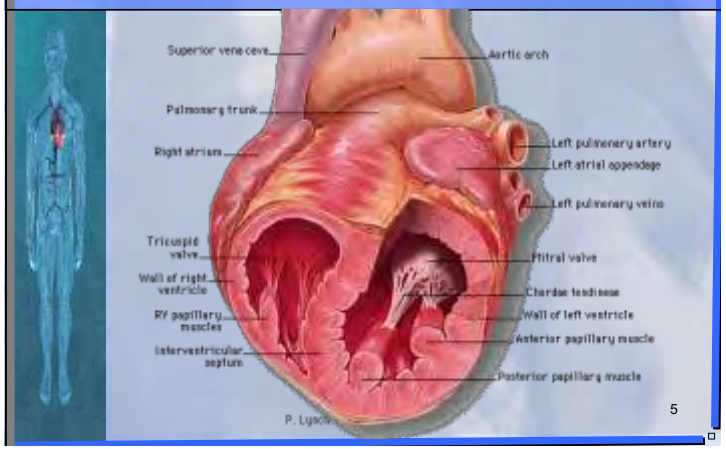
Papillary Muscles



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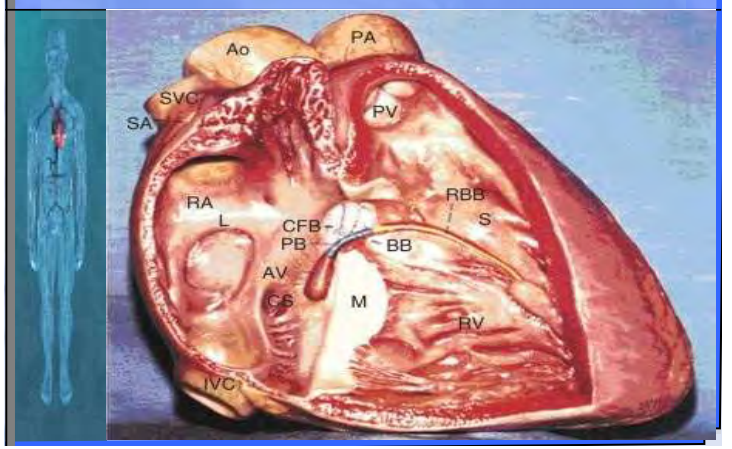
Myocardial Layers



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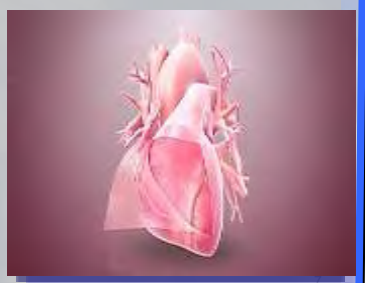
Heart Layers

- **Pericardium** – Visceral and Parietal
- **Myocardium**
  - Epicardium
  - Myocardium
  - Endocardium



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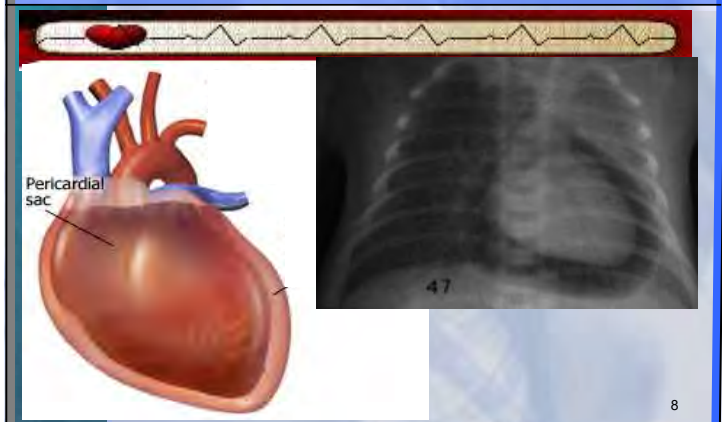
Effusion



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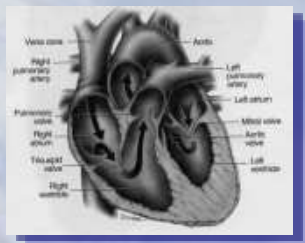
Heart Layers

- Coronaries
  - RCA
  - LCA
    - LAD
    - LCX
  - Collaterals
  - Veins
  - Bloodflow



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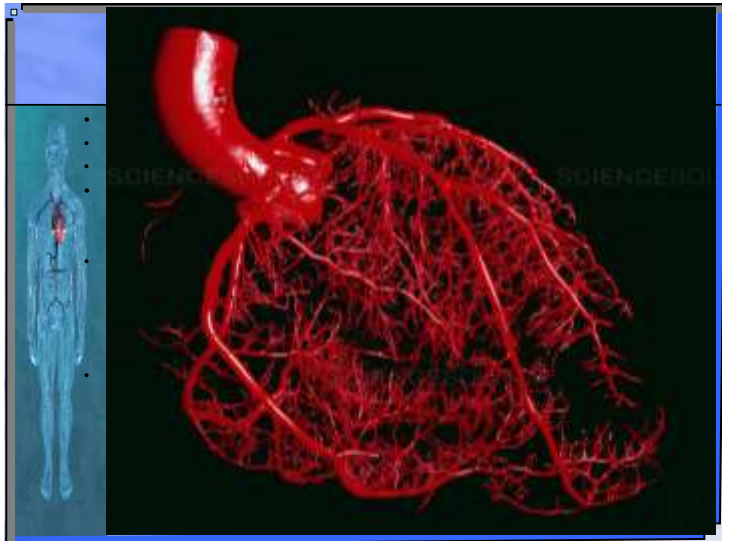

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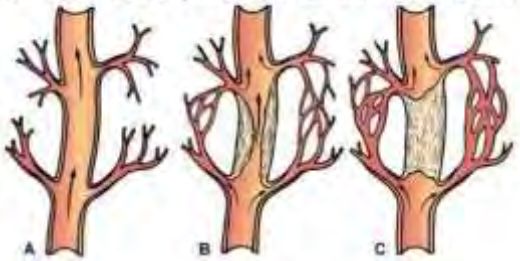
Occlusion/Collateral Circulation



Vessel Occlusion with Collateral Circulation
A Open, functioning coronary artery
B Partial coronary artery closure with collateral circulation being established
C Total coronary artery occlusion with collateral circulation bypassing the occlusion to supply the myocardium

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Veins

16

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Systemic Circulation

Arteries

Veins

Capillaries

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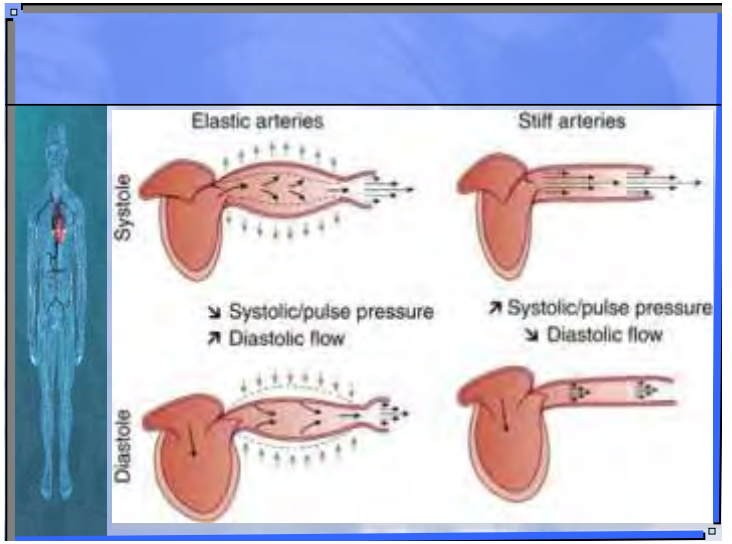

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Systemic Vasculature

- **Systemic vasculature** Blood volume – 5 L
- **Arteries**
  - » Strong compliant elastic-walled vessels that branch off the aorta and carry blood away from the heart
  - » High-pressure circuit
  - » Able to stretch during systole and recoil during diastole
- **Arterioles**
  - » Control SVR and arterial pressure
  - » Strong smooth muscle walls innervated by the ANS
    - » **Adrenergic** (stimulatory) system:
      - » **epi** stimulates beta receptors
      - » **Norepi** – stimulates alpha receptors
    - » **Cholinergic** (inhibitory) releases acetylcholine (decreases HR, releases nitric oxide causing vasodilatation)

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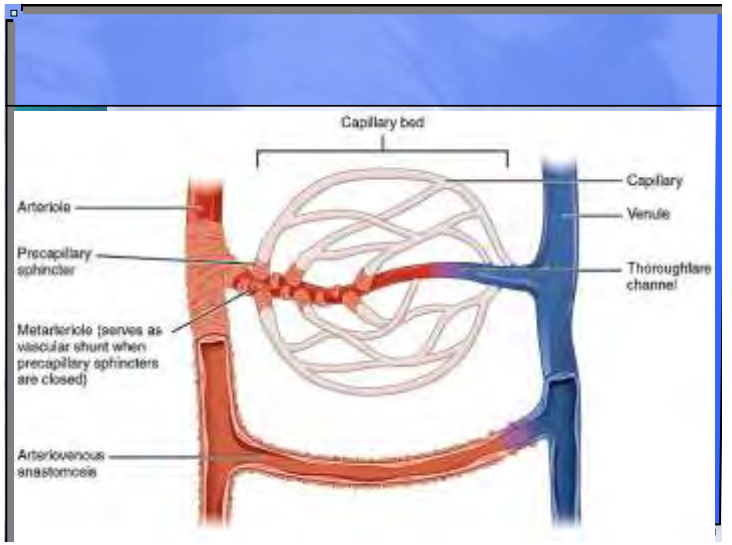

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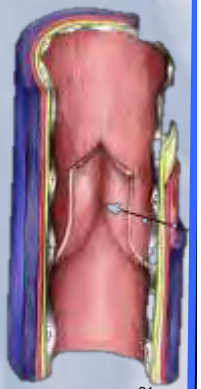
Venous System

- Stores 65% of blood volume
- No muscle layer, compressed by the contraction of surrounding skeletal muscle
- Valves in veins prevent reverse blood flow
- Venous pressure in the LEs is normally 20mmHg or less



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Capillaries

Venules	Arterioles
Capacitance vessels	Resistance vessels
Dilation = ↓ preload	Dilation = ↓ afterload

Nitro
Morphine
Natrecor
Position
Volume

Nipride
Natrecor
Catecholamines
Norepinephrine
Dopamine

Arterial constriction= increases afterload
Venous Constriction= increase preload

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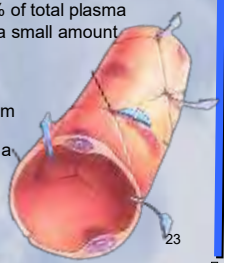
Fluid Homeostasis

- ↑**Capillary hydrostatic** pressure moves fluid from the vessel into the interstitium
- ↑**Capillary osmotic** pressure moves fluid from the interstitium into the vessels
  - **Plasma protein** Albumin accounts for 75% of total plasma osmotic pressure, fibrinogen accounts for a small amount
  - Retains fluid in the intravascular space
  - Prevents edema formation in the interstitium
  - Serum albumin level is a good indicator of a patient's colloid osmotic pressure
- Capillaries lack smooth muscle



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FUNCTION

Extrinsic control

ANS – SNS & PNS
RAAS – Renal/Adrenal axis

Intrinsic

Calcium
Action potential
Contractility

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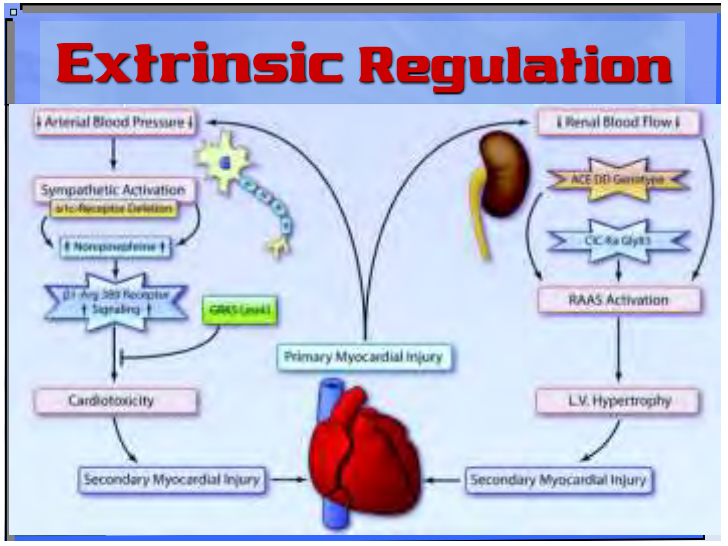
Extrinsic Regulation
↓ Arterial Blood Pressure ↓
↓ Renal Blood Flow ↓
Sympathetic Activation
↑ Norepinephrine ↑
↑ Signaling ↑
ACE DD Genotype
RAAS Activation
Primary Myocardial Injury
Cardiotoxicity
L.V. Hypertrophy
Secondary Myocardial Injury
Secondary Myocardial Injury

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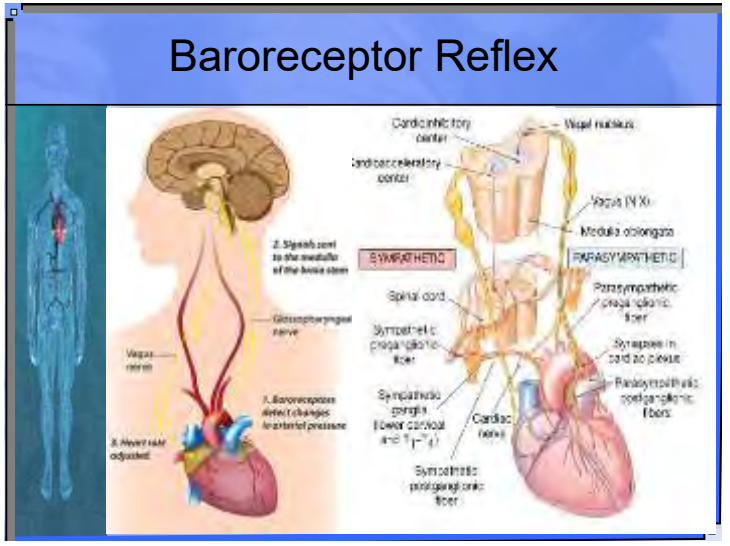
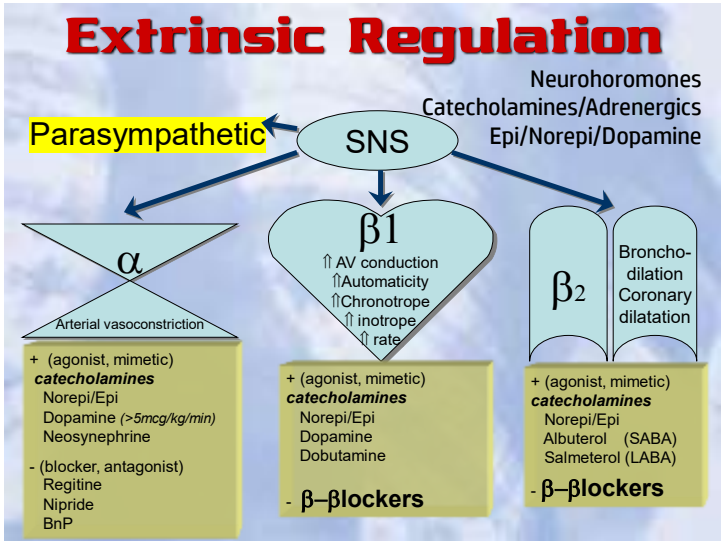

Baroreceptor Reflex
SYMPATHETIC
PARASYMPATHETIC
Vagus (N X)
Medulla oblongata
Spinal cord

30


Extrinsic Regulation
Neurohoromones
Catecholamines/Adrenergics
Epi/Norepi/Dopamine
Parasympathetic
SNS
α
Arterial vasoconstriction
+ (agonist, mimetic)
catecholamines
Norepi/Epi
Dopamine (>5mcg/kg/min)
Neosynephrine
- (blocker, antagonist)
Regitine
Nipride
BnP
β1
⇑ AV conduction
⇑Automaticity
⇑Chronotrope
⇑ inotrope
⇑ rate
+ (agonist, mimetic)
catecholamines
Norepi/Epi
Dopamine
Dobutamine
- β–βlockers
β2
Broncho-
dilation
Coronary
dilatation
+ (agonist, mimetic)
catecholamines
Norepi/Epi
Albuterol (SABA)
Salmeterol (LABA)
- β–βlockers

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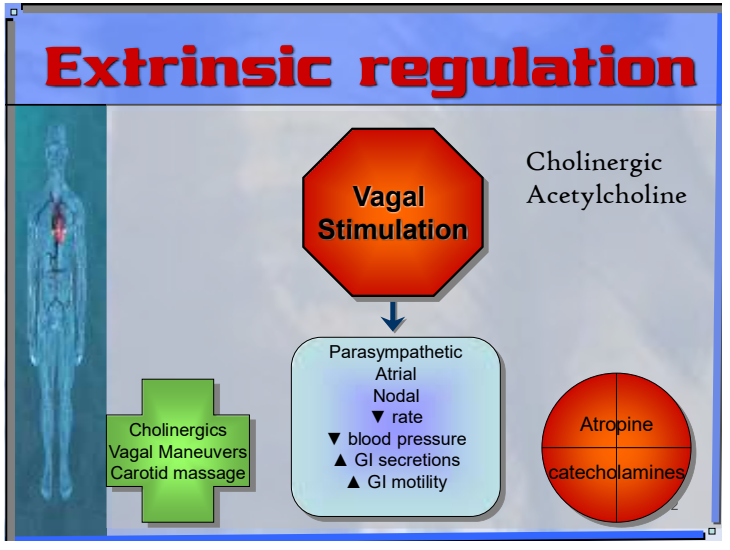
Extrinsic regulation
Vagal
Stimulation
Cholinergic
Acetylcholine
Cholinergics
Vagal Maneuvers
Carotid massage
Parasympathetic
Atrial
Nodal
▼ rate
▼ blood pressure
▲ GI secretions
▲ GI motility
Atropine
catecholamines

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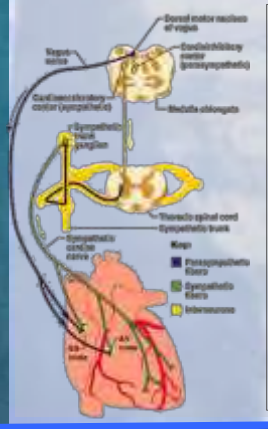
Increased Pressure



Increased pressure

- Stretches arterial walls
- Impulse transmitted from the baroreceptors in the AO arch via the vagus nerve to the medulla
- PNS stimulated, SNS inhibited
- Result: decreased HR
  - contractility
  - dilation of peripheral vessels
  - decreased SVR
  - decreased blood pressure

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Decreased pressure

Decreased pressure:

- SNS stimulated
- PNS inhibited
- Result:
  - increased HR and contractility
  - arterial and venous constriction and
  - increased BP

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CALCIUM

Myocyte Contraction

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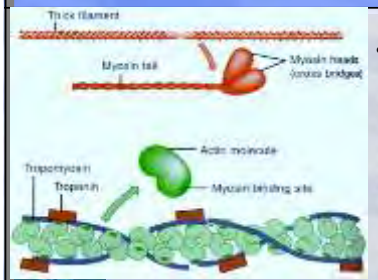
Filament Proteins



- Thick Filament
  - –Myosin
    - head and tail regions
    - 2 chains wrapped around each other (twisted golf clubs)
    - hinge regions
    - movement requires ATP
    - filament has many Myosin units lined up
    - bidirectional arrangement
- Thin Filament
  - Actin - many units in helix (Myosin binding sites)
  - Tropomyosin - covers myosin binding site on actin
  - Troponin - has Ca++ binding site

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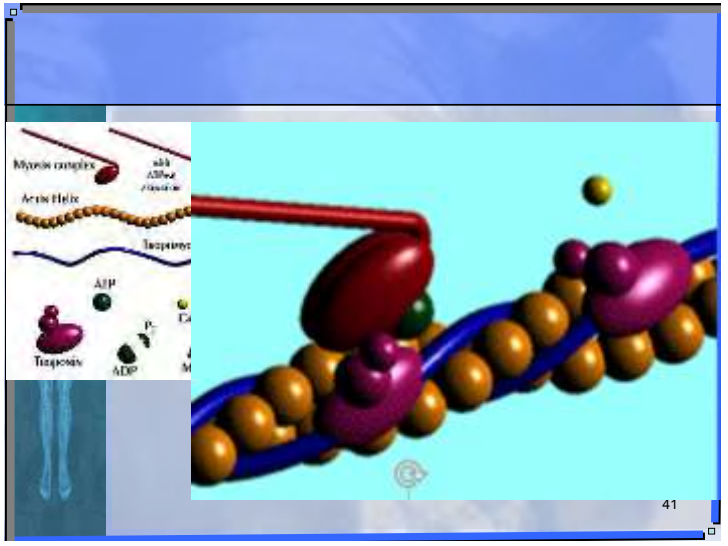

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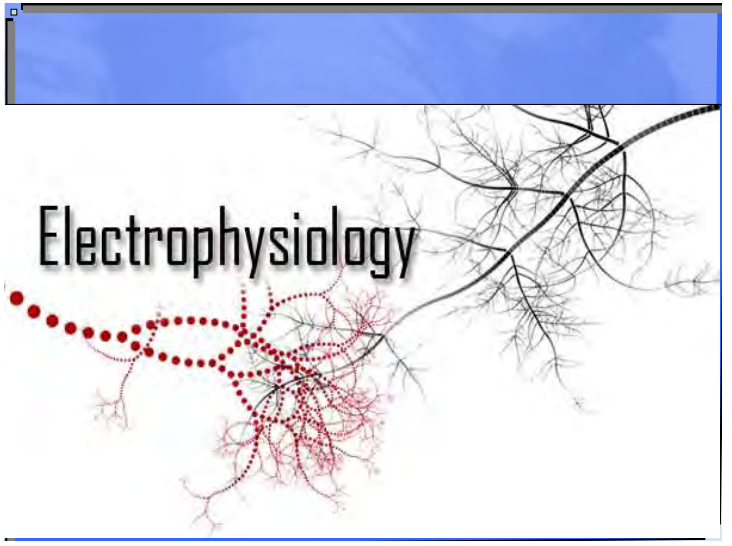

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ELECTROPHYSIOLOGY

Excitability – ability to depolarize and form an action potential

Automaticity: ability to generate an impulse without an outside stimulus

Conductivity: ability to conduct an electrical impulse to neighboring cells spreading the impulse throughout the heart to achieve total depolarization

Refractoriness: Temporary inability of the depolarized cell to become excited

RMP: Resting membrane potential: e- charge of cardiac muscle cell at rest.

Depolarization: Change in the electrical charge of a stimulated cell from negative to positive by the flow of ions across the cell membrane.

Repolarization: recovery or recharging.

Threshold potential: voltage level at which cardiac cells produce an action potential

Stimulation of myocardial cells

- Stimulus may be chemical, electrical or mechanical
- When stimulated, the electrical charge inside the cell becomes less negative and depolarization occurs
- When the threshold potential is reached, changes occur in the membrane
- SA and AV nodes achieve threshold potential first
- Cell membrane permeability is altered and specialized channels in the membrane open allowing the entry of sodium and calcium ions into the cell

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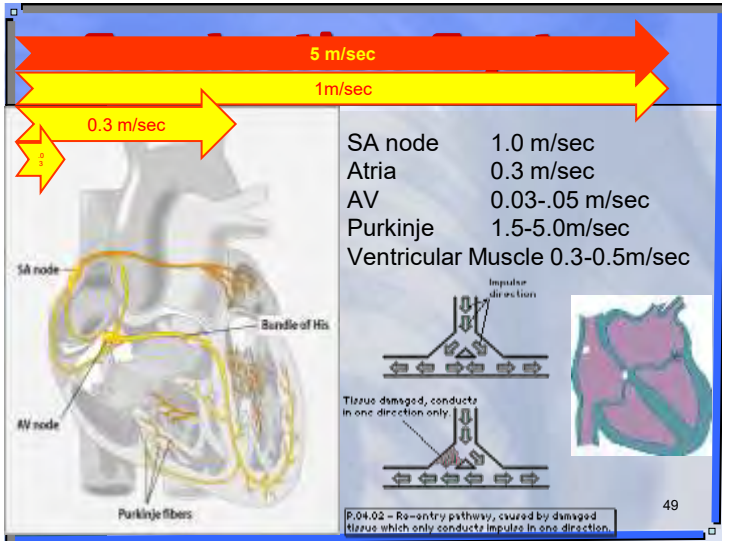

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Pacemaker Potentials In Sinus Fibre
30
0
mV
-40
-80
Threshold potential
Slope
Reduced slope
Phase 4 slope
Phase 4 slope: Na+ - influx through specific channels
Fig. 11-a

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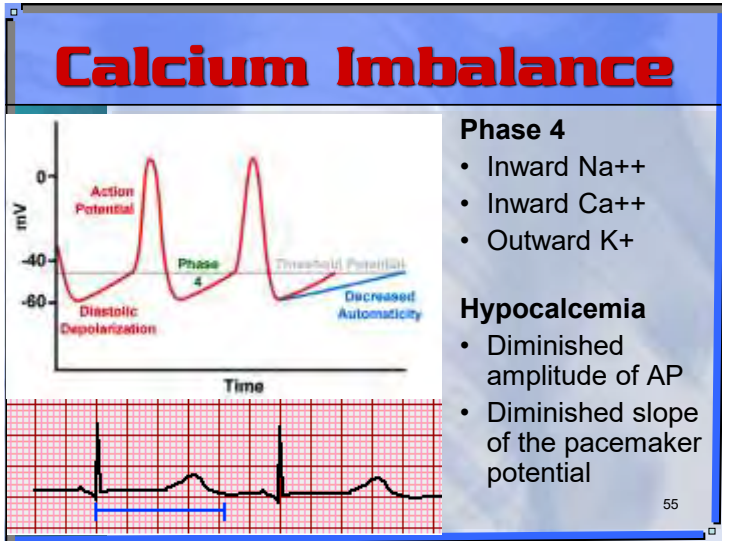
Calcium Imbalance
0
mV
-40
-60
Action Potential
Phase 4
Diastolic Depolarization
Decreased Automaticity
Time
Phase 4
• Inward Na++
• Inward Ca++
• Outward K+
Hypocalcemia
• Diminished amplitude of AP
• Diminished slope of the pacemaker potential
55

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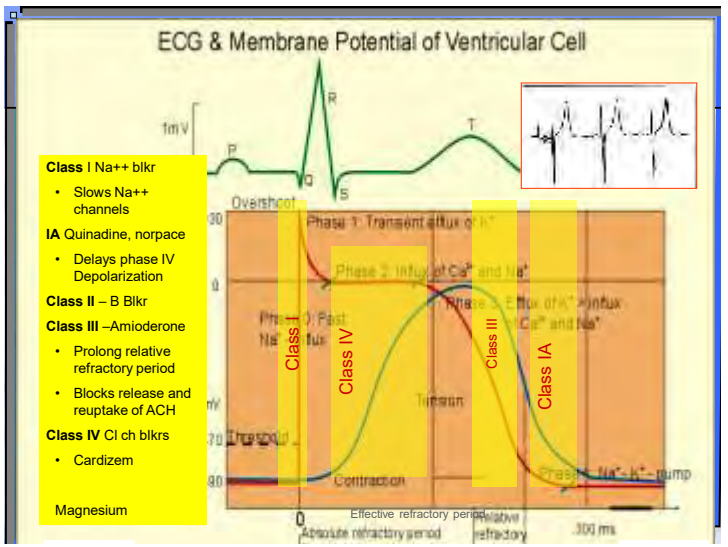
ECG & Membrane Potential of Ventricular Cell
1mV
P
Q
R
S
T
Overshoot
Phase 1: Transient efflux of K+
Phase 2: Influx of Ca++ and Na+
Phase 3: Efflux of K+, influx Ca++ and Na+
Phase 4: Resting membrane potential
Class I
Class IV
Class III
Class IA
Tension
Contraction
Effective refractory period
Absolute refractory period
Relative refractory
300 ms
Class I Na++ blkr
• Slows Na++ channels
IA Quinadine, norpace
• Delays phase IV Depolarization
Class II – B Blkr
Class III –Amioderone
• Prolong relative refractory period
• Blocks release and reuptake of ACH
Class IV Cl ch blkrs
• Cardizem
Magnesium

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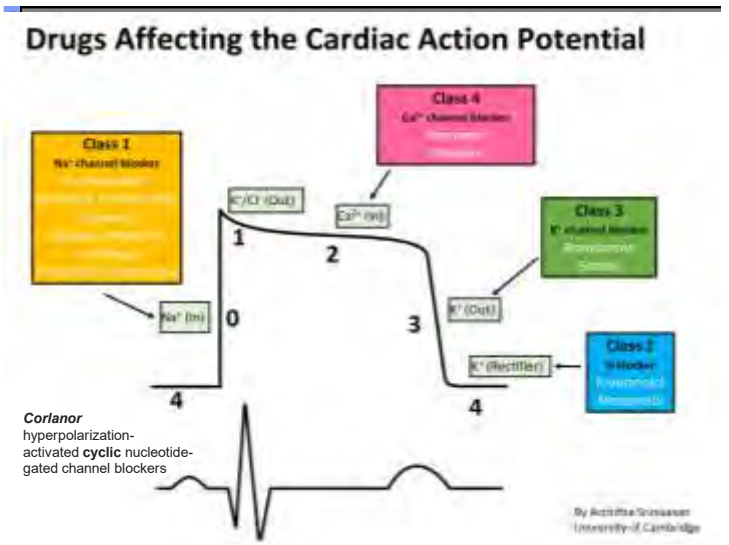
Drugs Affecting the Cardiac Action Potential
Class 1
Class 4
Class 3
Class 2
K+/Cl- (Out)
Ca++ (in)
Na+ (in)
K+ (Out)
K+ (Rectifier)
0
1
2
3
4
4
Corlanor
hyperpolarization-activated cyclic nucleotide-gated channel blockers

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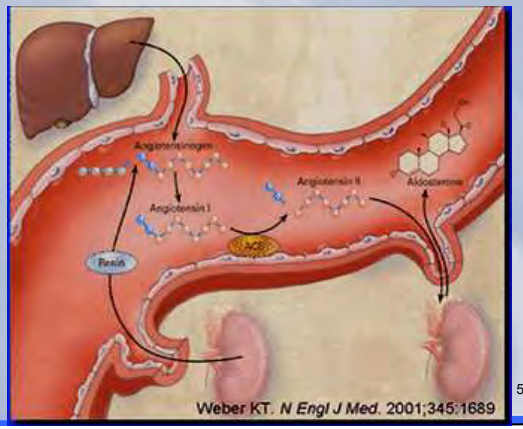
Renin-Angiotensin-Aldosterone System



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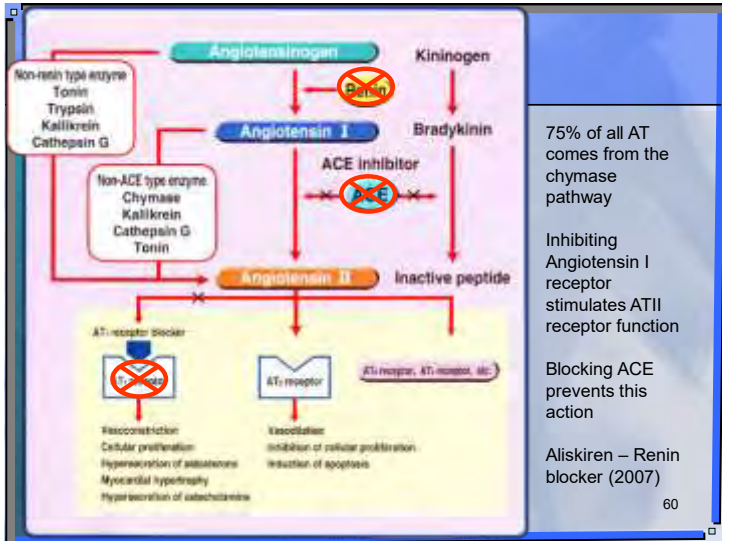

75% of all AT comes from the chymase pathway

Inhibiting Angiotensin I receptor stimulates ATII receptor function

Blocking ACE prevents this action

Aliskiren – Renin blocker (2007)

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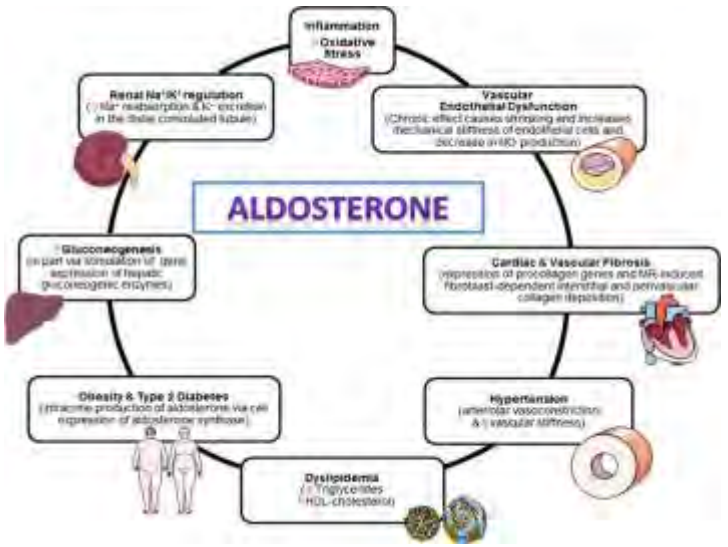

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When blood pressure drops

Decreased blood flow to the kidneys (↑BUN, eGFR)

Renin – angiotensin = ↑ BP/ SVR/ afterload

Inhibited with Meds:

Renin blockers	(aliskiren)
ACE Inhibitors	(*Prils)
ARBs	(*sartans)

Aldosterone = ↑ Na/water retention / preload

Inhibited with Meds:

Aldosterone blockers (spironolactone, Eplerenone)

Lasix to remove water

Natrecor to remove sodium and water

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Application of Learning

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APPLICATION

Preload
Afterload
Contractility
Heart Rate
Heart Sounds

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Preload

Increased by

- Volume Overload
  - Venous pressure, volume, compliance
- Rate of venous return
  - Volume, gravity, mechanical activity
    - Diuretic resistance
    - Renal Insufficiency
    - Antidiuretic Hormone
    - Dietary Indiscretions
    - Aldosterone

Lowered by:

- Sepsis
- Nitro
- BNP
- + HR
- Inflow resistance
- Dehydration

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Afterload

Increased by

- Hypertension
- Aortic Stenosis
- Atherosclerosis
- Norepinephrine
- Angiotensin
- Endothelin

Drugs

- Dopamine
- Catecholamines
- Cocaine/Meth/Crack
- Epi/Norepi

Lowered by:

- Sepsis
- Nipride
- IABP
- BNP

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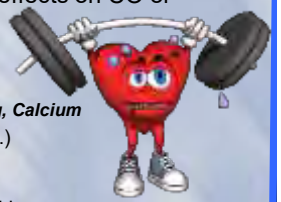
Contractility



- Assessed indirectly through effects on CO or with noninvasive imaging
- **Effectors** include:
  - » Positive inotropic drugs
    - » ***Dobutamine, Primacore, Dig, Calcium***
  - » Increased HR (Bowditch phen.)
  - » SNS stimulation
- **Decreased contraction** is caused by:
  - » Negative inotropes
    - » ***IA antiarrhythmic, βblkrs, CaCH blkrs, barbs***
  - » Hypoxia
  - » Hypercapnia
  - » Myocardial ischemia/infarction
  - » Metabolic acidosis

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Heart Rate

- **Heart rate:** Influenced by
  - – Blood volume status
  - – SNS and PNS tone
  - – Drugs
  - – Temp
  - – Resp
  - – Arrhythmias
  - – Peripheral vascular tone
  - – Emotions
  - – Metabolic status (hyperthyroidism)

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Learning Objectives

Can you now…

- Review of basic anatomy, cardiac cycle and conduction system
- Explore the interaction and implications of neurohormones
- Participate in an analysis of the action potential to understand the action of antiarrhythmics by class
- Explain the description of the heart as an "*endocrine organ*"
- Use an understanding of the mechanics of myocardial contraction to explain various medications effects

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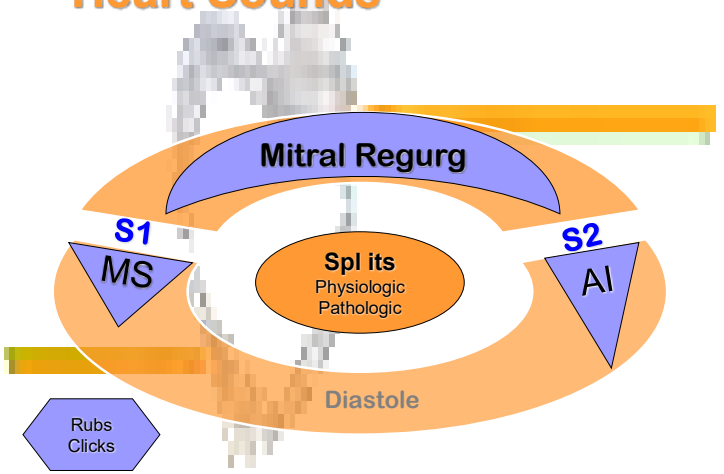
Heart Sounds



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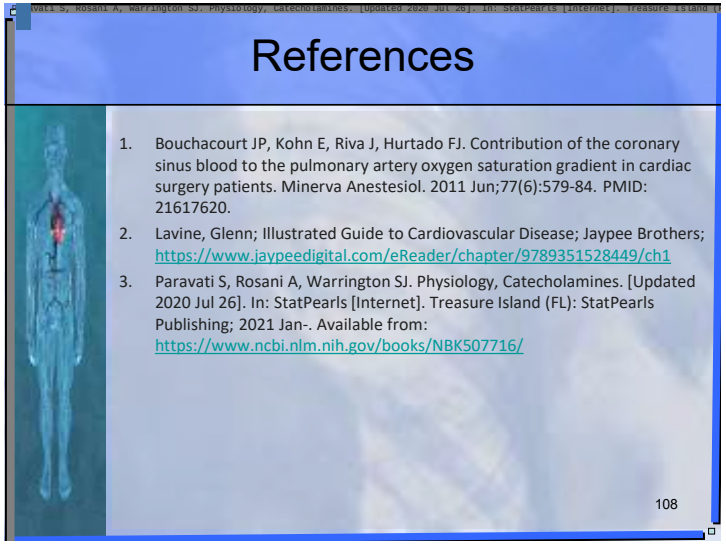
Vati S, Rosani A, Warrington SJ. Physiology, Catecholamines. [Updated 2020 Jul 26]. In: StatPearls [Internet]. Treasure Island (
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1. Bouchacourt JP, Kohn E, Riva J, Hurtado FJ. Contribution of the coronary sinus blood to the pulmonary artery oxygen saturation gradient in cardiac surgery patients. Minerva Anestesiol. 2011 Jun;77(6):579-84. PMID: 21617620.
2. Lavine, Glenn; Illustrated Guide to Cardiovascular Disease; Jaypee Brothers; https://www.jaypeedigital.com/eReader/chapter/9789351528449/ch1
3. Paravati S, Rosani A, Warrington SJ. Physiology, Catecholamines. [Updated 2020 Jul 26]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK507716/
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ECG Dysrhythmias

Lynnette Flynn
CCRN-CMC/CSC, RCIS, CRAT, CEI
February 2022

1

Rhythm disorders

- **Origin**
  - Atrial
    - Afib
    - Aflutter
  - Junctional
  - Ventricular
- **Rate Related**
  - Brady
  - Accelerated
  - Tachy

Conduction related

AV node

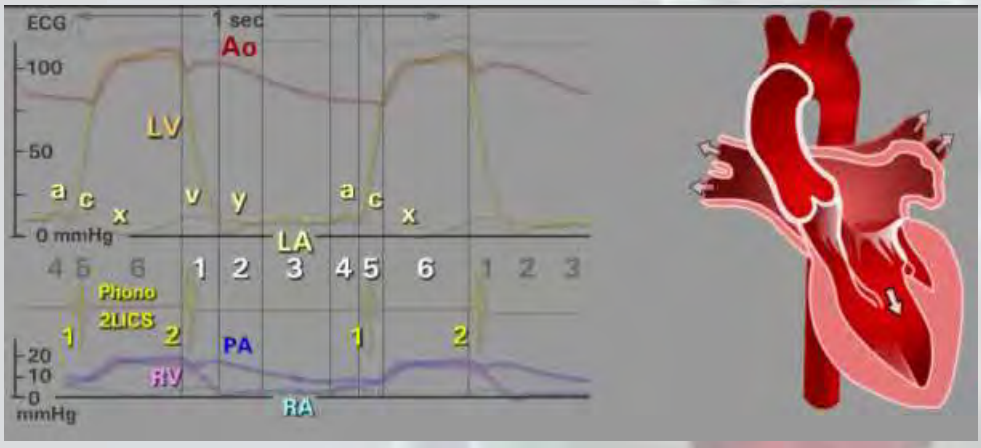
1st degree
2nd degree type I
2nd degree type II
3rd degree

Bundle Branches

BBB

3

Cardiac cycle

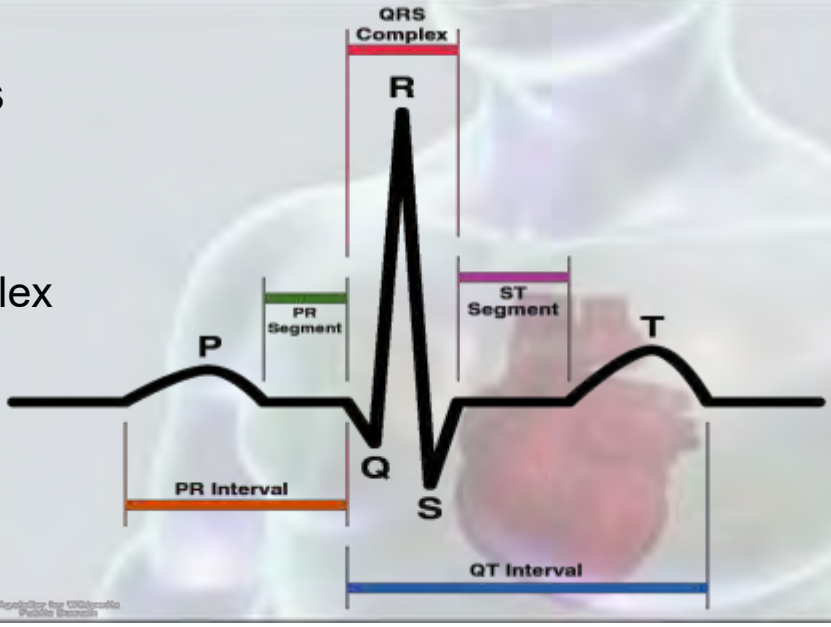


http://blaufuss.org/tutorial/index1.html

4

Waveforms

- **Waveforms**
  - P wave
  - PRI
  - QRS complex
  - T wave
  - U wave

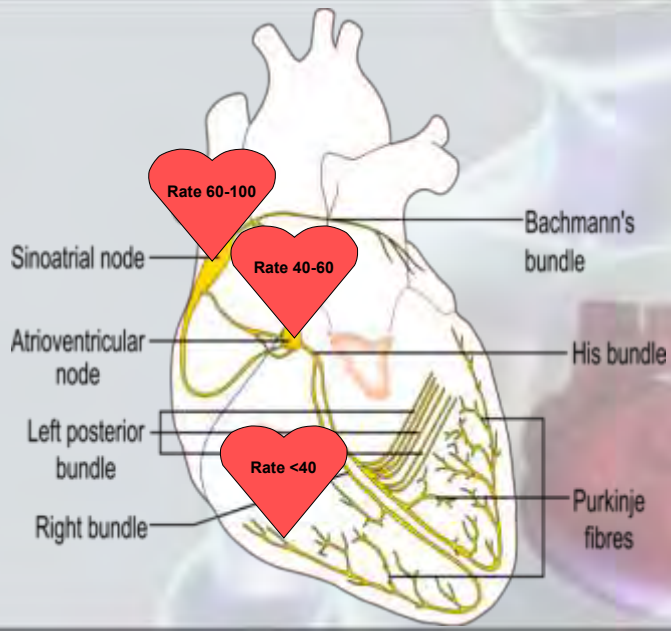


5

Paper

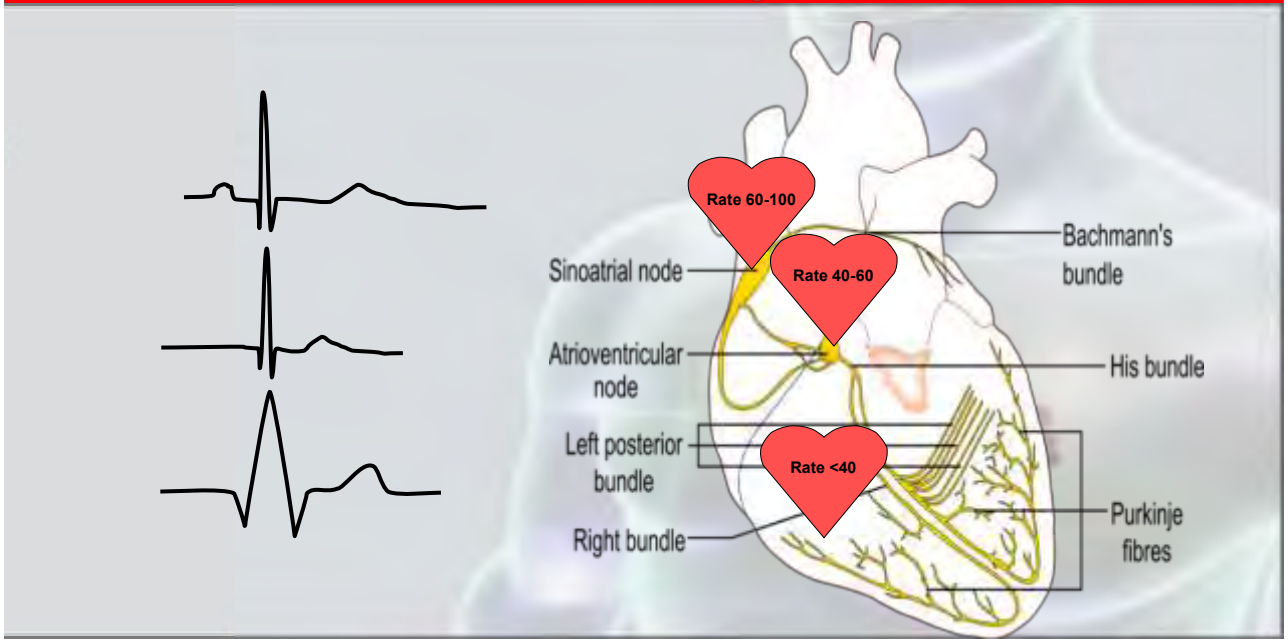
6

Anatomy



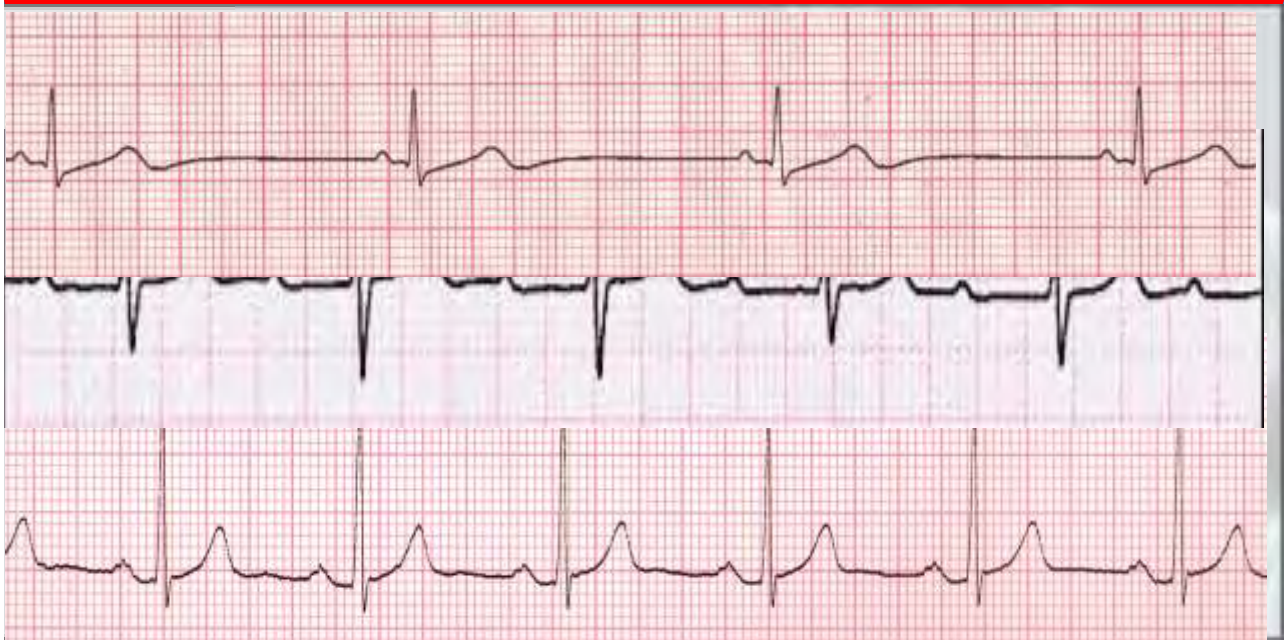
7

Conduction system



8

Not in Handout – View Screen



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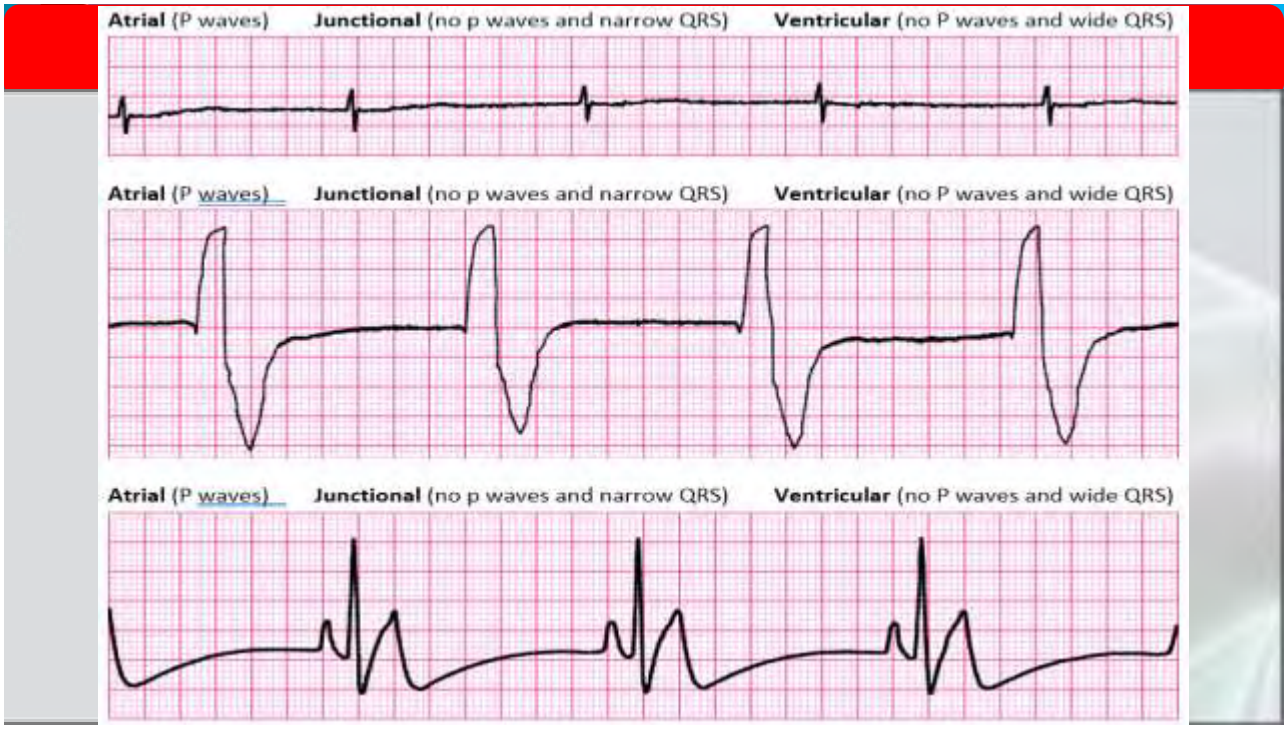
Atrial (P waves) Junctional (no p waves and narrow QRS) Ventricular (no P waves and wide QRS)
Atrial (P waves) Junctional (no p waves and narrow QRS) Ventricular (no P waves and wide QRS)
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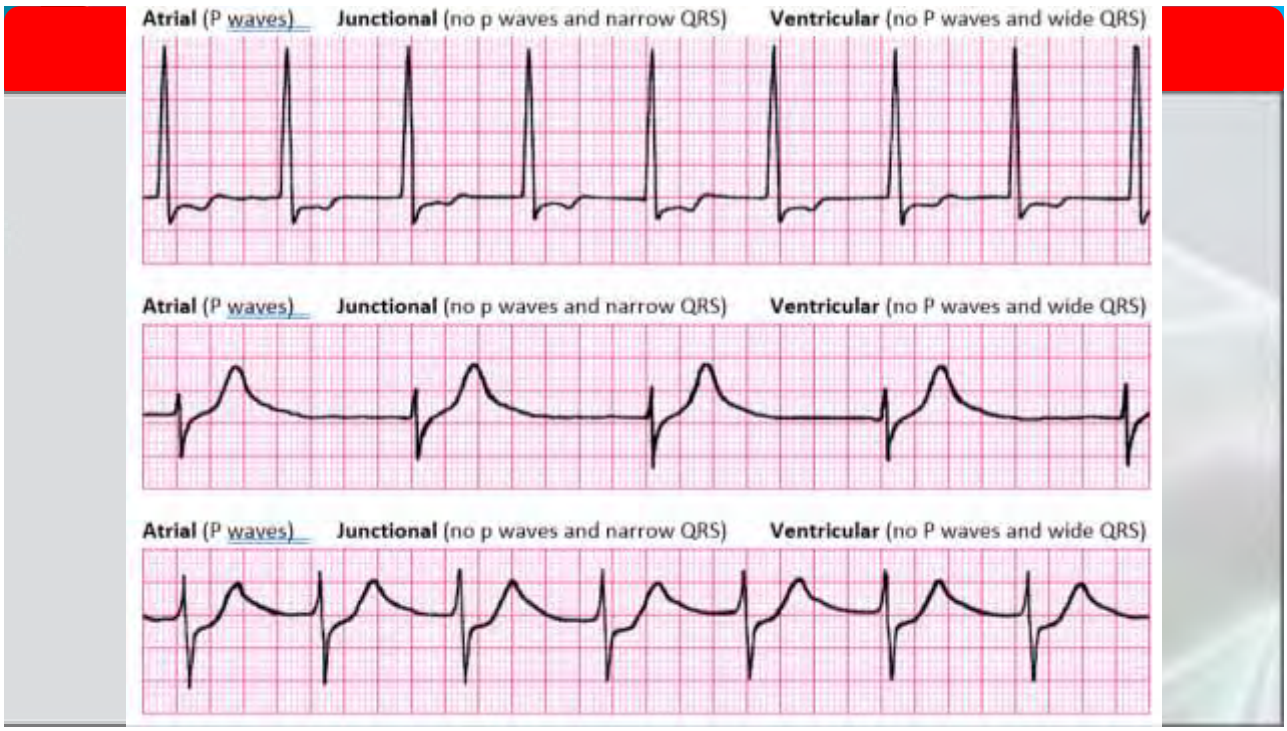
10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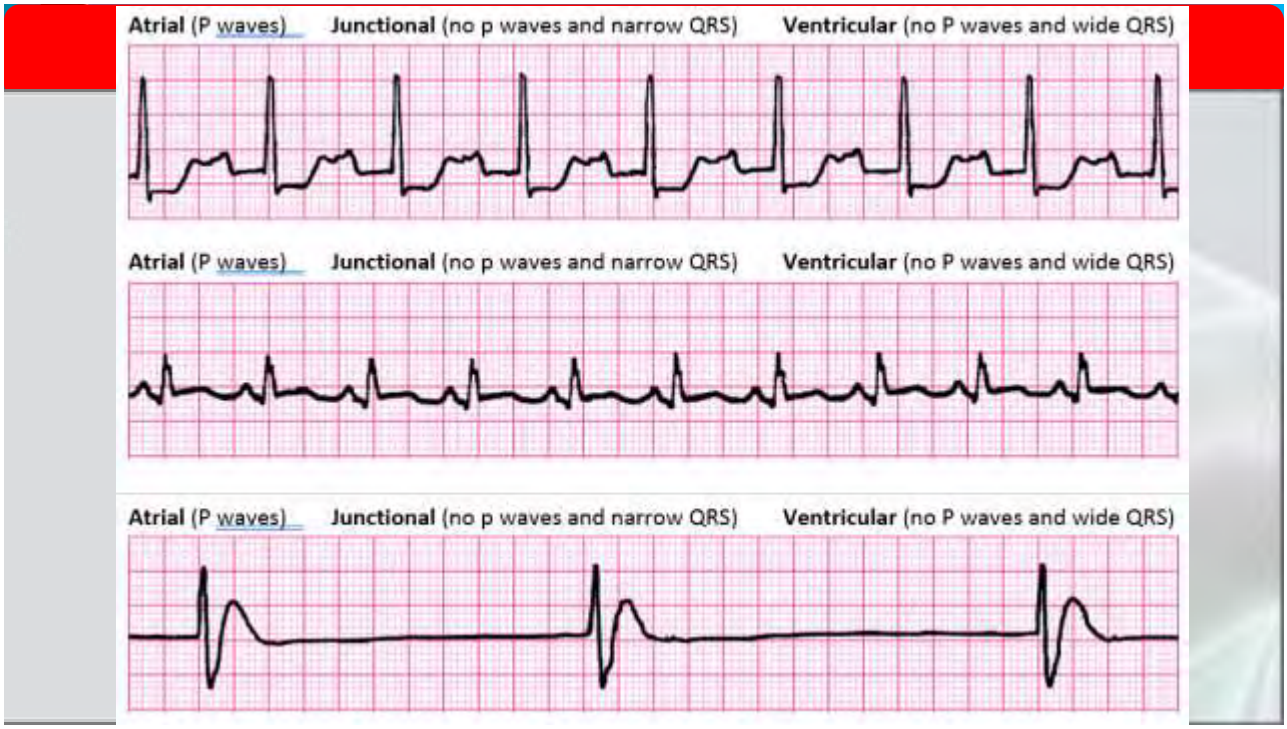
11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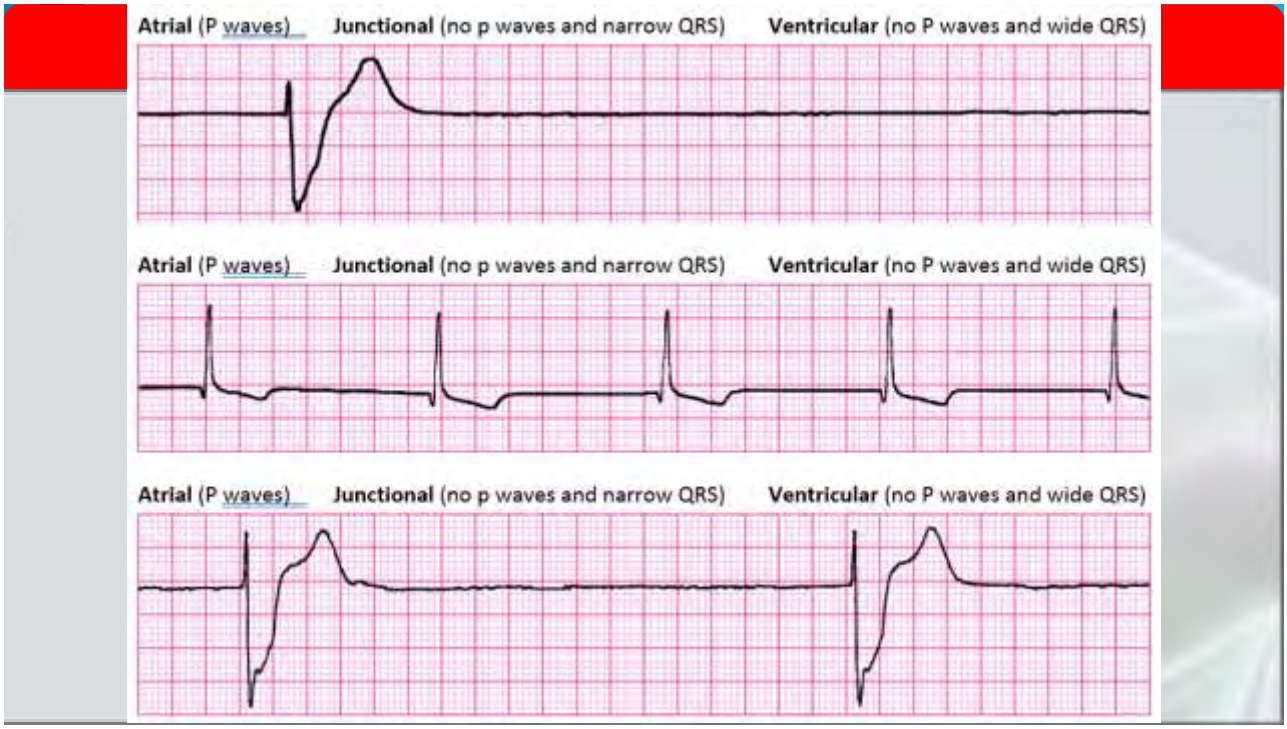
15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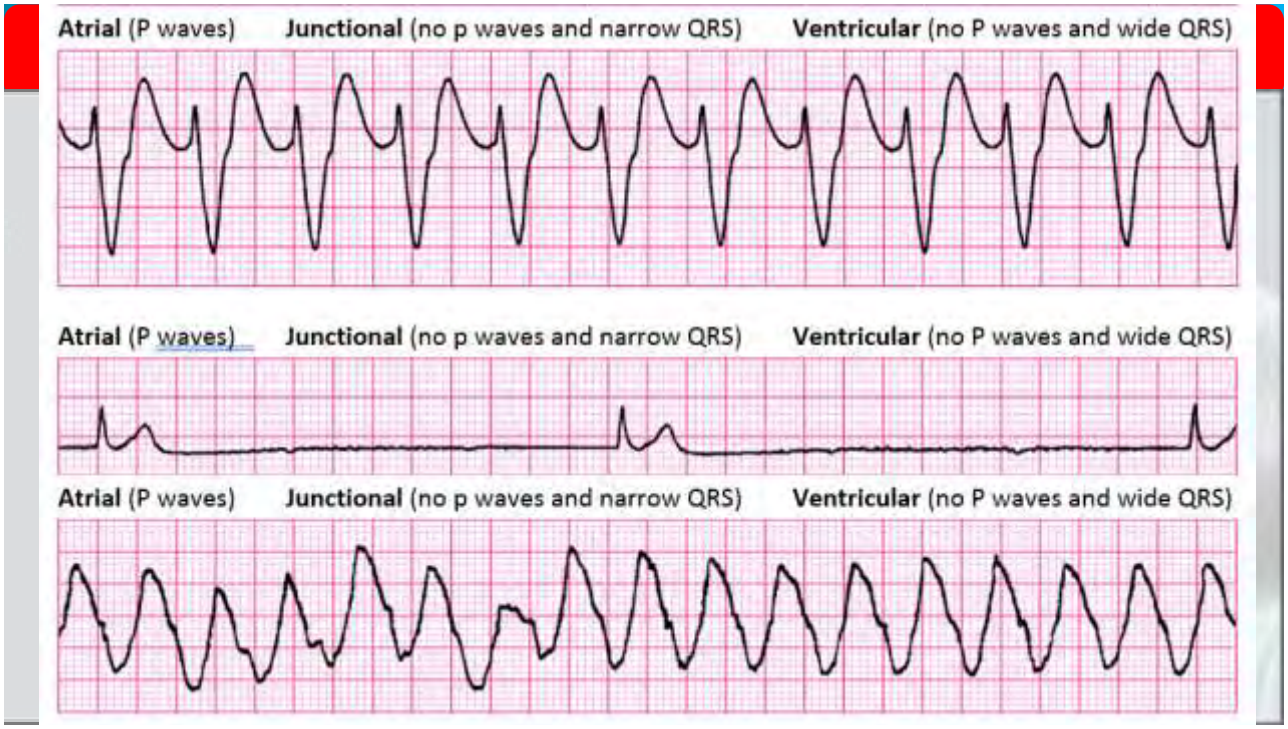
16



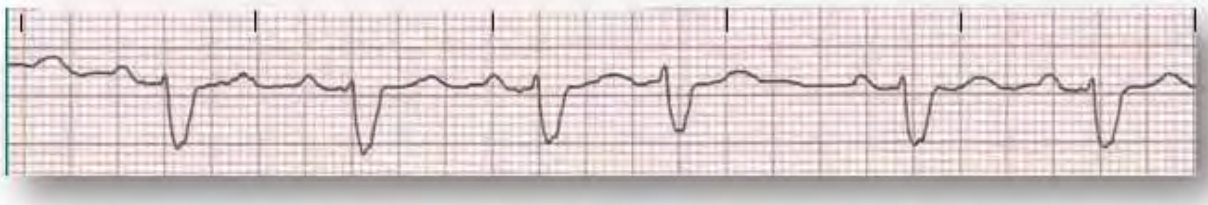
17

Premature atrial contraction

Atrial Rhythms:

PAC

Cause: irritability, atrial enlargement, caffeine, stress
Causative Medications inhalers,
Treatment: none



Atrial rate	any	**Rhythm:**	any – early beat causes irregularity
Ventricular Rate	any	**Rhythm:**	any – early beat causes irregularity
P waves:	upright, one to one, - **early beat may look different**		
PR Interval:	normal or long		
QRS Complex:	any		

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Premature junctional contraction

PJC – (Premature Junctional Contraction)

Atrial rate	any	Rhythm:	any – early beat causes irregularity
Ventricular Rate	any	Rhythm:	any – early beat causes irregularity
P waves:	upright, one to one, - early beat has junctional P wave formation		
PR Interval:	normal or long- early beat has junctional PR interval formation		
QRS Complex:	any		

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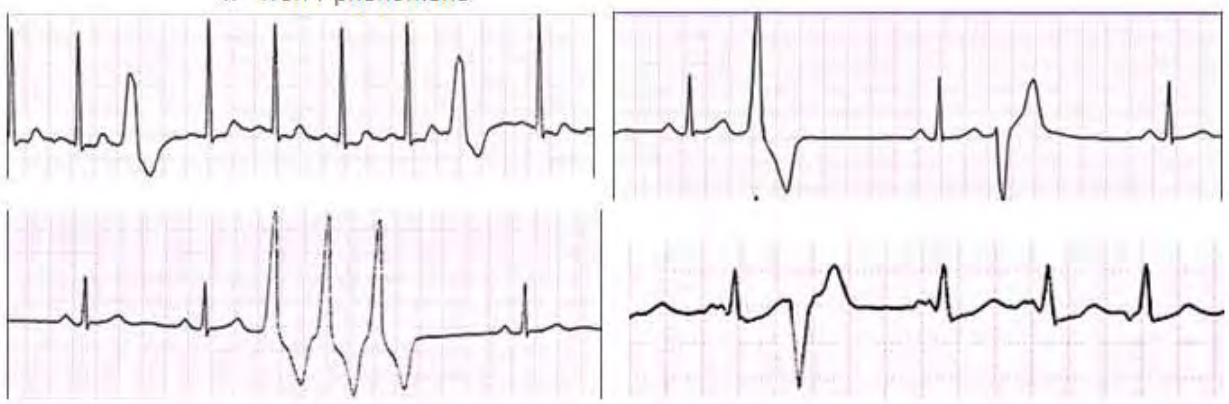
Premature ventricular contraction

PVC - (Premature Ventricular Contraction)

Reportable types:

1. Frequent (more than 6 per minute)
2. Multifocal (more than one shape of PVC = more than one focus)
3. Occurring in Pairs or Chains
4. R on T phenomena



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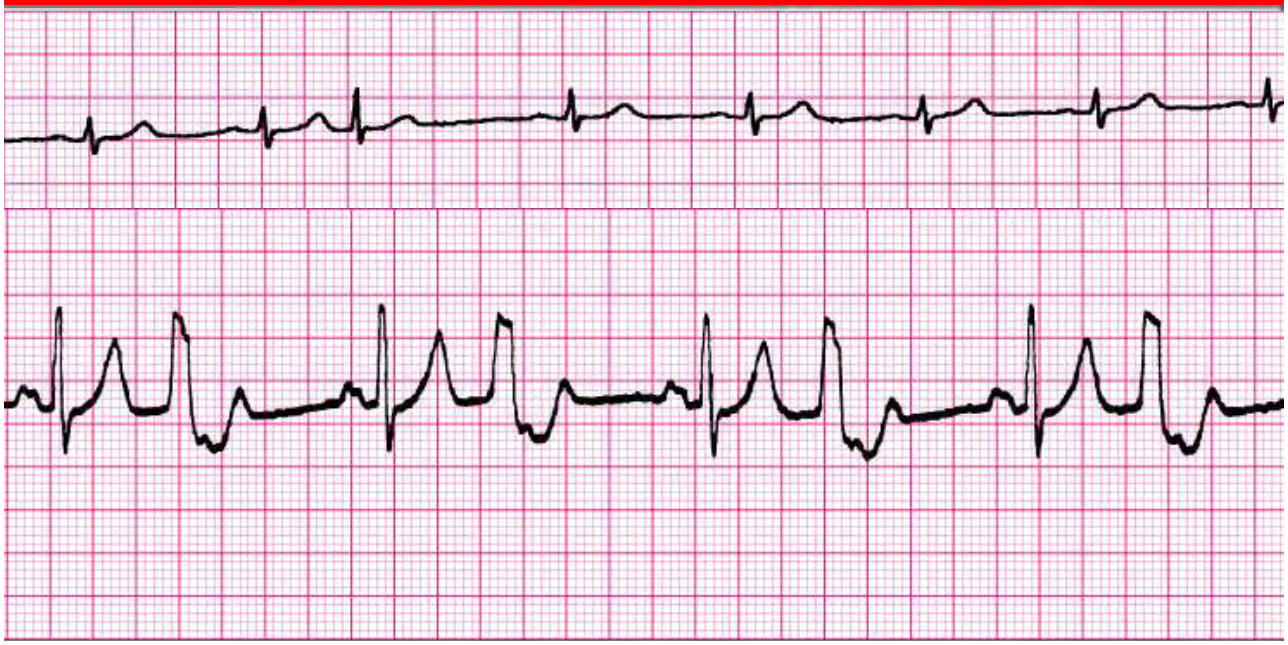
Premature Contractions



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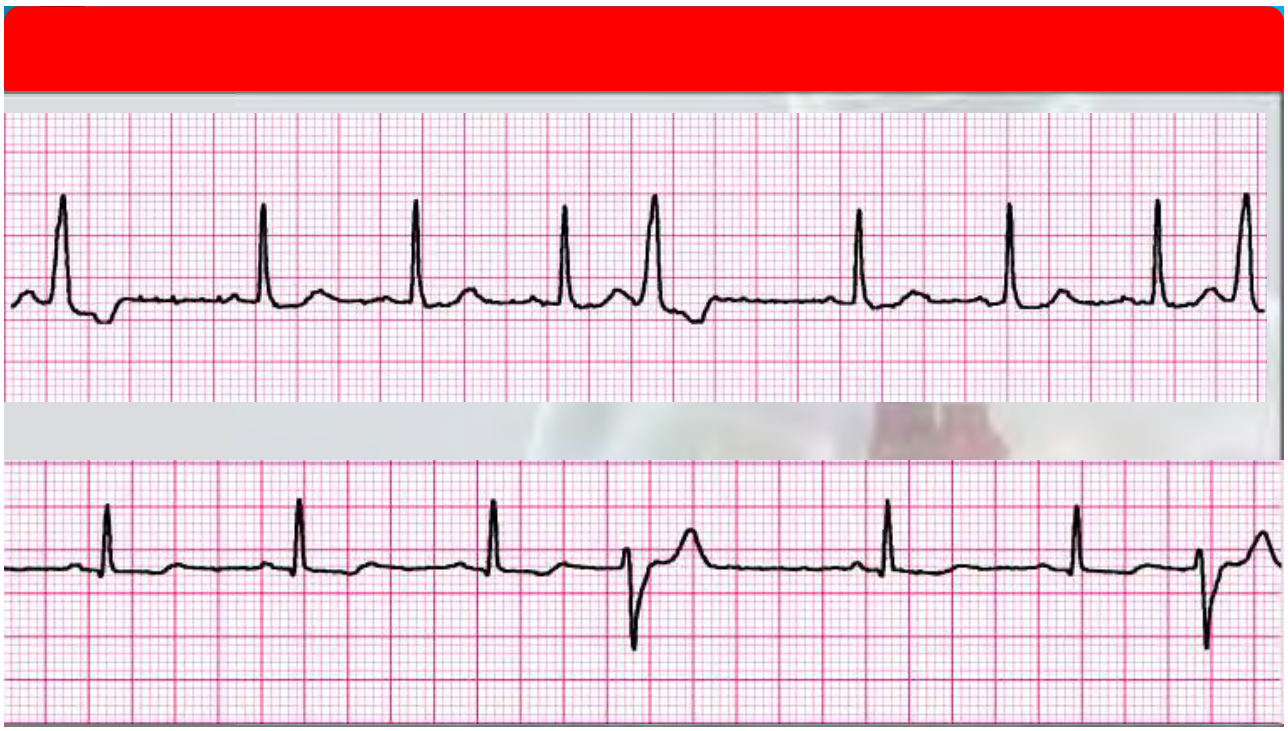

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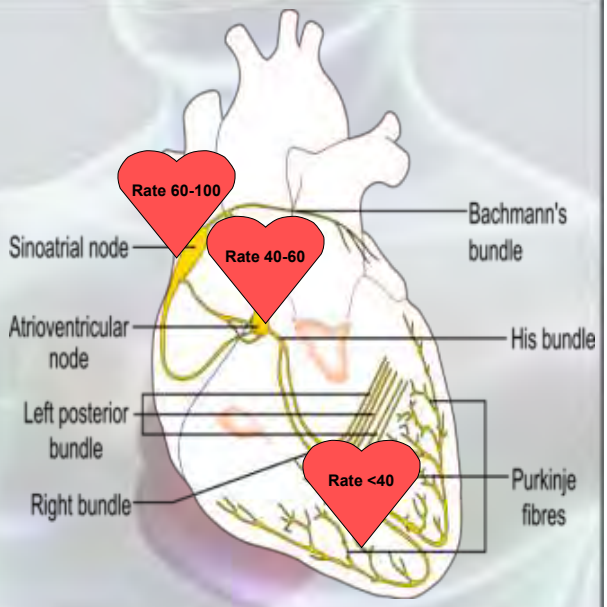
Rate Related



38

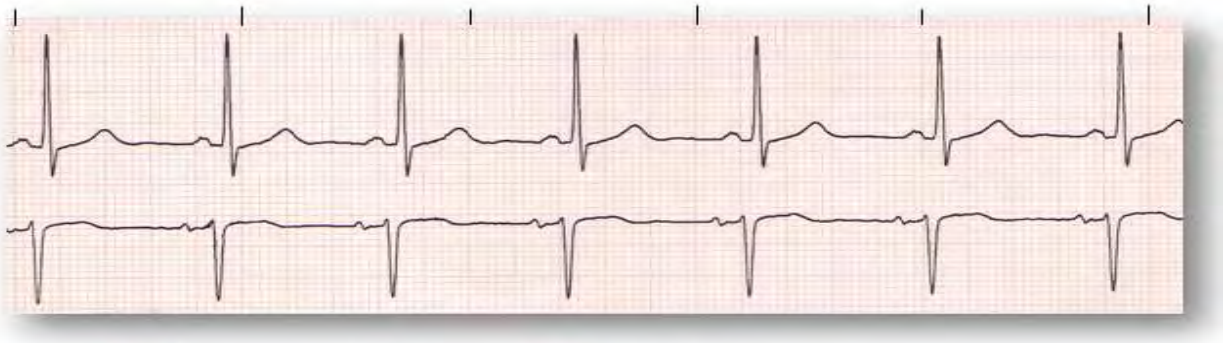
Sinus Rhythm

Rhythms:

Sinus Rhythm:

Cause: *Normal Rhythm*

Treatment: *None needed*



Atrial rate	60-100	**Rhythm:**	Regular
Ventricular Rate	60-100	**Rhythm:**	Regular
P waves:	upright, one to one, and all look the same		
PR Interval:	0.12-0.20 milliseconds in length *(3-5 small blocks)*		
QRS Complex:	less than 0.12 milliseconds *(less than 3 small blocks)*		

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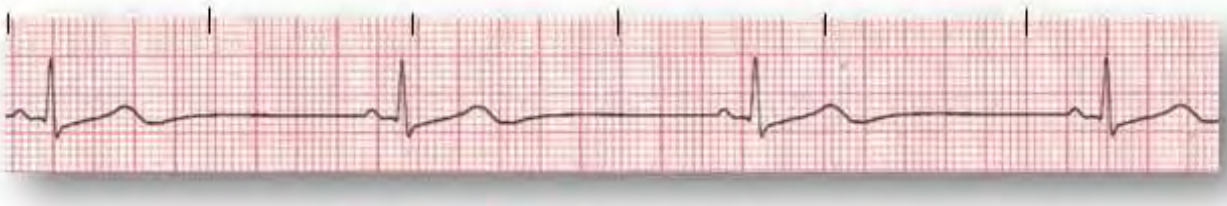
Sinus Brady

Sinus Brady

Cause: normal for athletes *- PNS (Vagal response, bearing down, straining) Hypoxia, age related degeneration* ***Causative Medications*** *(BBlkrs, CaChBlkrs, antiarrhythmics or Digoxin)*
Treatment: assess tolerance *- 0.5 mg Atropine OR, 2-10 mcg/kg Adrenaline infusion, pacing*



Atrial rate	**less than 60**	**Rhythm:**	Regular or irregular
Ventricular Rate	**less than 60**	**Rhythm:**	Regular or irregular
P waves:	upright, one to one, and all look the same		
PR Interval:	0.12-0.20 milliseconds in length *(3-5 small blocks)*		
QRS Complex:	less than 0 .12 milliseconds *(less than 3 small blocks)*		

40

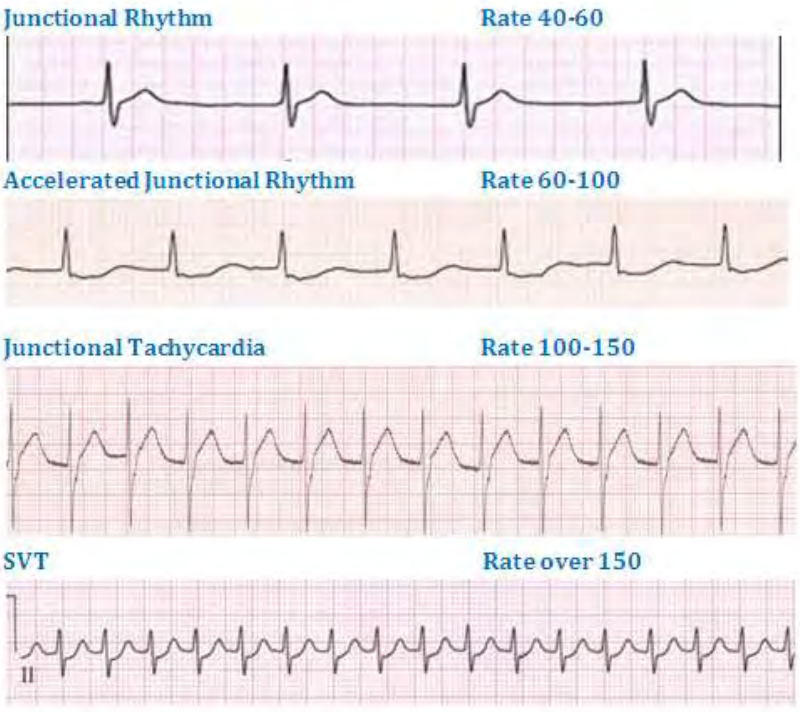
Sinus Tachycardia

Sinus Tachycardia

Cause: SNS (stress response), fever, pain, low cardiac output, hypoxia, inhalers, adrenaline
Treatment: none – Identify cause and resolve

Atrial rate	**100-150**	**Rhythm:**	Regular or irregular
Ventricular Rate	**100-150**	**Rhythm:**	Regular or irregular
P waves:	upright, one to one, and all look the same		
PR Interval:	0.12-0.20 milliseconds in length *(3-5 small blocks)*		
QRS Complex:	less than 0.12 milliseconds *(less than 3 small blocks)*		

41



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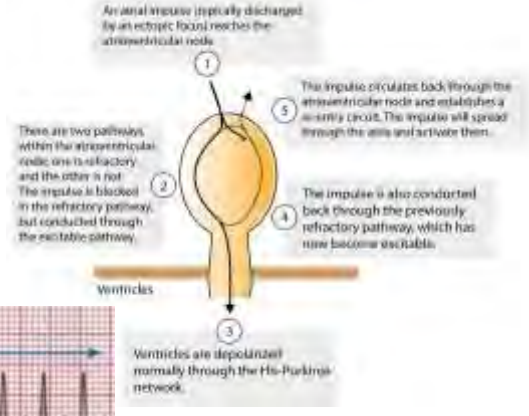
SVT – Supraventricular Tachycardia

- Re-entry mechanism
- Sudden onset and resolution
- Narrow QRS
- Rate >150

Treatment
Vagals
Adenosine
Cardiovert if unstable



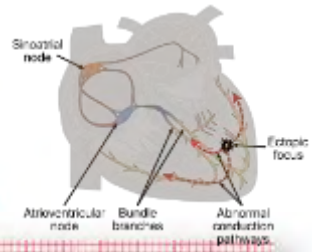
43

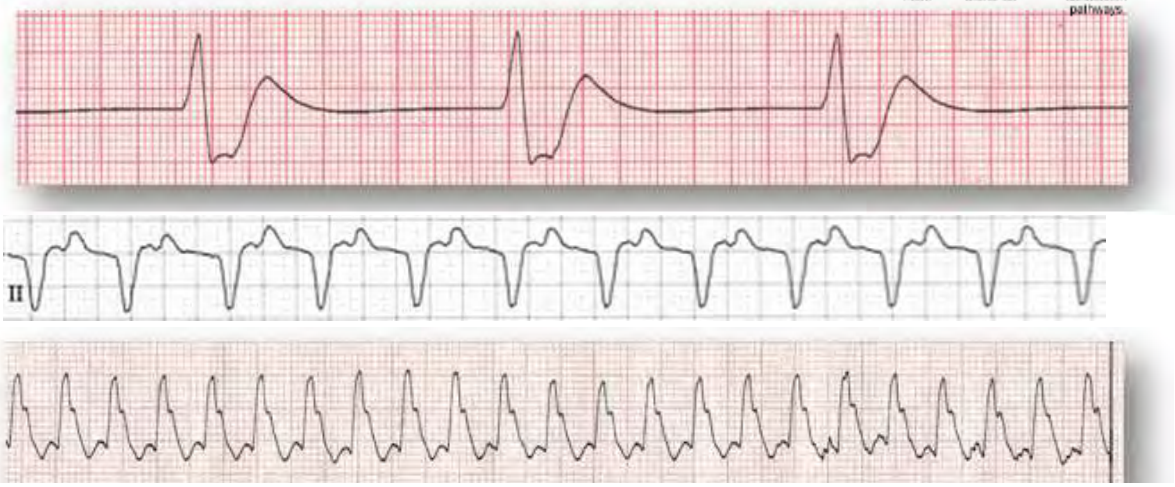
IVR – Idioventricular Rhythm

Atrial rate	none	Rhythm:	NA
Ventricular Rate	less than 40	Rhythm:	Regular
P waves:	none		
PR Interval:	none		
QRS Complex:	greater than 0.12 milliseconds *(3 small blocks)*		

Rate less than 40 **IVR – Idioventricular Rhythm**

Rate 40-100 **AIVR – Accelerated Idioventricular Rhythm**





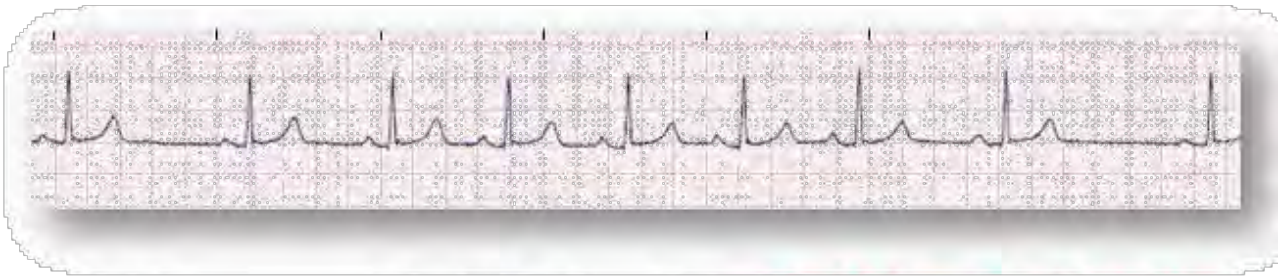
44

Sinus Arrhythmia

Sinus Arrhythmia

Cause: increased venous return to the heart on inspiration

Treatment: none



Atrial rate	nml tachy or brady	Rhythm:	**Regularly Irregular with breathing**
Ventricular Rate	nml tachy or brady	Rhythm:	**Regularly Irregular with breathing**
P waves:	upright, one to one, and all look the same		
PR Interval:	0.12-0.20 milliseconds in length *(3-5 small blocks)*		
QRS Complex:	less than 0.12 milliseconds *(less than 3 small blocks)*		

45

Atrial fibrillation

Atrial Fibrillation

Cause: *irritability, atrial enlargement, Heart failure*
Causative Medications *: Digoxin toxicity*
Treatment: *if less than 48 hours – convert with meds or cardioversion. If more than 48 hours just slow ventricular rate with BBlkrs, CaChBlkrs, antiarrhythmics or Digoxin*

Atrial rate	any	**Rhythm:**	**Irregularly Irregular**
Ventricular Rate	any	**Rhythm:**	**Irregularly Irregular**
P waves:	none – quivery baseline is atrial activity		
PR Interval:	none		
QRS Complex:	any – normal or wide		

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Atrial flutter

Atrial Flutter

Cause: *irritability, atrial enlargement, Heart failure*
Causative Medications *: Digoxin toxicity*
Treatment: *chemical or electrical cardioversion. Digixin, Amiodarone, BBlkrs, CaChblkrs*

Atrial rate	over 250	**Rhythm:**	Regular
Ventricular Rate	over 250	**Rhythm:**	Regular or irregular
P waves:	**replaced with saw-toothed flutter waves**		
PR Interval:	none		
QRS Complex:	any – normal or wide		

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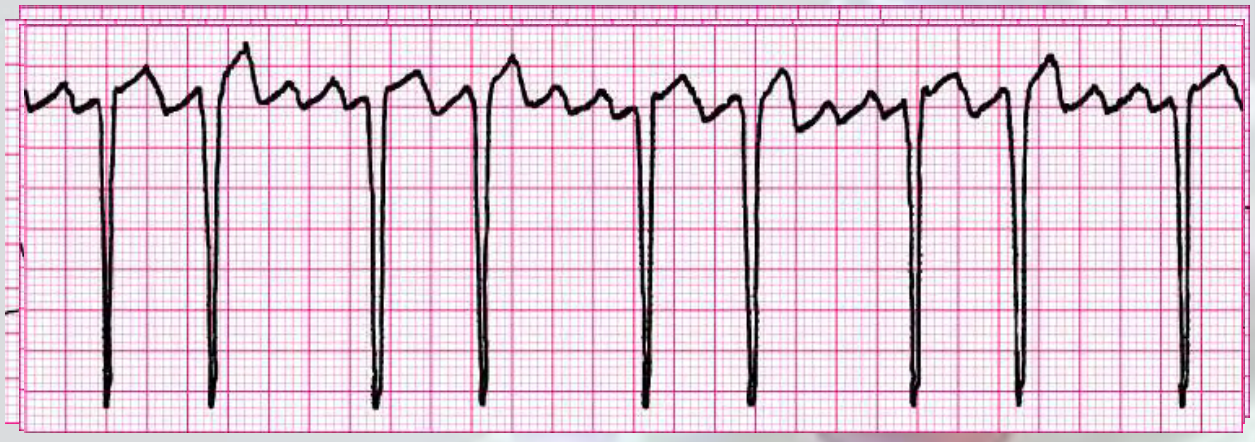
Irregular rhythm check

Not in Handout – View Screen



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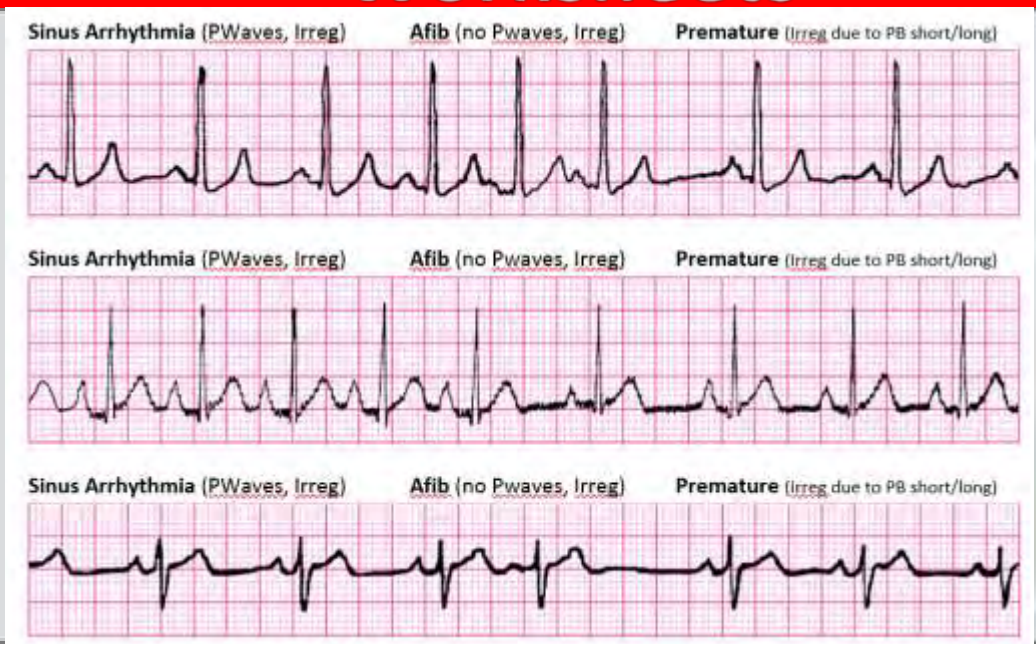
Worksheets



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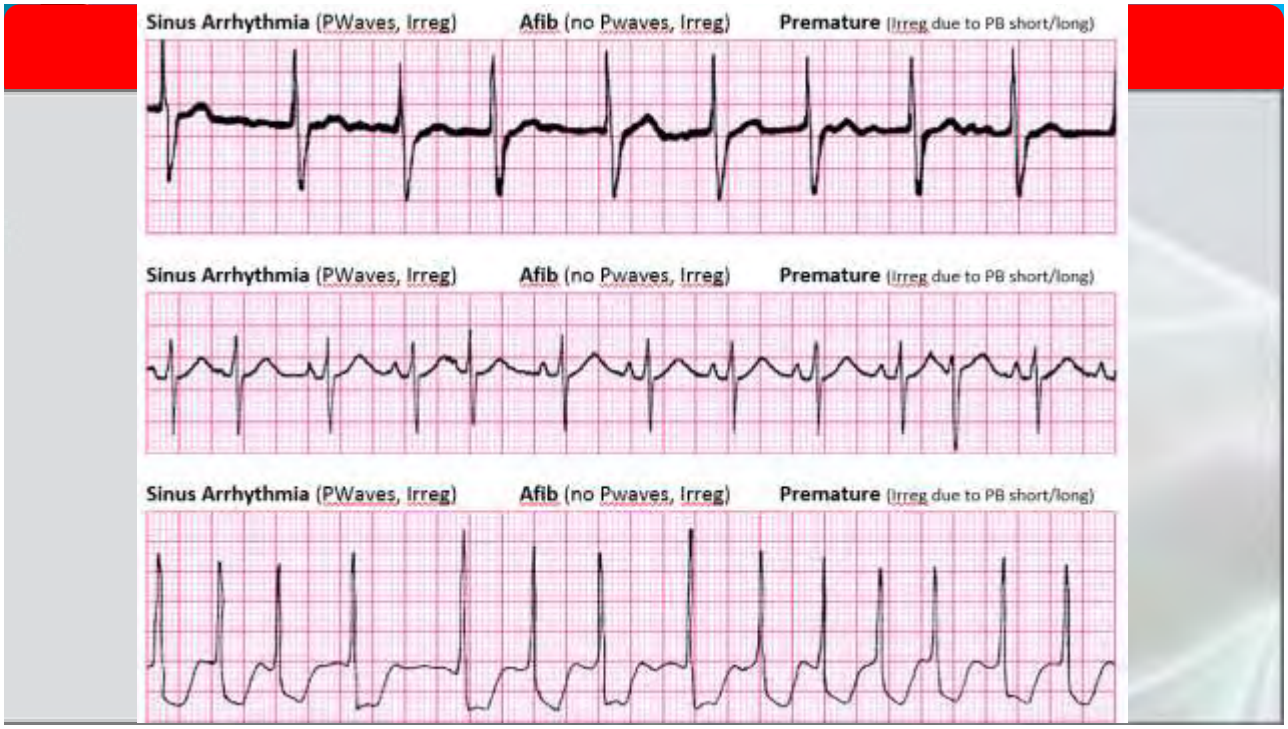
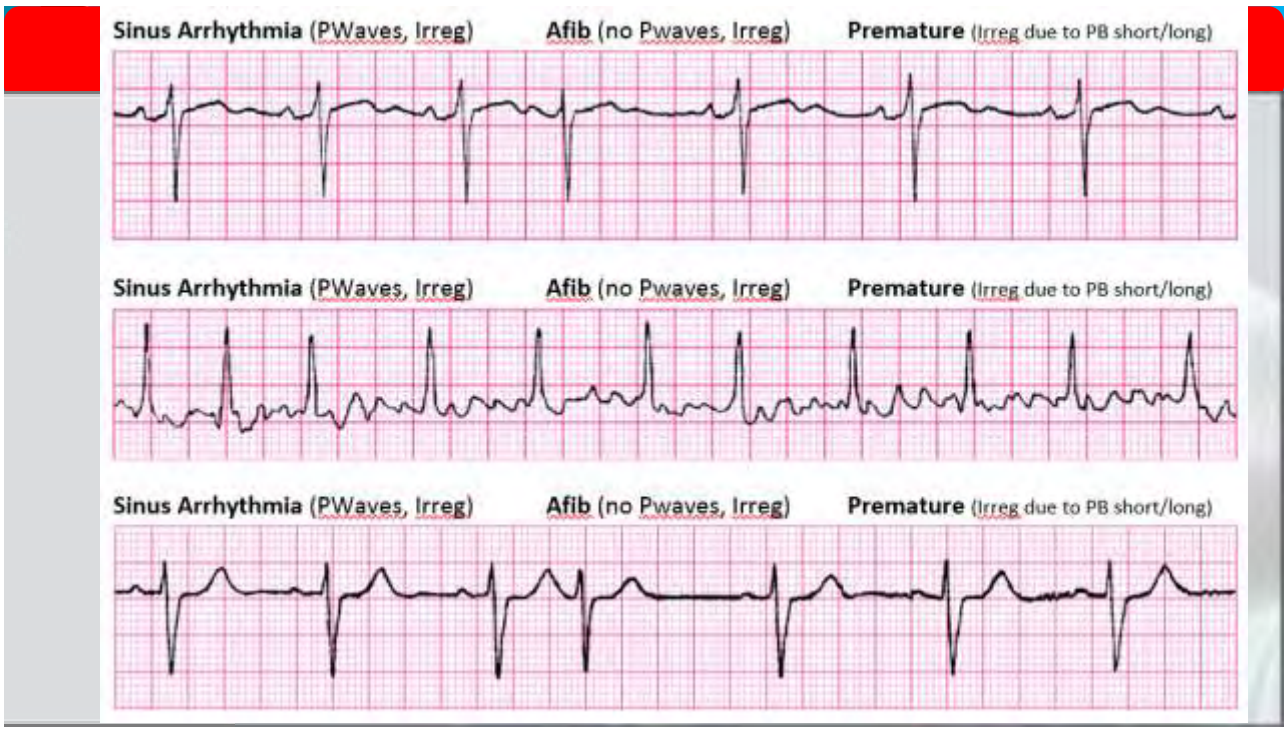

Sinus Arrhythmia (PWaves, Irreg)
Afib (no Pwaves, Irreg)
Premature (Irreg due to PB short/long)
Sinus Arrhythmia (PWaves, Irreg)
Afib (no Pwaves, Irreg)
Premature (Irreg due to PB short/long)
Sinus Arrhythmia (PWaves, Irreg)
Afib (no Pwaves, Irreg)
Premature (Irreg due to PB short/long)

52


Sinus Arrhythmia (PWaves, Irreg)
Afib (no Pwaves, Irreg)
Premature (Irreg due to PB short/long)
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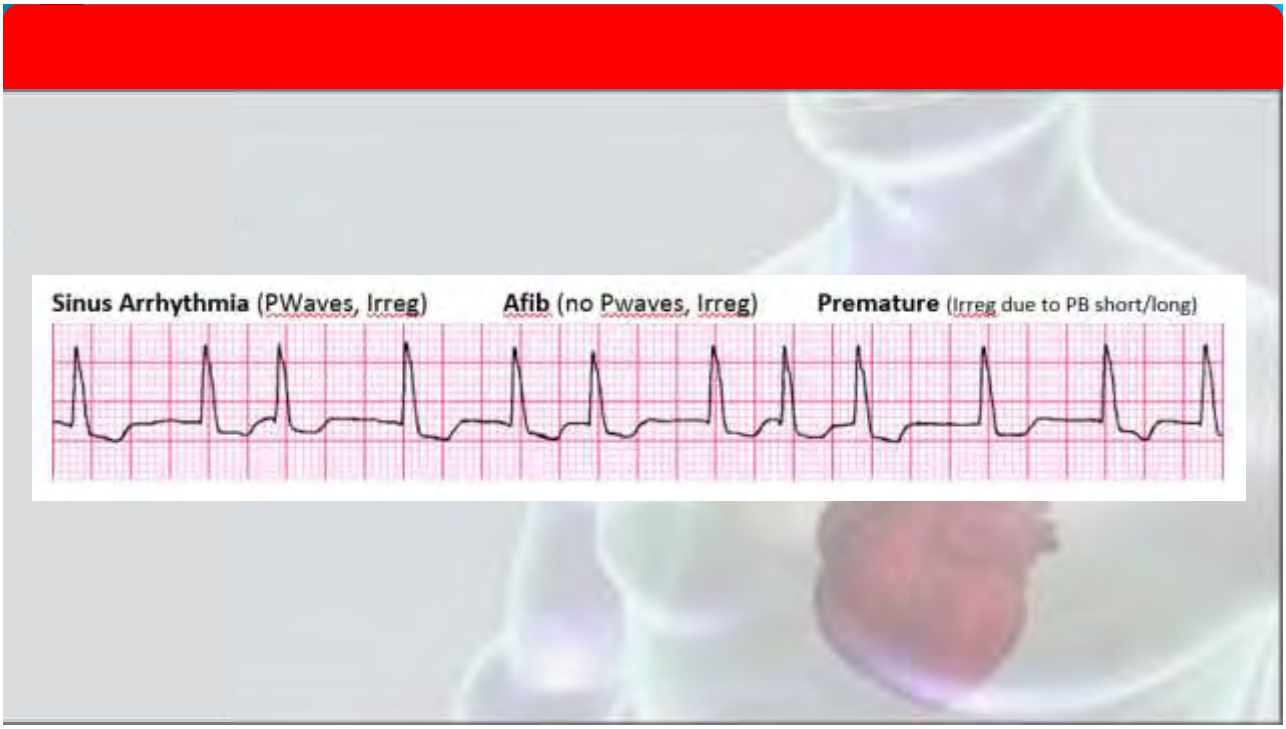
Sinus Arrhythmia (PWaves, Irreg)
Afib (no Pwaves, Irreg)
Premature (Irreg due to PB short/long)

53


Sinus Arrhythmia (PWaves, Irreg)
Afib (no Pwaves, Irreg)
Premature (Irreg due to PB short/long)

54


Disorders of Conduction
DETOUR
ROAD CLOSED
DETOUR
DETOURS
DETOUR

55

First Degree AV Block

First Degree AVBlock

Cause: PNS (vagal, straining), age- related degeneration,
Causative Medications (BBlkrs, CaChBlkrs, Dig)
Treatment: none – watch for advancing block

Atrial rate	nml tachy or brady	**Rhythm:**	Regular or irregular
Ventricular Rate	nml tachy or brady	**Rhythm:**	Regular or irregular
P waves:	upright, one to one, and all look the same		
PR Interval:	**greater than 0.20 milliseconds in length (3-5 small blocks)**		

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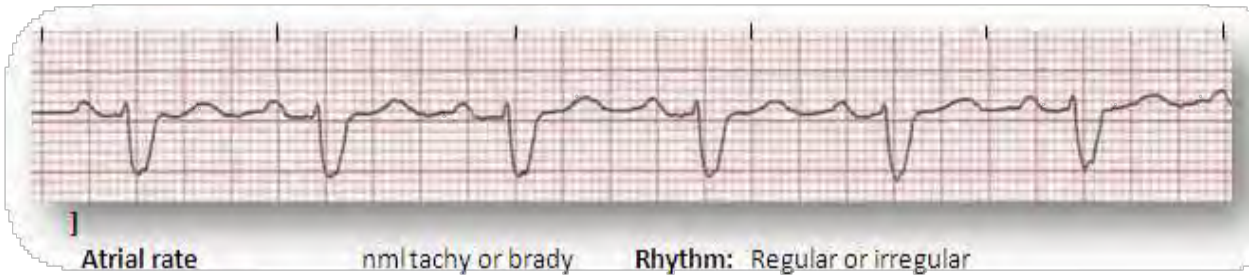
Bundle Branch Block

Bundle Branch Block

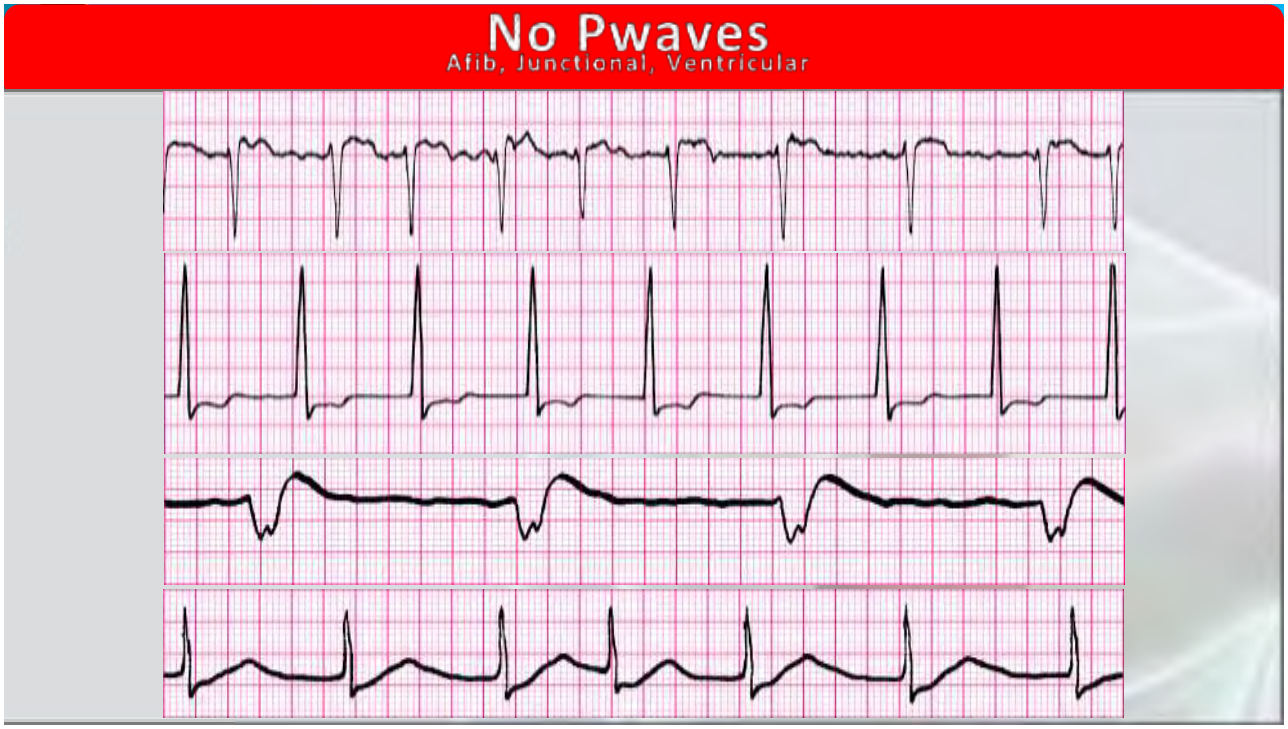
Cause: PNS (vagal, straining), age- related degeneration, MI
Causative Medications (BBlkrs, CaChBlkrs, Dig)
Treatment: none – watch for advancing block



]

Atrial rate	nml tachy or brady	**Rhythm:**	Regular or irregular
Ventricular Rate	nml tachy or brady	**Rhythm:**	Regular or irregular
P waves:	upright, one to one, and all look the same		
PR Interval:	normal, short or long		
QRS Complex:	**greater than 0.12 milliseconds (less than 3 small blocks)**		

57



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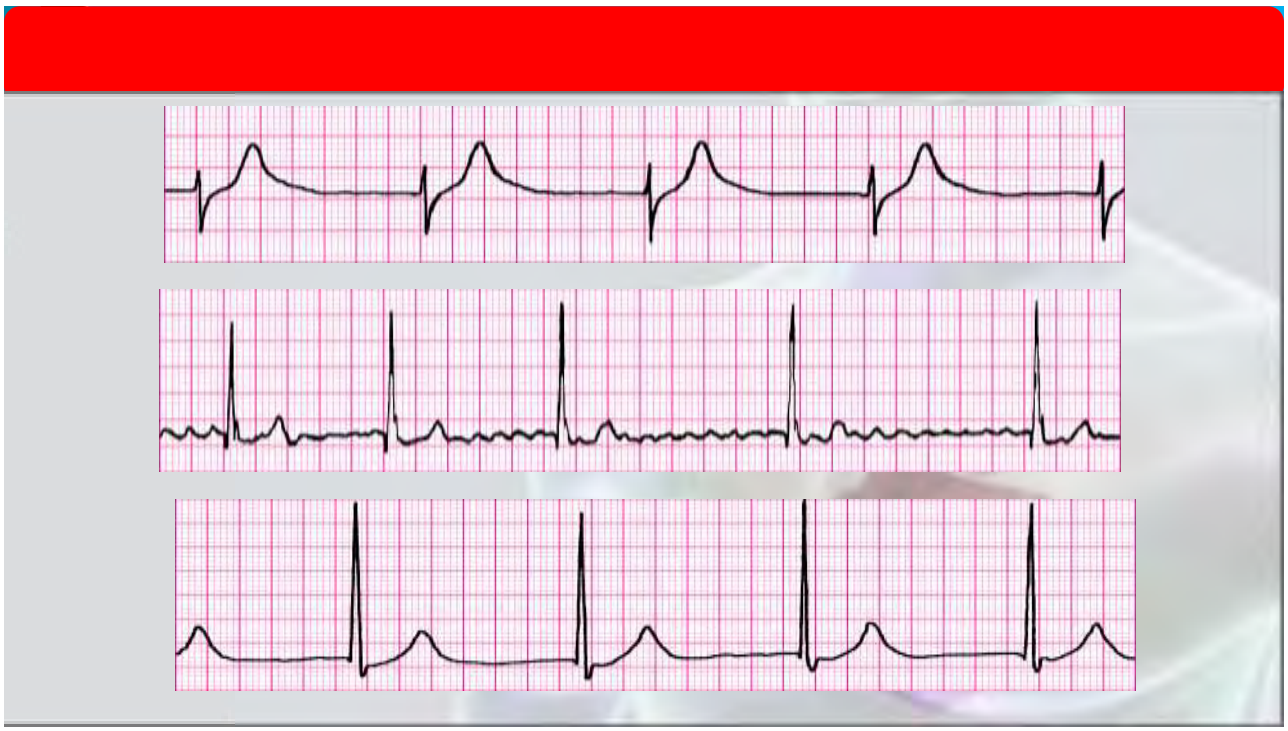

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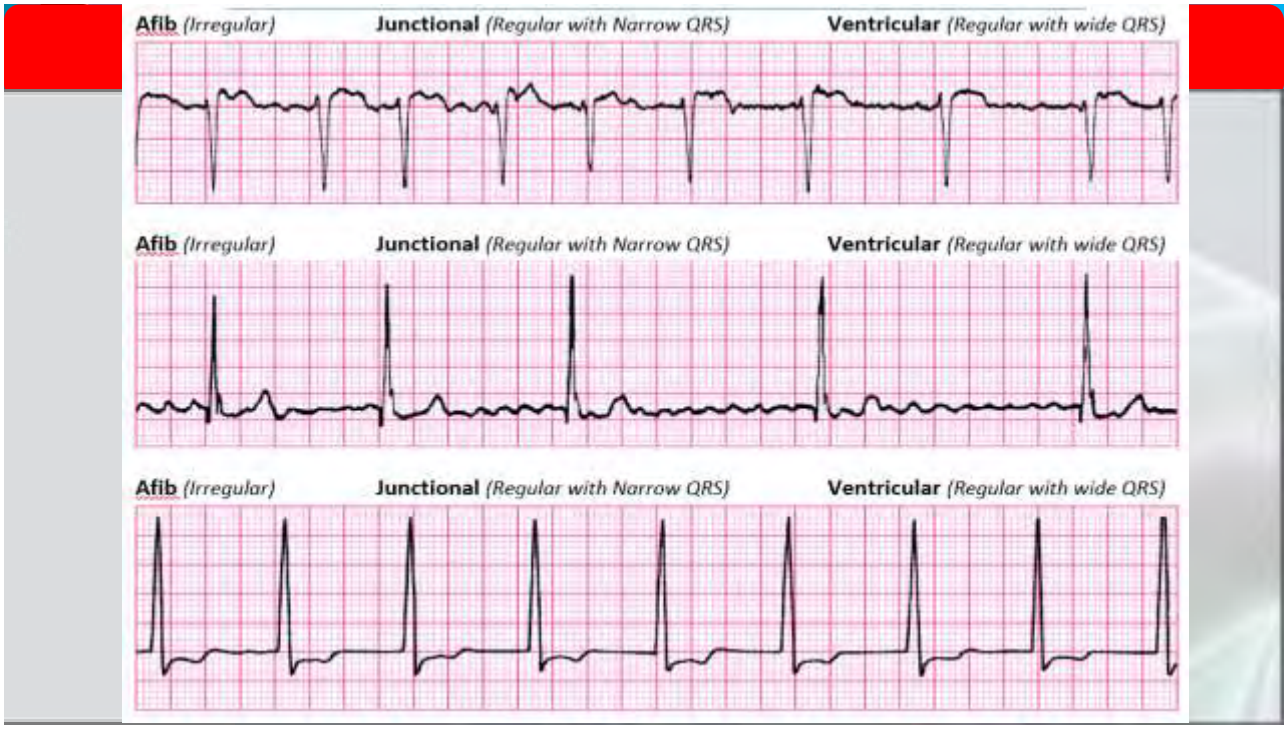
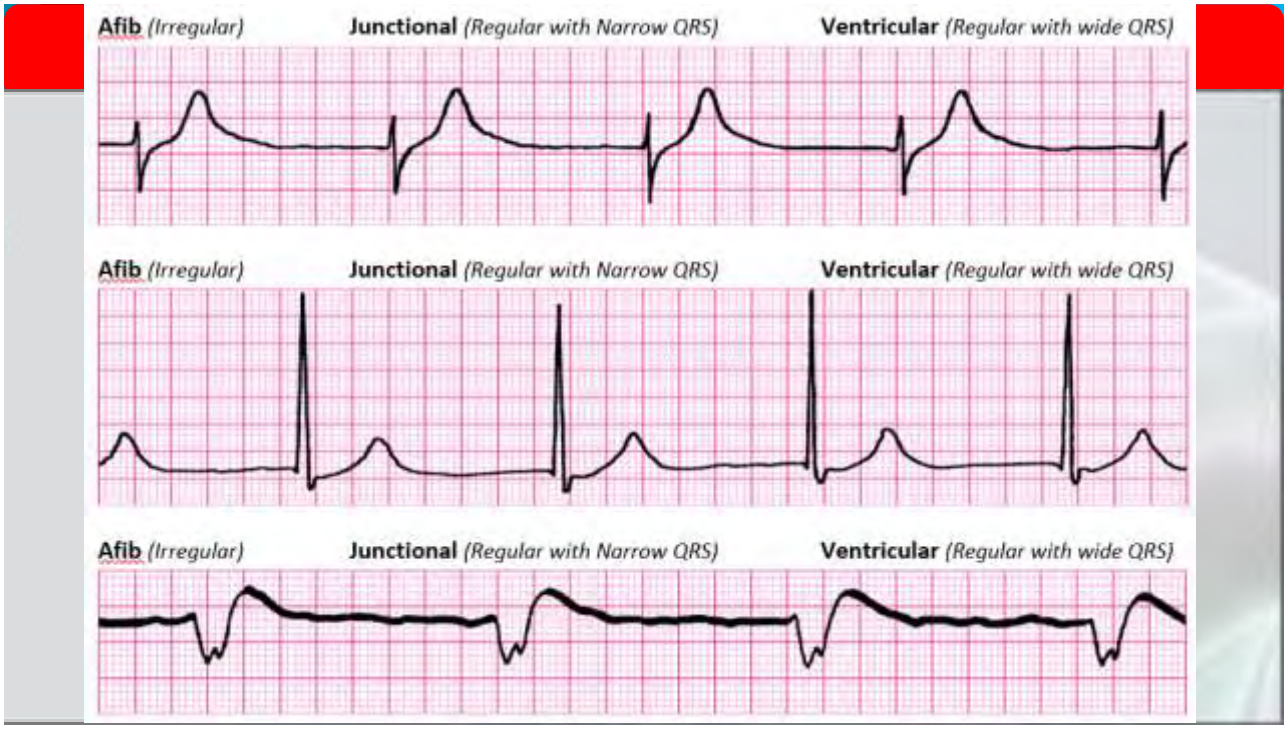
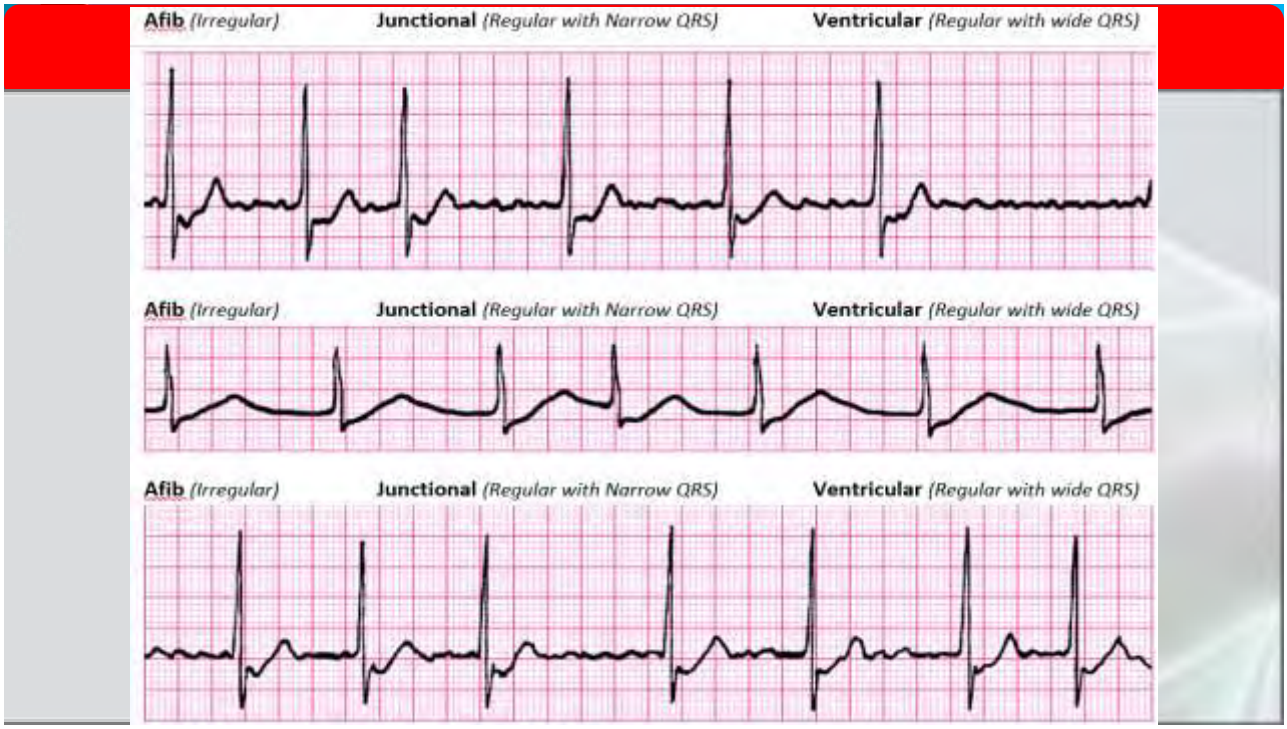

Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)
Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)
Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)

87


Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)
Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)
Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)

88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Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)
Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)
Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)

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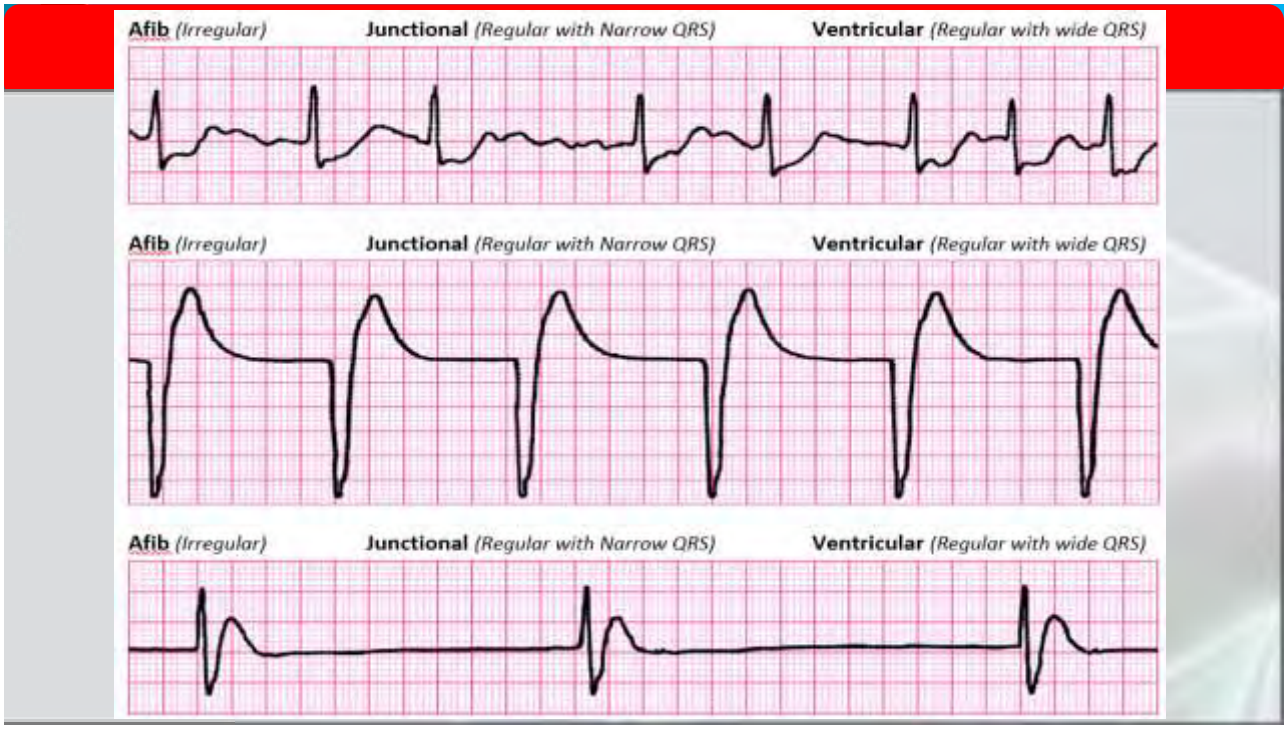
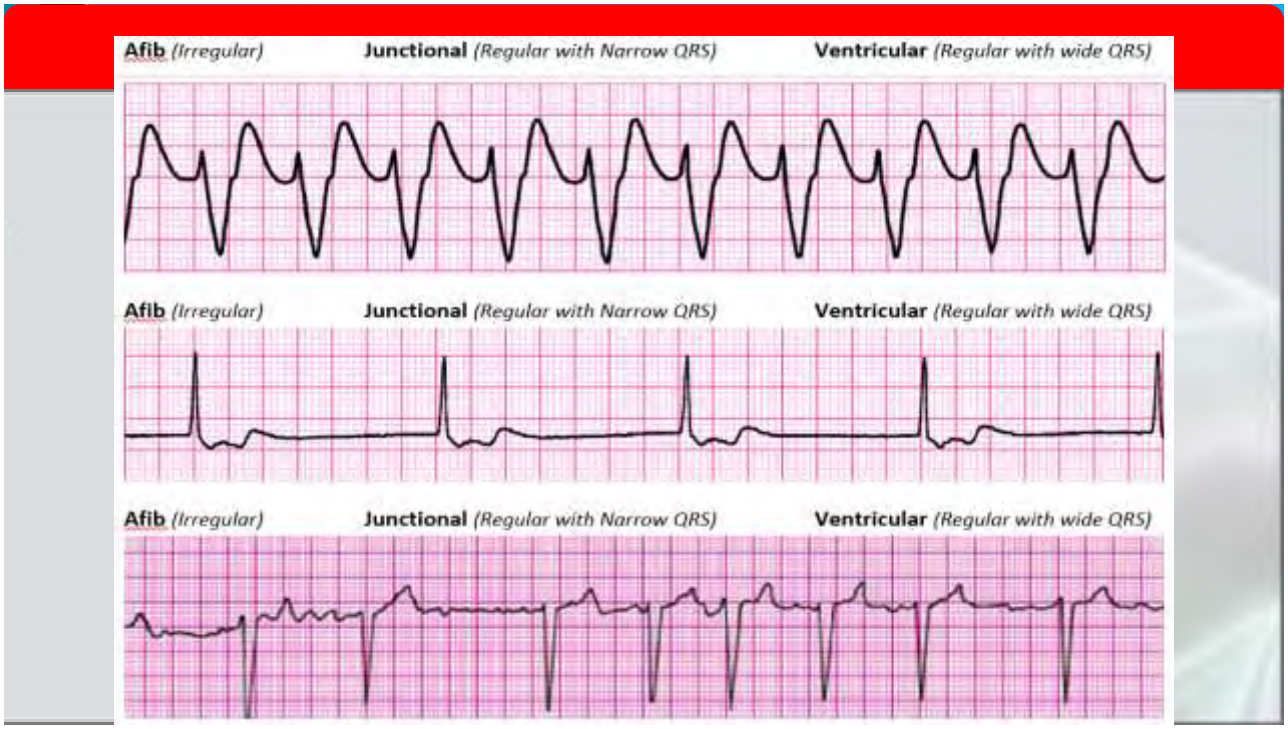

Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)
Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)
Afib (Irregular)
Junctional (Regular with Narrow QRS)
Ventricular (Regular with wide QRS)

90



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Ventricular fibrillation

VF – Ventricular Fibrillation

Cause: electrical shock, electrolyte imbalance, MI, R on T PVC, hypoxia etc
Causative Medications: anything that prolongs the QT interval, R on T PVC, hypokalemia
Treatment: CPR and immediate defibrillation, adrenaline and amiodarone

Atrial rate	none	Rhythm: NA
Ventricular Rate	none	Rhythm: chaos
P waves:	none	
PR Interval:	none	
QRS Complex:	none	

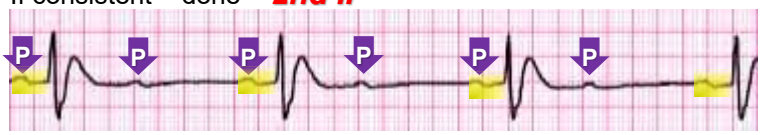
99

Advanced Heart Blocks

More Ps than QRS complexes and Ps are regular

1. **Is PR interval consistent or variable?**

   If consistent – done – ***2nd II***



2. **If variable – is the ventricular rhythm regular or irregular?**

   If irregular – ***2nd I***

   If regular – ***3rd***

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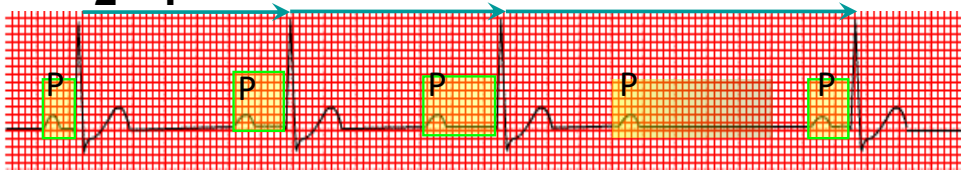
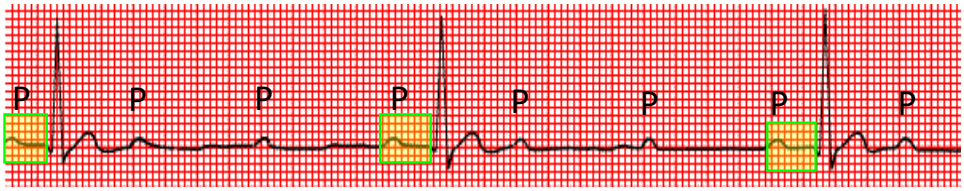
Review

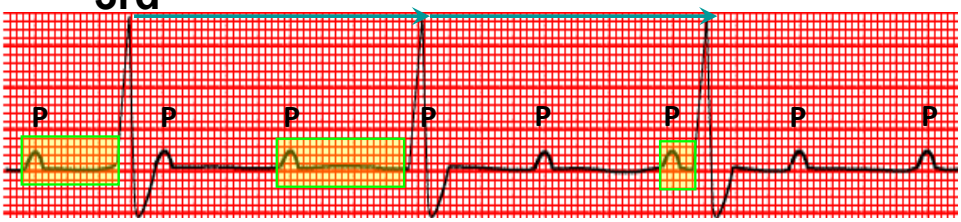
- **2nd I**



- **2nd II**



- **3rd**



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Heart Block Practice

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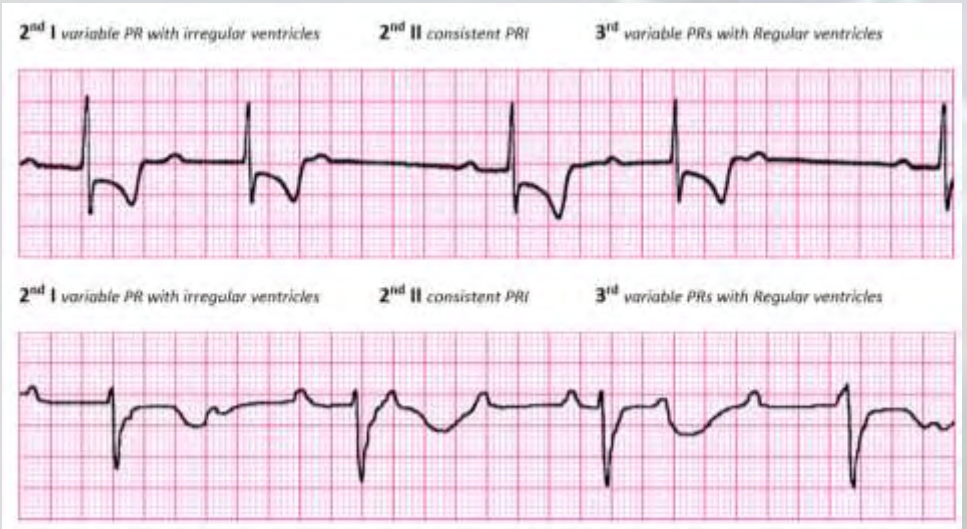
Worksheets



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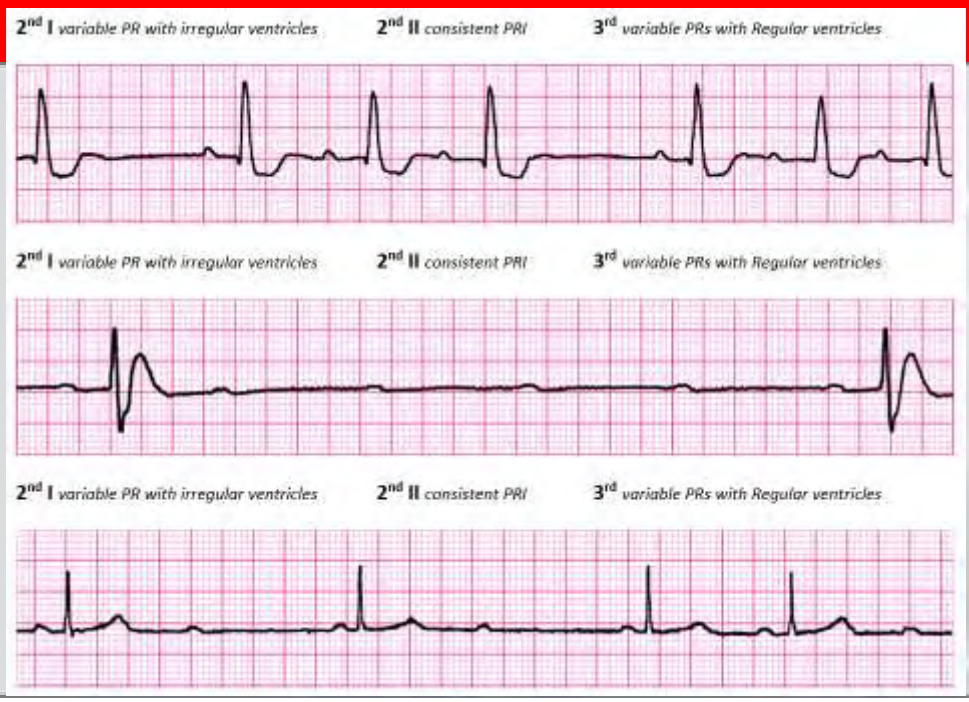
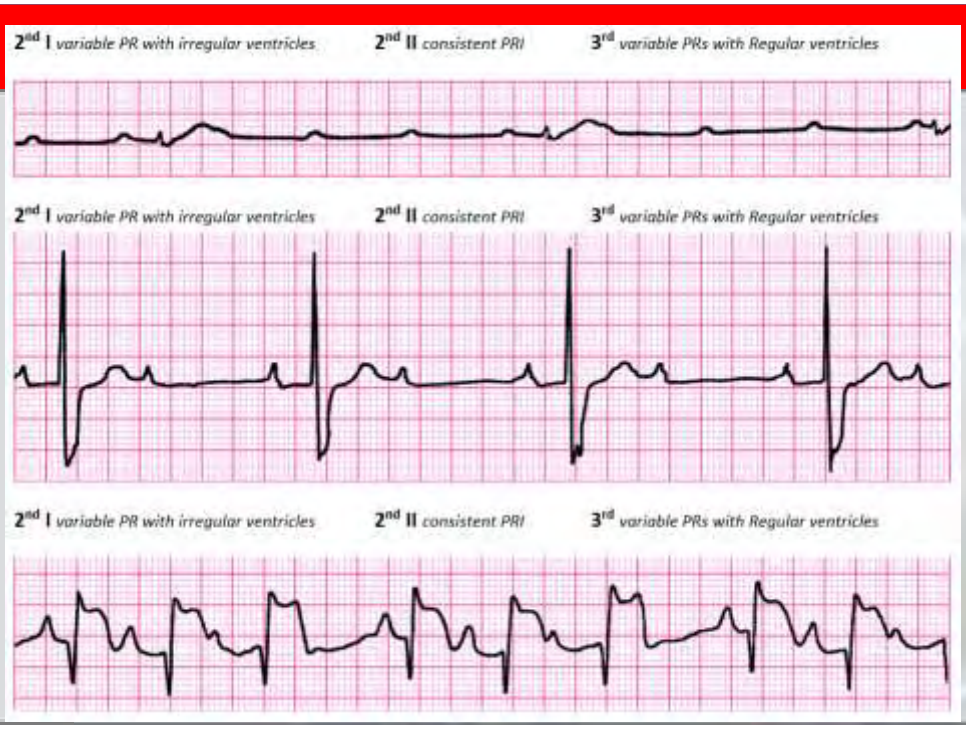
2nd I variable PR with irregular ventricles
2nd II consistent PRI
3rd variable PRs with Regular ventricles

107

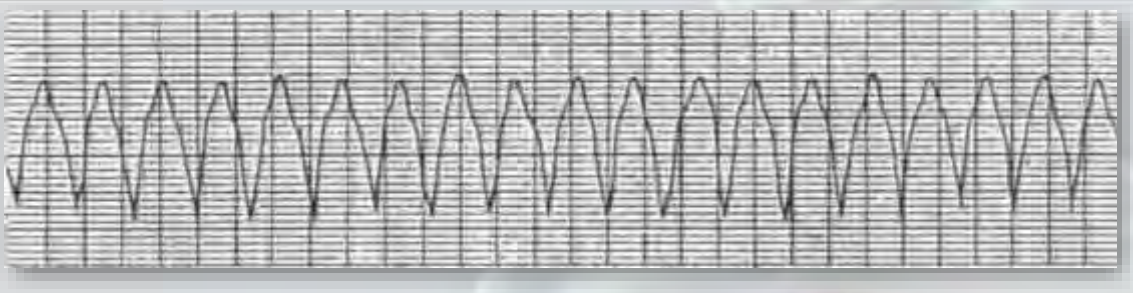

2nd I variable PR with irregular ventricles
2nd II consistent PRI
3rd variable PRs with Regular ventricles
2nd I variable PR with irregular ventricles
2nd II consistent PRI
3rd variable PRs with Regular ventricles
2nd I variable PR with irregular ventricles
2nd II consistent PRI
3rd variable PRs with Regular ventricles

108



109

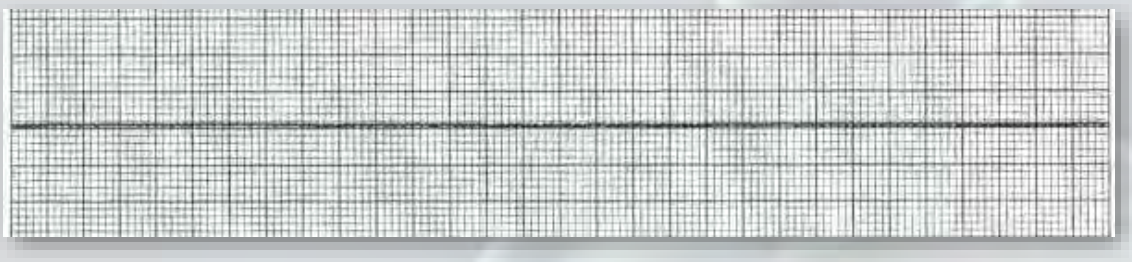
Question 1



24. While you are at the bedside, Mr. B goes into the rhythm above. He is alert, responsive, BP 130/80, and has a pulse. The MOST appropriate next step is to:

A. Check the carotid pulse for 45 seconds
B. Cardiovert with 100 joules monophasic or 50 joules biphasic
C. Administer 150 mg amiodarone IV STAT
D. Defibrillate with 200 joules monophasic, or 120 joules biphasic

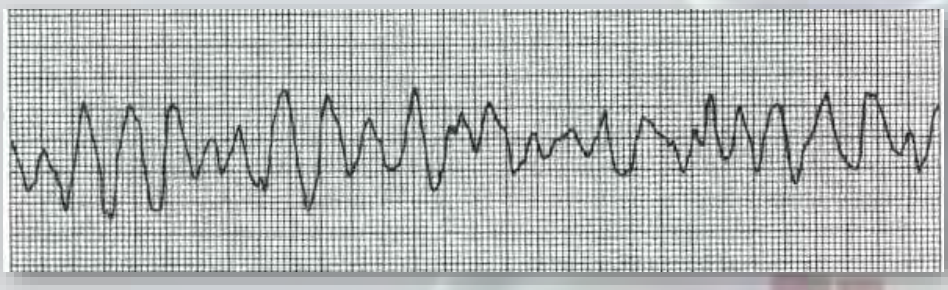
142



20. Your patient has the following rhythm and no pulse. You should:

A. Call a code and begin CPR
B. Defibrillate the patient
C. Cardiovert the patient
D. Call the family for a Do Not Resuscitate Order

143



27. Your patient is unconscious and pulseless. His monitor shows the rhythm above. Considering all are **IMMEDIATELY** available, what should be done next?

A. Begin CPR 5:2
B. Administer electrical countershock (defibrillation)
C. Immediately administer sodium bicarbonate and call a Code
D. Administer a lidocaine bolus

144


P waves Different
Wandering Pac
Rhythm Irregular
P waves same
Sinus arrhythmia
A, 1:1, same
< 60 Bradycardia
Regular
Sinus
60-100 Sinus Rhythm
100-150 Sinus Tach
> 150 SVT
Wide QRS with P wave Bundle Branch Block
Yes
P waves
None
vent reg
3rd degree
PRI variable
Vent Irreg
2nd type I
More than QRS
PR consistent
2nd type II
< 100 Controlled
A-fib
Rhythm Irregular
> 100 Rapid Vent Response
40-60 JR
60-100 AJR
< 12 Junctional
100-150 JT
> 150 SVT
QRS duration
Rhythm Regular
< 40 IVR
40-100 AIVR
> 12 Ventricular
> 100 VT

145

Cardiac Diagnostic Tests & Procedures

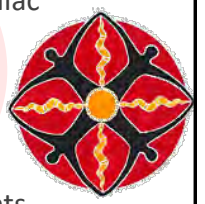
Modified 2/2019
by
Lynnette Flynn
MBA, CHFN, CCRN-CMC, RCIS
Education Team Leader
Arkansas Heart Hospital

1

Learning Objectives

By the end of this session you will be able to:

- List the indications for common cardiac diagnostic tools & procedures
- Review new procedures being performed in our region
- Describe the appropriate nursing management when caring for patients having different diagnostic procedures



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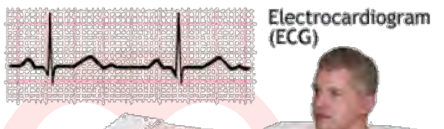
2

ECG

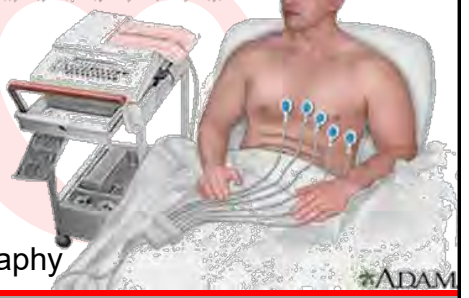
12 LEAD
STRESS TESTING
AMBULATORY ECG

3

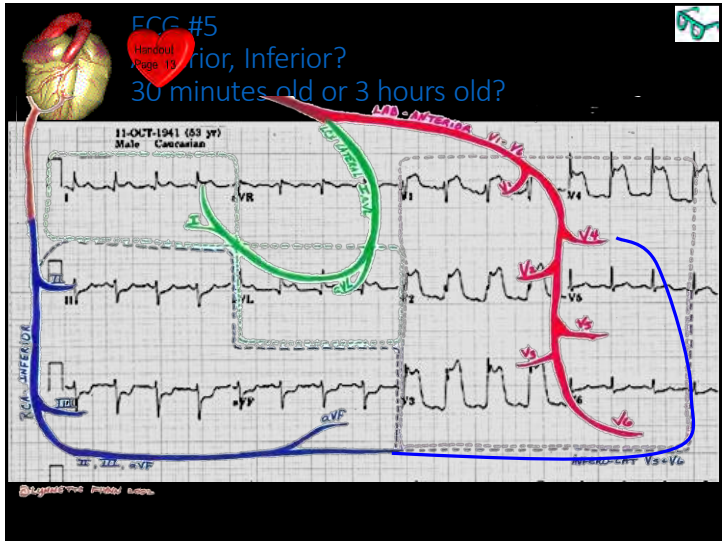
12-Lead ECG



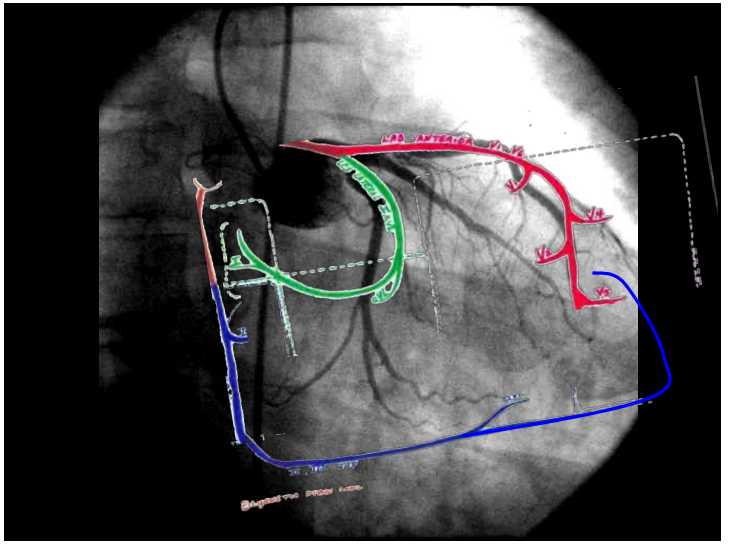
- ECG
- Exercise Stress Testing (EST)
- Ambulatory Electrocardiography



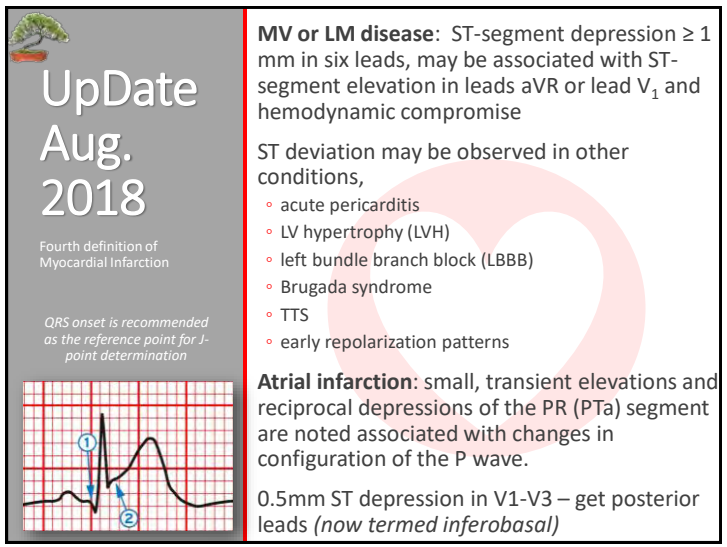
4


ECG #5
Handout Page 13
rior, Inferior?
30 minutes old or 3 hours old?
11-OCT-1941 (53 yr)
Male Caucasian
RCA - INFERIOR
LAD ARTERIOGRAM
V1-V6
aVF

5


LAD ARTERIOGRAM
V1-V6

6


UpDate
Aug.
2018
Fourth definition of Myocardial Infarction
QRS onset is recommended as the reference point for J-point determination
1
2
MV or LM disease: ST-segment depression ≥ 1 mm in six leads, may be associated with ST-segment elevation in leads aVR or lead V1 and hemodynamic compromise
ST deviation may be observed in other conditions,
acute pericarditis
LV hypertrophy (LVH)
left bundle branch block (LBBB)
Brugada syndrome
TTS
early repolarization patterns
Atrial infarction: small, transient elevations and reciprocal depressions of the PR (PTa) segment are noted associated with changes in configuration of the P wave.
0.5mm ST depression in V1-V3 – get posterior leads (now termed inferobasal)

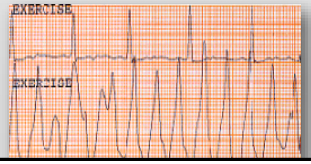
7


Exercise Stress Test

9

Clinical Relevance

- **Prognostic**
  - **Risk stratification**
    - after MI
    - hypertrophic cardiomyopathy
  - **Evaluation**
    - Revascularization or drug treatment
    - Exercise tolerance and cardiac function
  - **Assessment**
    - Cardiopulmonary function with dilated cardiomyopathy/HF
    - Treatment of arrhythmia
- **Diagnostic**
  - **Assessment**
    - Chest pain with intermediate probability of CAD
    - Of symptoms: presyncope before or during exercise
  - **Arrhythmia provocation**



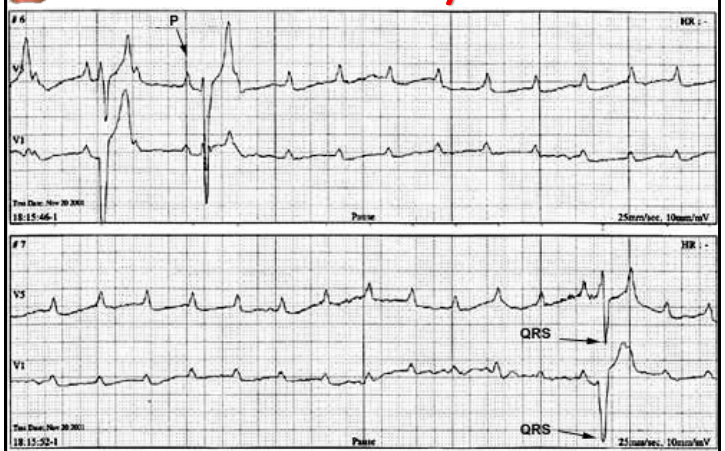
10

When to Stop the Stress

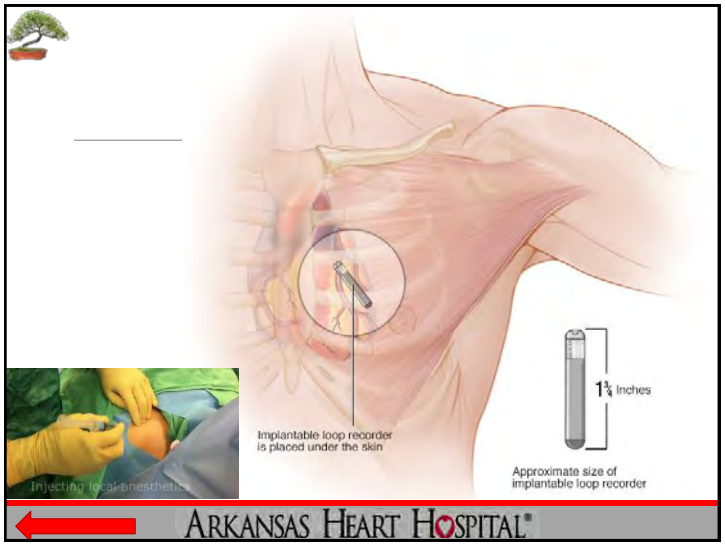
- ST elevation (> 1 mm) in leads without diagnostic Q waves (other than V_1 or aVR)
- Sustained ventricular tachycardia
- Signs of poor perfusion (cyanosis or pallor)
- Increasing nervous system symptoms (eg, ataxia, dizziness, near-syncope)
- Moderate-to-severe angina
- Drop in systolic blood pressure > than 10 mm Hg from baseline, despite an increase in workload, when accompanied by other evidence of ischemia
- Technical difficulties in monitoring ECG tracings or SBP
- Subject's desire to stop

17

Ambulatory ECG

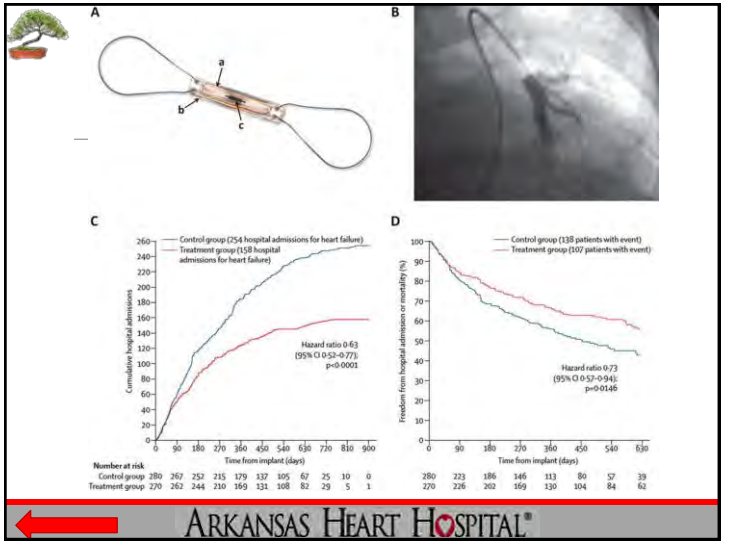


18

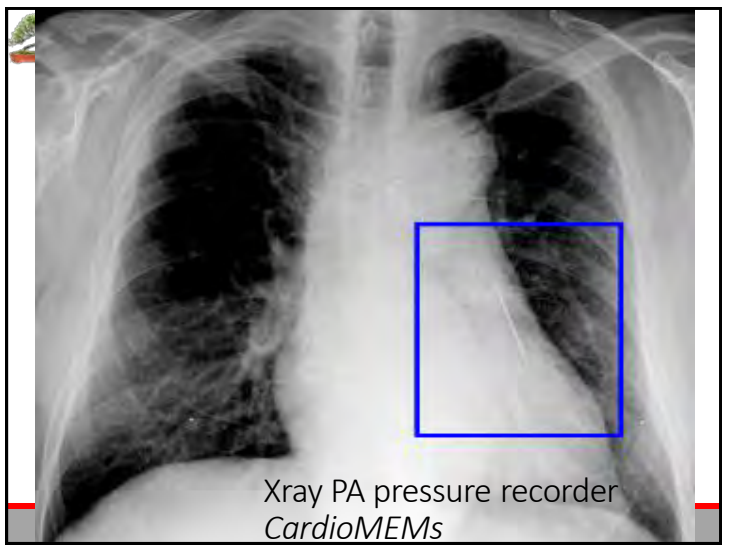


19

20



21



22

CALCIUM SCORING - CAC
ECHOCARDIOGRAM
MUGA & NUCLEAR MEDICINE SCANS
ANGIOGRAPHY
MRA/MRI
EPICARDIAL MAPPING

Imaging

23

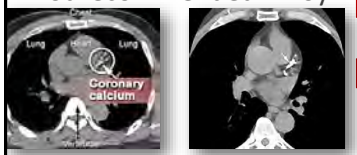
CAC – Calcium Scoring

Calcium is measured using a noncontrast limited CT

3-5 second breath hold

Scored 0 to 500

Not recommended < 40y



CAC score of 0

< .1% risk
Ø statin unless strong indicators exist
No ACEi or ASA required
Lifestyle changes only

CAC score of 100

would benefit from ASA therapy
75% of all events occurred when > 100

CAC score of < 300 *< 75th percentile*

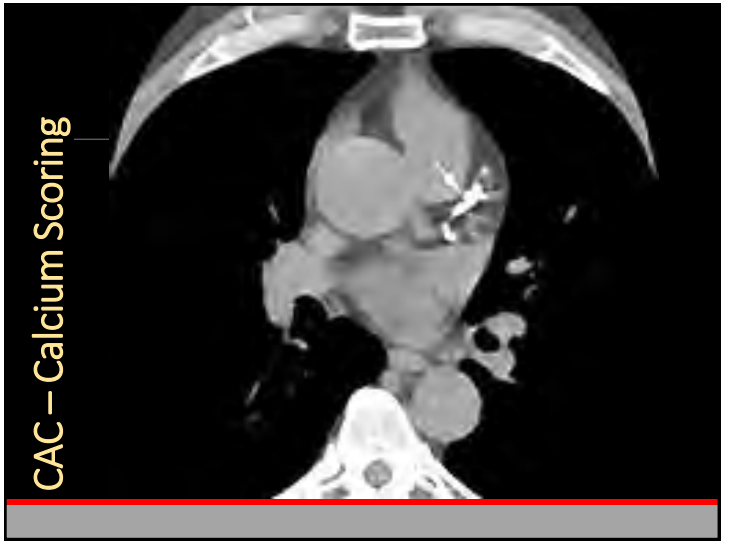
Low to moderate dose statins

CAC score of > 300 *> 75th percentile*

High risk
High dose statins ↓ CV death by 42%

Budoff, Matthew J MD, Coronary Artery Calcium Scoring Can Help Guide Statin Therapy; theheart.org on Medscape; http://www.medscape.com/viewarticle/857126#vp_2

24

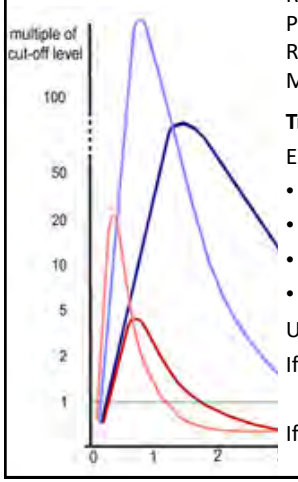


25

Lab

ENZYMES

26



Troponin T Nml < 0.3 µg/L
Rise 3-6 hours
Peak 10-24 hours
Remains elev 10-14 d
More sensitive to UAP

Troponin I Nml < 10 µg/L
Elev in any disease causing myoc cell injury:

- UAP
- AMI
- Contusions
- Cardiomyopathy

Useful when CP with neg ECG
If not elev on adm and at 6 hours –
low risk and may be sent home
If CP, no ECG change and elev Troponin I –
High incidence of CAD on angiography

30

Echocardiography

Noninvasive ultrasonic imaging to visualize heart

Diagnose & guide therapy for problems with structure, function & motion of heart, chambers, valves and great vessels

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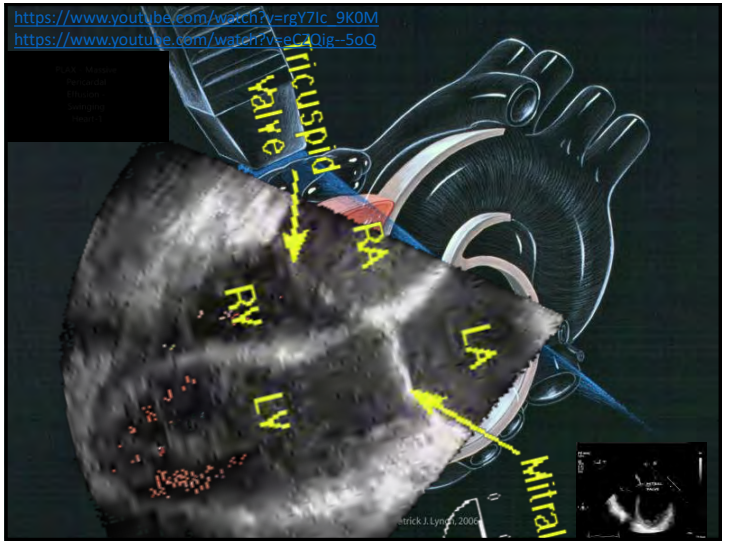
https://www.youtube.com/watch?v=rgY7Ic_9K0M
https://www.youtube.com/watch?v=eC7Qig--5oQ



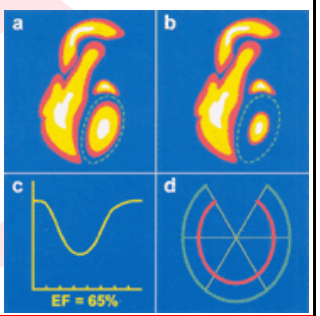
32

Nuclear Medicine

★Radioactive agents to provide information on heart function, extent of ischemia & infarction

2 main varieties:

1. Myocardial Perfusion Scan
   - Perfusion
2. First Pass Scan & Multiple gated acquisition (MUGA) blood pool imaging
   - Pumping ability



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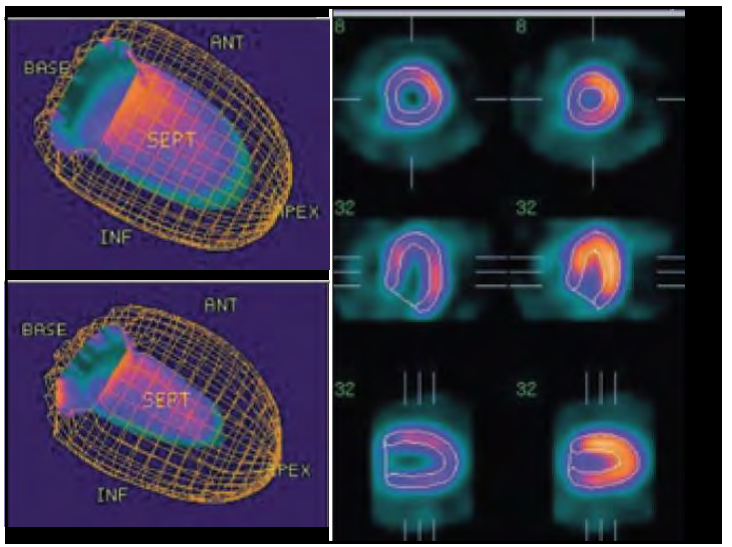
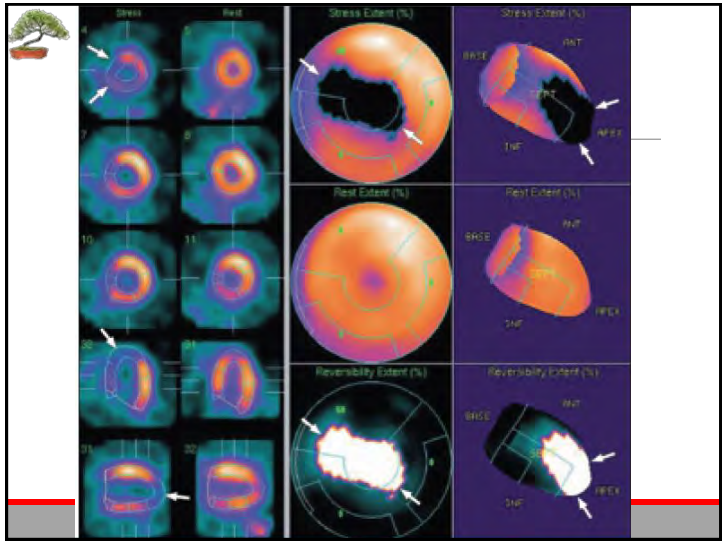
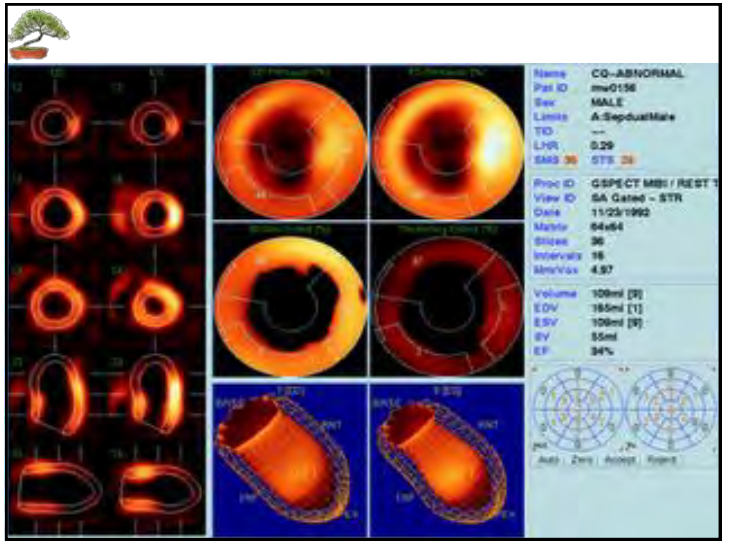
41



42



43



44

Patient care Nuclear Medicine

- *Cardiac* exams involve fasting for at least 4 hours before the exam
- Stress/persantine tests require no caffeine for 24 hours prior to the scan.
- We advise that you wait 3 hours before getting very close to children, pregnant women or pets.
- Thyroid iodine treatments, which require approximately 3 days before close contact with the above parties.)

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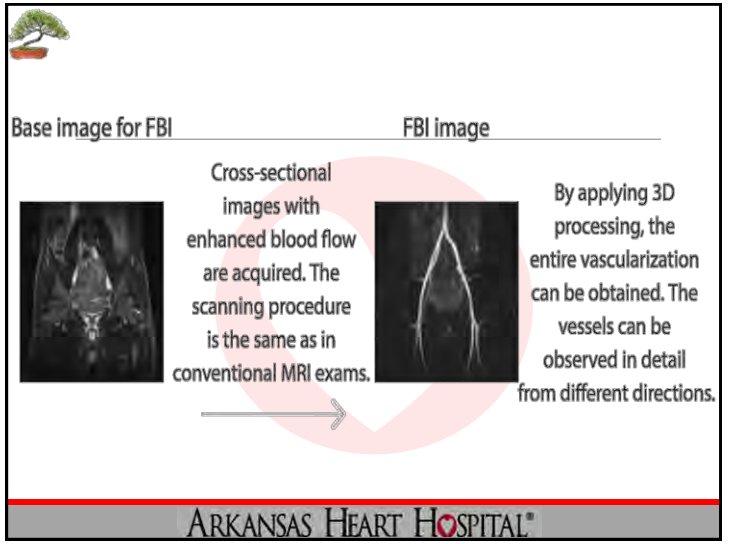
45

Non-contrast MRA

- Anatomy is Key
- Great for patients with renal insufficiency
  - New FDA warning on Gadolinium
- Time of Flight (TOF) MRA *(technique to visualize flow within vessels, without the need to administer contrast.)*
- Phase Contrast MRA *(technique that can be used to visualize moving fluid.)*
- Fresh blood imaging technique (FBI)
  - Fast
  - STIR (fat suppression)
  - ECG gated
  - Arterial and Venous flow
    - Subtract venous from arterial

48


Base image for FBI
FBI image
Cross-sectional images with enhanced blood flow are acquired. The scanning procedure is the same as in conventional MRI exams.
By applying 3D processing, the entire vascularization can be obtained. The vessels can be observed in detail from different directions.
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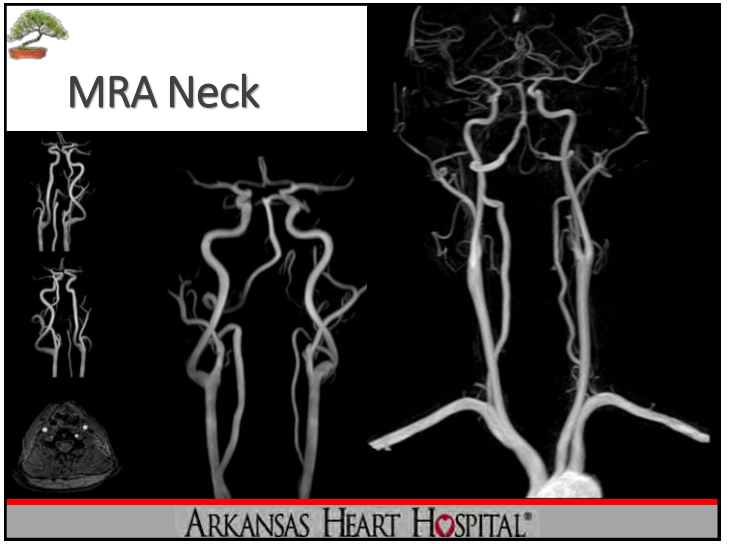
MRA Neck
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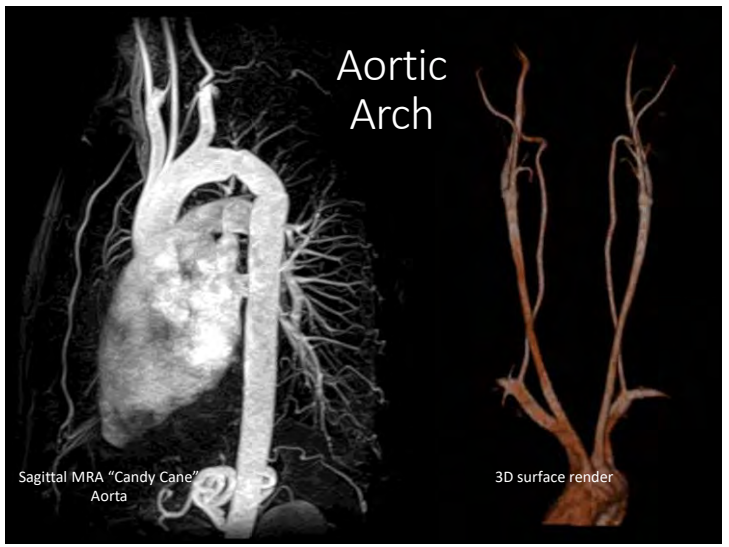
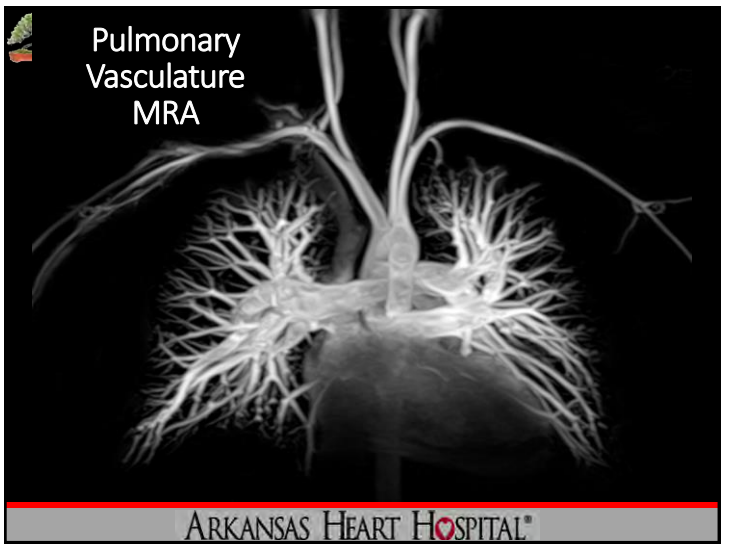
50


Aortic Arch
Sagittal MRA "Candy Cane" Aorta
3D surface render

51


Pulmonary Vasculature MRA
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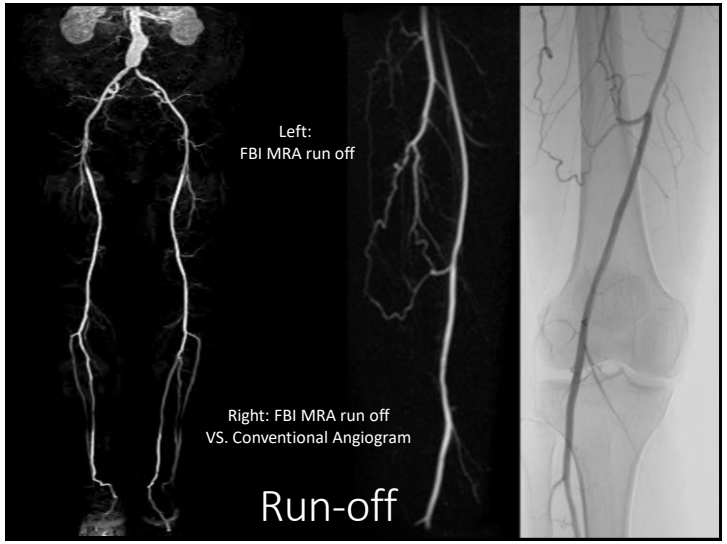
52



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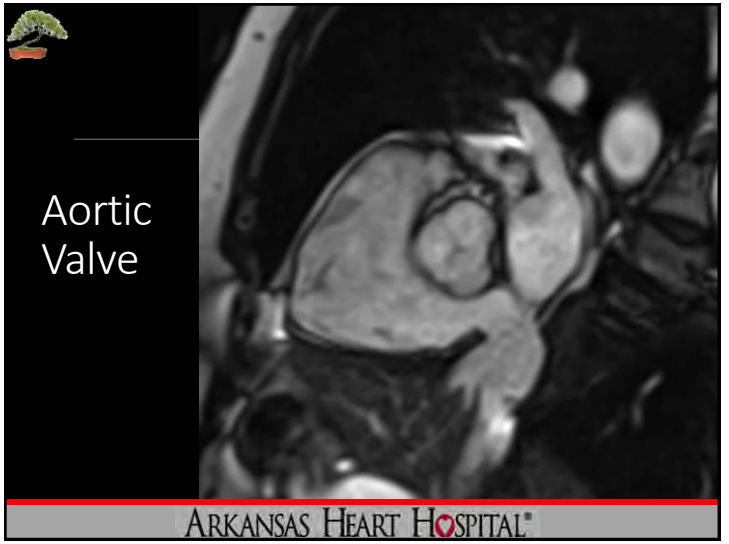
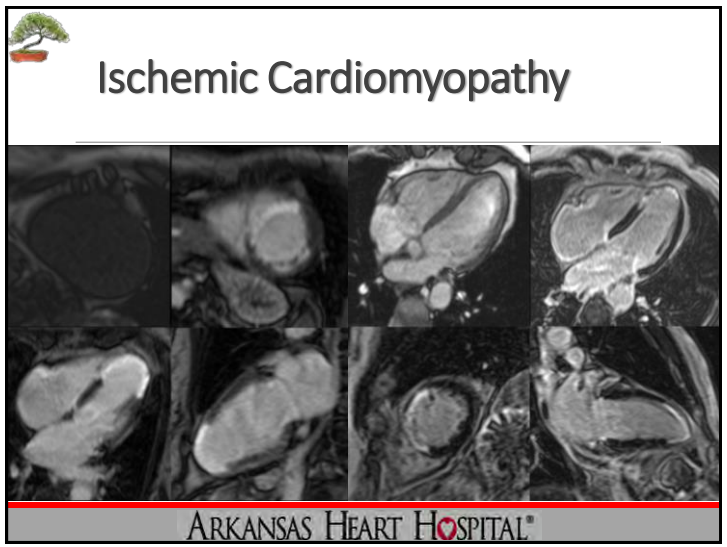


62



64

Cardiac MRI Indications

Anatomy
- ASD
- VSD
- Tri vs. Bicuspid delineation
- Myocardial wall thickness

Cardiac masses
- Myxomas
- Thrombus
- Tumors

DCE
- Ischemic vs. Non-Ischemic Cardiomyopathy

Excellent tool for functional imaging
- EF calculation
- Shunt fractions using flow quantification
- Myocardial motion

Valvular pathology
- Stenosis
- Regurgitation
- Valvular vegetation

65

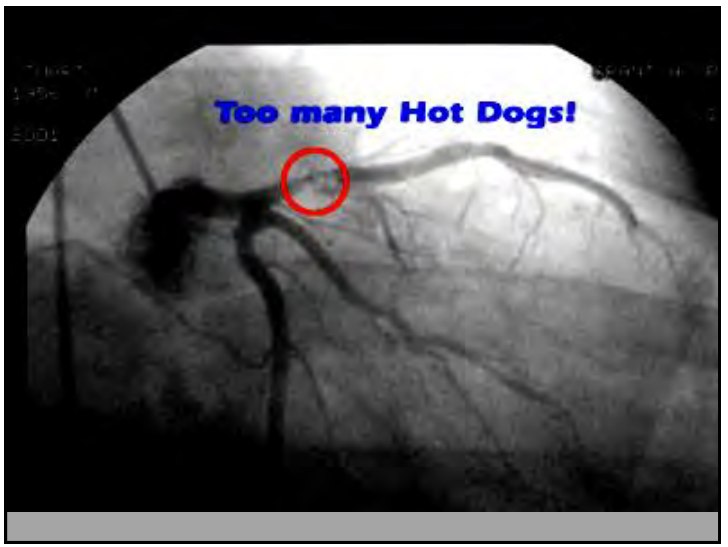
Cardiac Cath and Angio

- LV gram
- Coronary angiogram
- Angioplasty
- Stent deployment
- Embolectomy
- Atherectomy
- IV ultrasound
- OCT

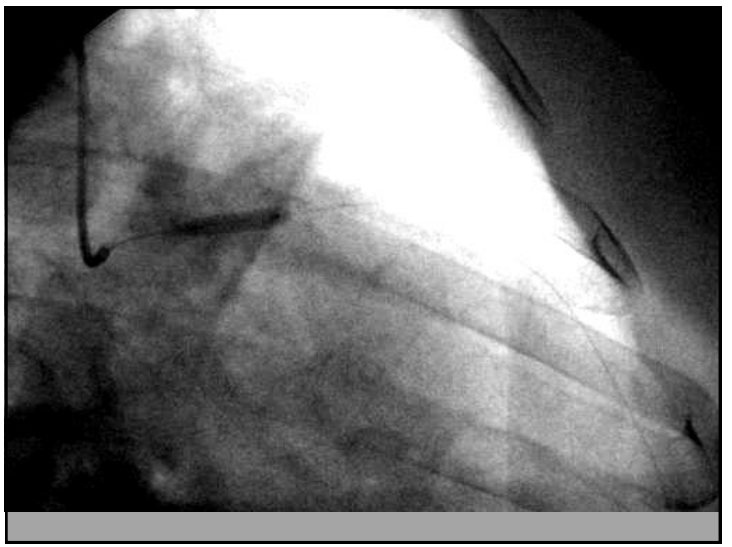
- Definitive techniques for establishing cause & severity of cardiac disease
- Combined hemodynamic & angiographic invasive procedure which may:
  - Confirm clinically suspected heart condition
  - Define anatomy & physiology
  - Provide mechanism for treatment

68

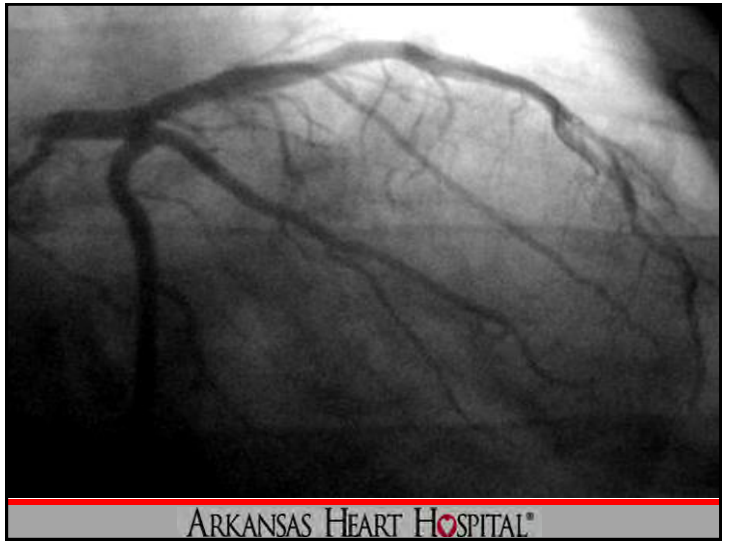
69



70



71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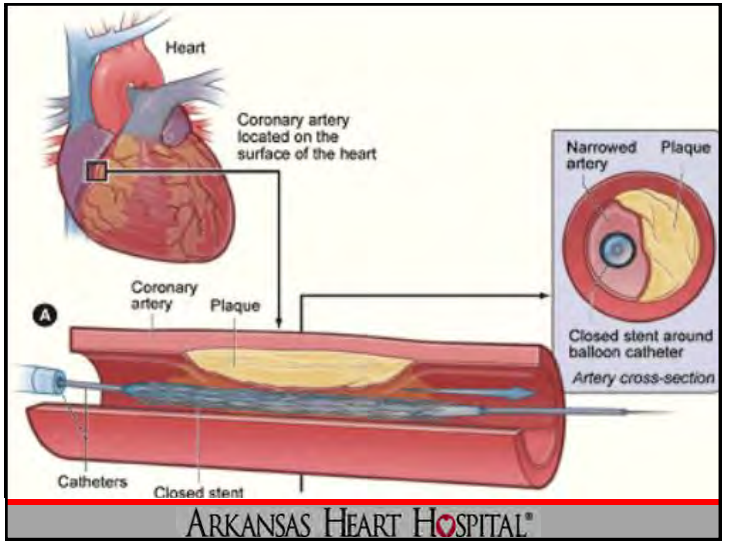
Heart
Coronary artery located on the surface of the heart
Narrowed artery
Plaque
Coronary artery
Plaque
A
Closed stent around balloon catheter
Artery cross-section
Catheters
Closed stent
ARKANSAS HEART HOSPITAL

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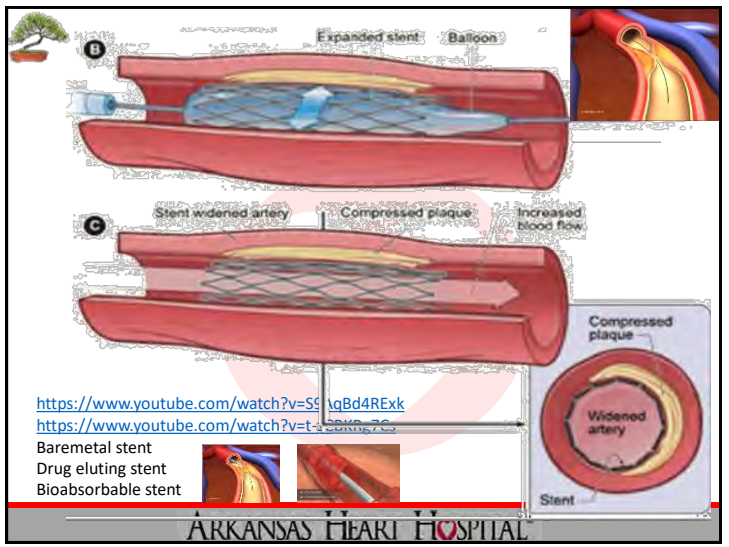
B
Expanded stent
Balloon
C
Stent widened artery
Compressed plaque
Increased blood flow
Compressed plaque
Widened artery
Stent
https://www.youtube.com/watch?v=S
qBd4RExk
https://www.youtube.com/watch?v=t-
Baremetal stent
Drug eluting stent
Bioabsorbable stent
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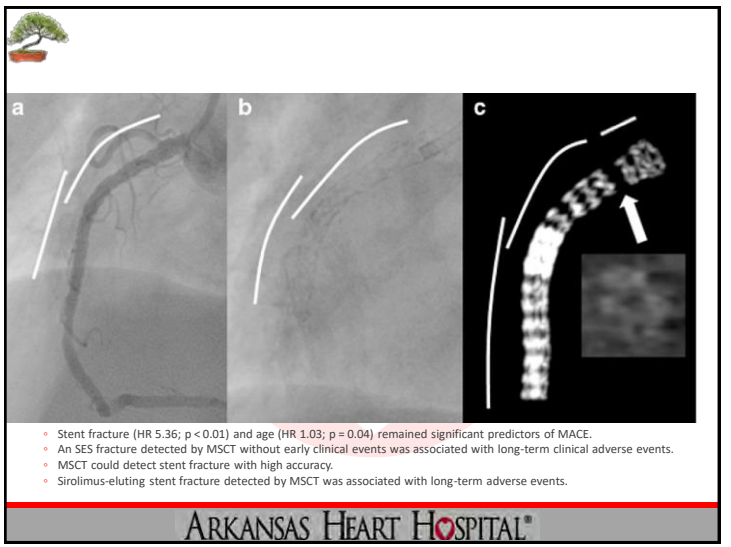
75


a
b
c
Stent fracture (HR 5.36; p < 0.01) and age (HR 1.03; p = 0.04) remained significant predictors of MACE.
An SES fracture detected by MSCT without early clinical events was associated with long-term clinical adverse events.
MSCT could detect stent fracture with high accuracy.
Sirolimus-eluting stent fracture detected by MSCT was associated with long-term adverse events.
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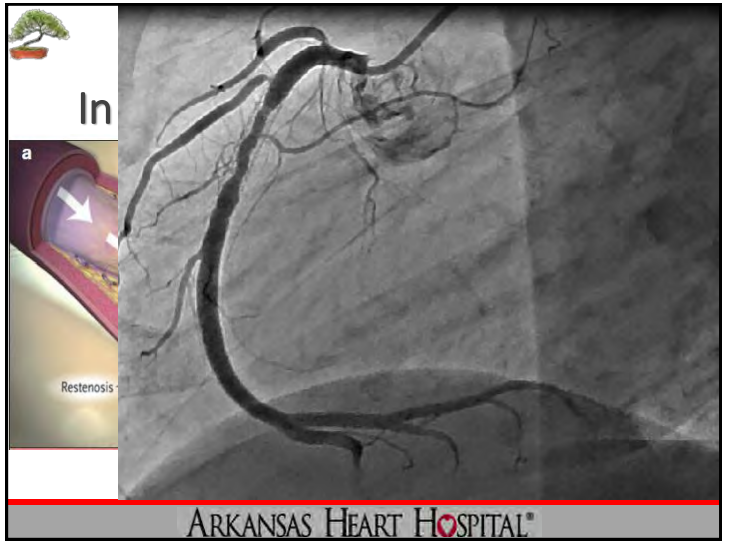
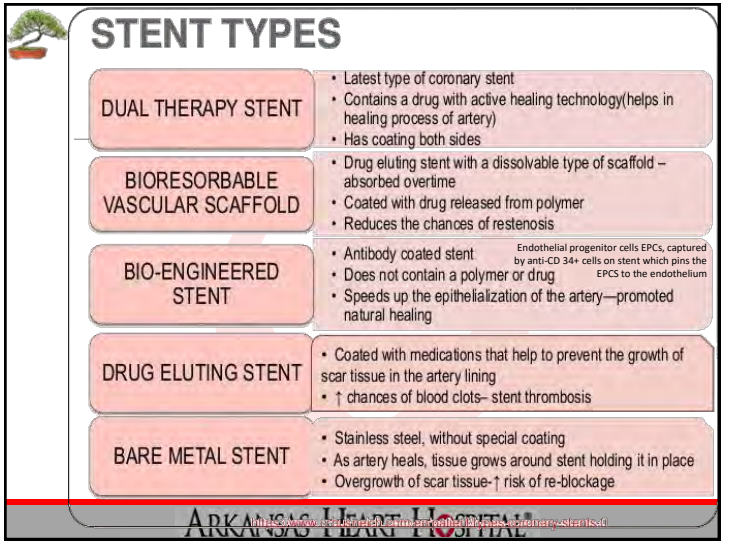
79


In
a
Restenosis
ARKANSAS HEART HOSPITAL®

80


STENT TYPES
DUAL THERAPY STENT
• Latest type of coronary stent
• Contains a drug with active healing technology(helps in healing process of artery)
• Has coating both sides
BIORESORBABLE VASCULAR SCAFFOLD
• Drug eluting stent with a dissolvable type of scaffold – absorbed overtime
• Coated with drug released from polymer
• Reduces the chances of restenosis
BIO-ENGINEERED STENT
• Antibody coated stent
• Does not contain a polymer or drug
• Speeds up the epithelialization of the artery—promoted natural healing
Endothelial progenitor cells EPCs, captured by anti-CD 34+ cells on stent which pins the EPCS to the endothelium
DRUG ELUTING STENT
• Coated with medications that help to prevent the growth of scar tissue in the artery lining
• ↑ chances of blood clots– stent thrombosis
BARE METAL STENT
• Stainless steel, without special coating
• As artery heals, tissue grows around stent holding it in place
• Overgrowth of scar tissue-↑ risk of re-blockage
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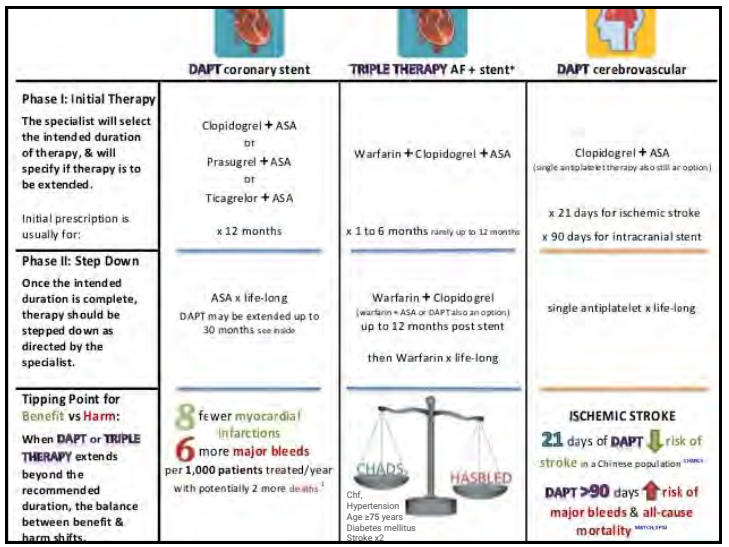
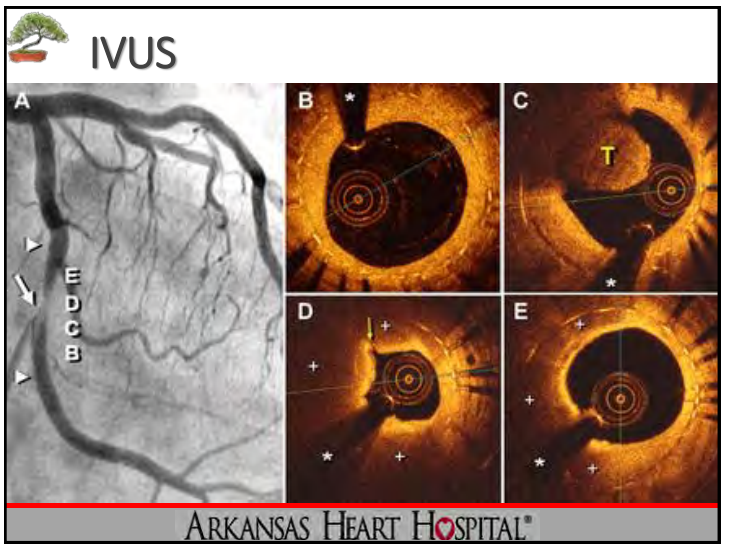

DAPT coronary stent
TRIPLE THERAPY AF + stent*
DAPT cerebrovascular
Phase I: Initial Therapy
The specialist will select the intended duration of therapy, & will specify if therapy is to be extended.
Initial prescription is usually for:
Clopidogrel + ASA
or
Prasugrel + ASA
or
Ticagrelor + ASA
x 12 months
Warfarin + Clopidogrel + ASA
x 1 to 6 months rarely up to 12 months
Clopidogrel + ASA
(single antiplatelet therapy also still an option)
x 21 days for ischemic stroke
x 90 days for intracranial stent
Phase II: Step Down
Once the intended duration is complete, therapy should be stepped down as directed by the specialist.
ASA x life-long
DAPT may be extended up to 30 months see inside
Warfarin + Clopidogrel
(warfarin + ASA or DAPT also an option)
up to 12 months post stent
then Warfarin x life-long
single antiplatelet x life-long
Tipping Point for Benefit vs Harm:
When DAPT or TRIPLE THERAPY extends beyond the recommended duration, the balance between benefit & harm shifts.
8 fewer myocardial infarctions
6 more major bleeds
per 1,000 patients treated/year
with potentially 2 more deaths
CHADS
HASBLED
Chf, Hypertension
Age ≥75 years
Diabetes mellitus
Stroke x2
ISCHEMIC STROKE
21 days of DAPT ↓ risk of stroke in a Chinese population
DAPT >90 days ↑ risk of major bleeds & all-cause mortality

82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A
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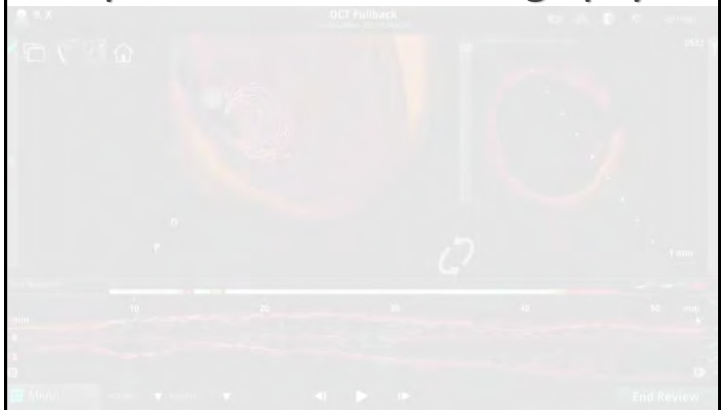
Optical Coherence Tomography



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Debulking techniques

Cutting and scoring balloons

Atherectomy
- Laser
- Directional
- Rotational

brachytherapy

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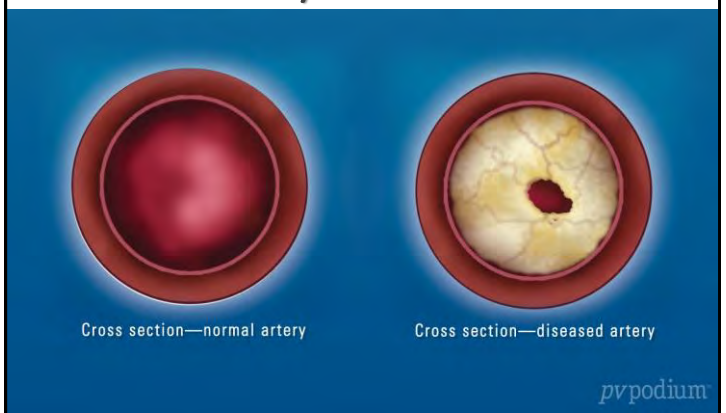
Atherectomy



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Embolectomy

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EKOS for submassive PE

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MCS – *Mechanical Cardiac Support*

Impella application:

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Watchman

https://www.youtube.com/watch?v=jffGNtcpLQU

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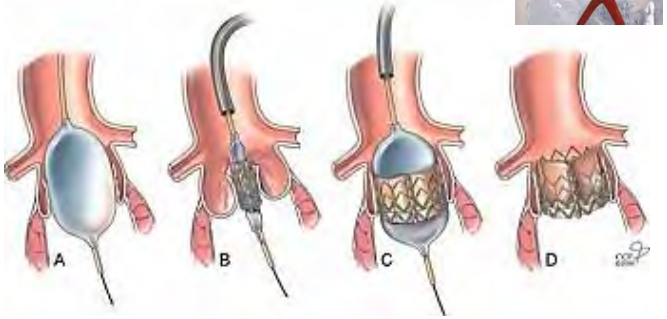
TAVR

https://www.youtube.com/watch?v=7TLfi



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Complications

- Heart Perforation
- Embolism
- Arrhythmias
- Allergic reaction
- MI
- Hematoma
- Hemorrhage
- Infection
- Artery dissection
- DEATH

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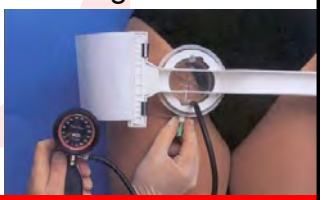
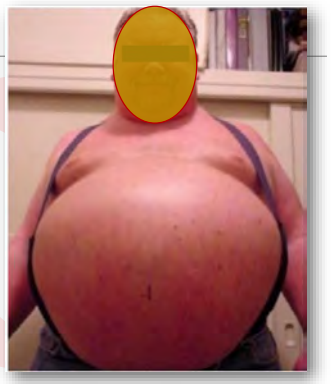
Post Procedure care

- Anticoagulation
- Care of femoral insertion site: distal circulation, use of femstop, bed rest.
- Continued Cardiac Monitoring
- Blood check: Coags
- Cardiac Rehab



96

Every case begins and ends with Access!



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Pulling an Arterial sheath:

1. Place your index, middle and sometimes your ring finger above the sheath to feel the pulse.
2. Slowly remove the sheath in a sterile manner, holding occlusive pressure to avoid bleeding.
3. Place your other hand on top of your pressure hand. *(enables alternation of which hand is holding the pressure)*
4. Do not use your fist or fingertips
5. Do not hold over padding

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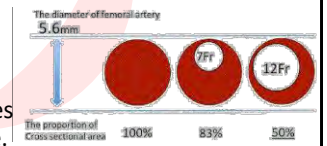
Hold times

Abide by your hospital's policies.

My own hold times are simple.
Any artery with ≤7 French hold for 20 minutes
Add 5 minutes per additional sheath size

Impella, mitraclip 13-14fr
AVR, 10-12 fr

- First 5 minutes, full pressure
- release slightly every 5 minutes
- last 5 minutes is light pressure.



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Venous sheath

1. Hold 10 minutes regardless of site
2. Do not hold as much pressure as with an artery
3. Primary hold below site
4. Control hold above

while holding pressure, whether arterial or venous, constantly feel around the site, look for bleeding.

Have atropine available if needed for vagal reactions.

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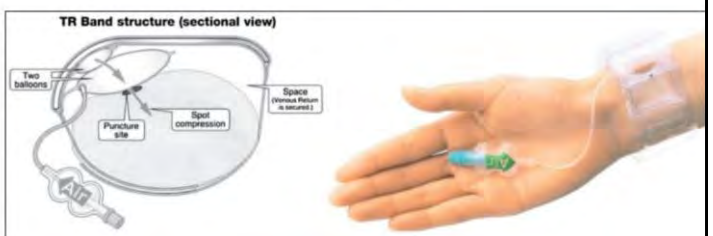
Radial Access

Access Site Care- Radial Artery

- TR band placed at termination of case



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TR Band

- Radial
  - While TR band is on, check wrist every 15 minutes for bleeding, hematoma, cap refill, color, warmth, and pulse oximetry signal
  - Start to deflate in 2 hours following diagnostic procedure, per protocol
  - Start to deflate in 4 hours following intervention
  - Once off, check site every 15 minutes for the first hour, then every 30 minutes for the next hour, then every hour for the next two hours
  - Bedrest is NOT necessary

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Radial Access Complication- Hematoma

- Notify MD
- Can follow forearm diameter
- Elevate arm
- Place additional TR band above/below initial band
- CoFlex wrap to moderate pressure up to forearm
- Inflate BP cuff to <20mmHg of systolic pressure if grade III/IV hematoma, deflate every 15 minutes
  - Can place pulse oximeter on finger to ensure presence of waveform
- Place bag of ice or cold compress on hematoma

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Radial vs Femoral Access Complications

Types	Radial	Femoral
Hematoma >5cm	<1%	3-5%
Pseudoaneurysm	<1%	<1%
A-V Fistula	<1%	1-2%
Skin Infection	<1%	<1%
Dissection/Rupture	<1%	<1%
Thrombosis	<1-10%	<1%
Distant bleeding	–	<1%
Nerve Injury	–	<1%

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In Addition to the Access Site, Monitor the Patient including Vitals…

- Every 15 minutes x 4, then every 30 minutes x 2, then every hour
- Assess:
  - Symptoms
    - Chest pain, SOB, back pain, Access site pain, pain in extremity
  - Mental status
    - Stroke, over-sedation
  - Blood pressure
    - SBP>160
    - SBP<90
  - Respiratory status
    - CHF, anaphylaxis (check body for rash)

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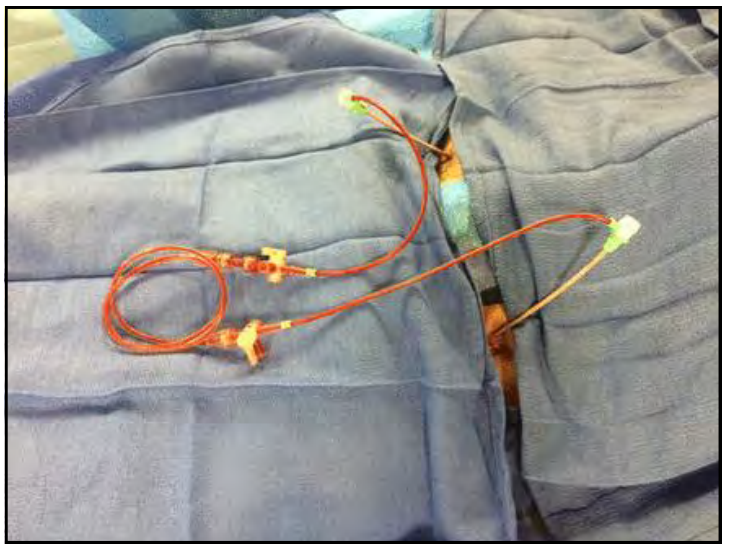

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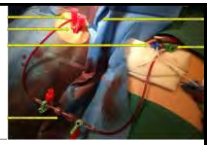
A & V sheaths



When both arterial and venous sheaths are present, pull the arterial sheath first.

- It takes longer to obtain hemostasis with an arterial sheath
- Large venous access is used for IV access

Experienced ppl may pull both sheaths together.

- Pull the arterial sheath first,
- After 10 minutes pull the venous sheath.
- Pulling one sheath at a time will reduce the risks of complications.

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Hematoma

- Usually evident within 12 hours of sheath removal
- Local discomfort, hypotension
- Avoid by careful puncture, compression, closure, immobility
- Risk factors
  - Women
  - SBP>160 mm Hg
  - Artery puncture >1
  - Sheath time >16 min
  - ACT≥175 sec
  - Glycoprotein (GP) IIB/IIIa inhibitors
  - Low Molecular Weight Heparin before procedure
  - Personnel change during compression
  - Anti-coagulant-treatment before procedure

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Electrophysiology

EP study
- Conduction Study
- Arrhythmia induction
- Cardiac Mapping

Ablations
- Atrial Fib
- Accessory Pathway
- AV node

Pacing Maneuvers

Tilt Table Study

Goals of Diagnostic EP

- To characterize physiologic and pathologic properties of the atria and ventricles, the AV conduction system, identify accessory pathways, and determine the sites and mechanisms of arrhythmias
- To correlate patient symptoms with arrhythmias and evaluate risks for life threatening events and/or differentiate arrhythmias
- To define arrhythmia induction and termination methods for EPS guided interventions.

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Tilt Test

Response/diagnosis Characteristics

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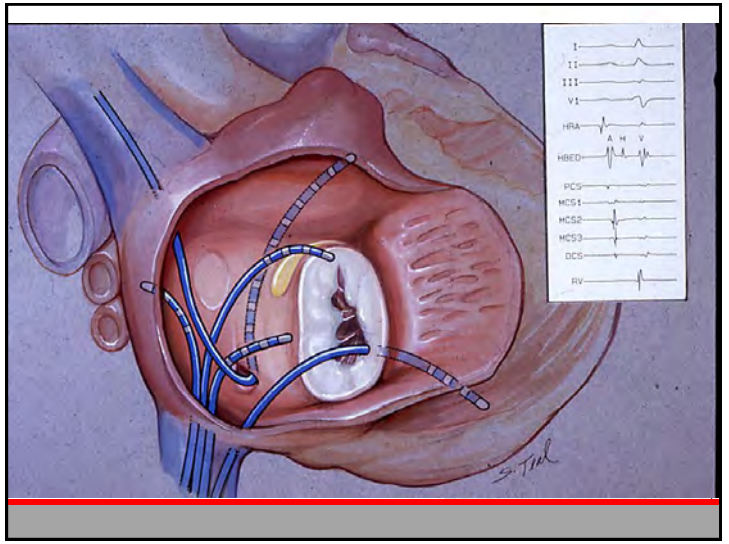

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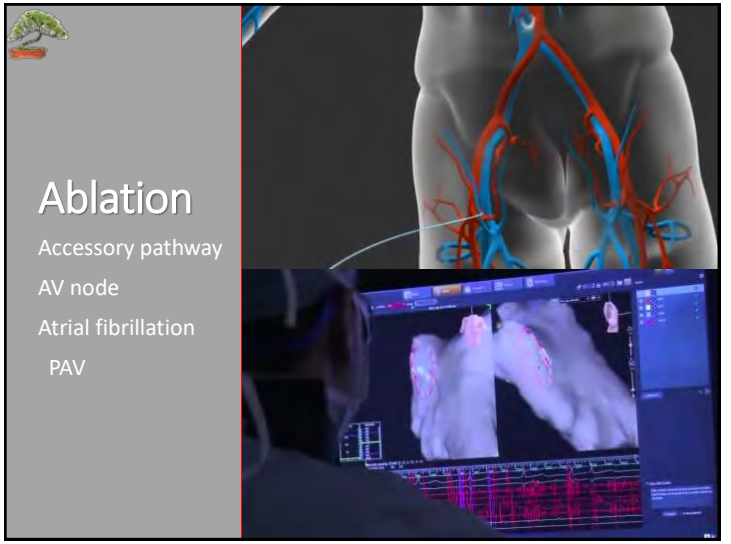

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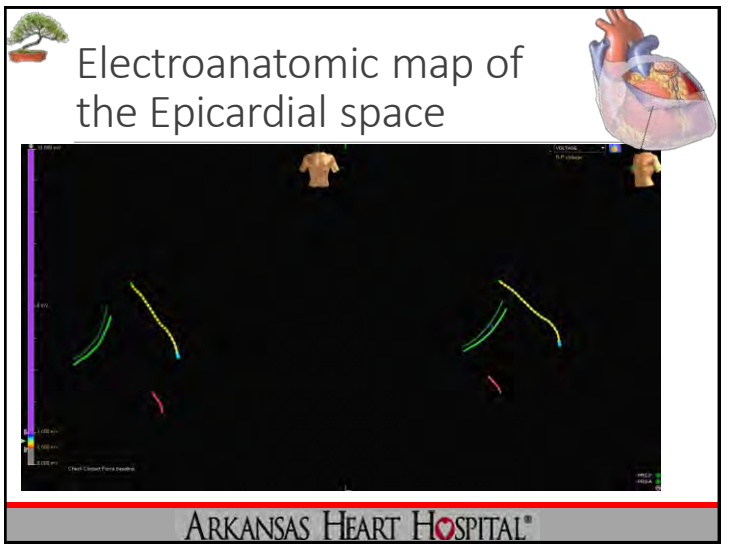

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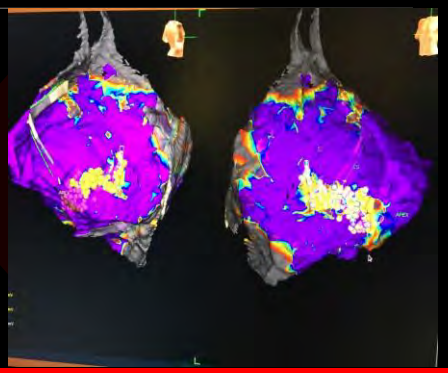
Final.

The final result, ablation of approximately 10% of the RV epicardial surface, from the apex to the mid RV free wall.

After our ablation we attempted to induce an arrhythmia

We were non inducible despite aggressive attempt.



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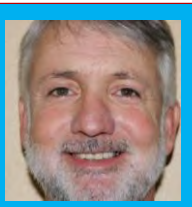
Why?



48 yo male ARVC with VT and ICD shocks

Amiodarone

Post Ablation: Now off Amiodarone and no VT in close follow up.

- 68 yo female with frequent PVCs, approx. 35000 PVCS/24 hour period
- NICM LVEF 30%
- BIVICD pacing at 70%
- **Post Ablation: No more dyspnea, BIV Pacing 90%**

- 40 yo female with frequent PVCs, 5000 PVCs/24hr
- Dizziness, syncope
- 3 prior ablations
- **"0" PVCs in follow up at 2 months, and asymptomatic**

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On the Horizon…

https://www.dicardiology.com/videos/video-photo-realistic-lighting-enhance-3d-echocardiography

Percutaneous fem-pop bypass
Radiation stewardship
Digital processing of nuclear studies
Lesion typing
Pacemaker pressure/volume monitoring
Septal occluder just approved
Photo realistic lighting for echocardiograms
Quad Chamber volume imaging
Intravascular lithotripsy
The future is NOW

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CABGs

- Suitable for multiple vessel disease
- Especially useful for:
  - AMI
  - TVD
  - Left main CAD
  - Angina unresponsive to medical therapy
- "Bypass graft"around the stenosis: use saphenous vein or internal mammary artery

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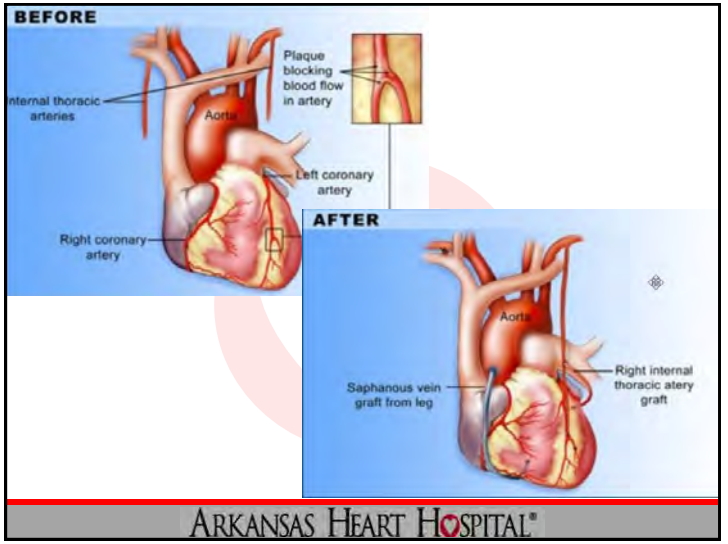

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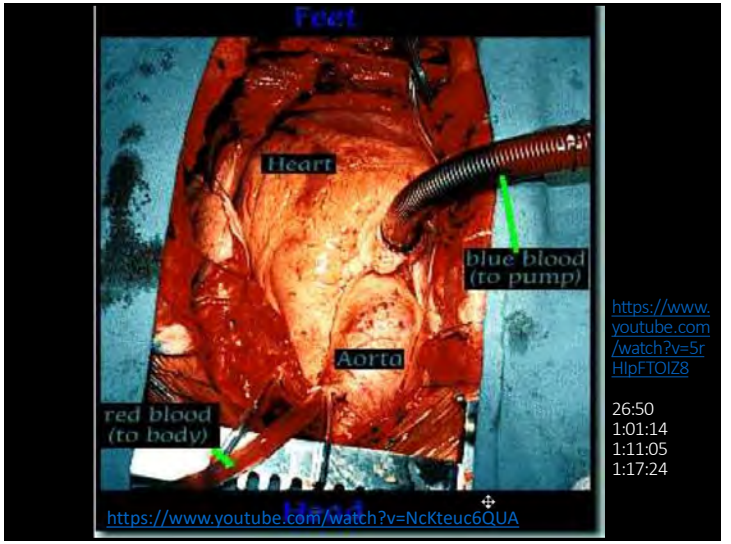

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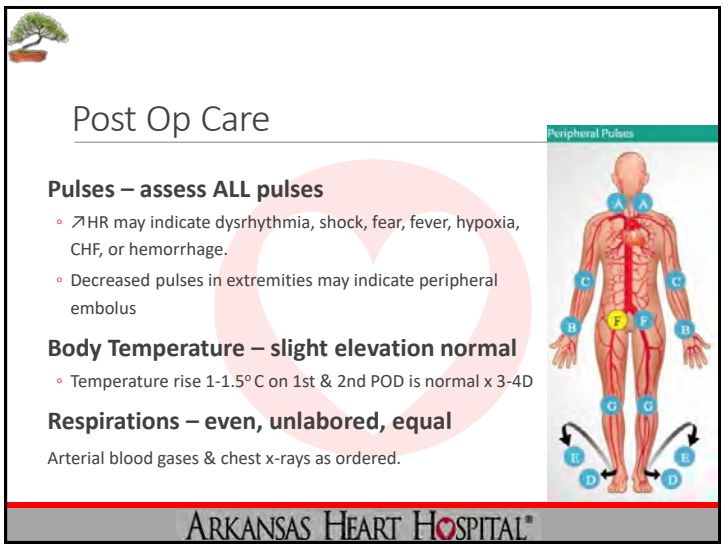

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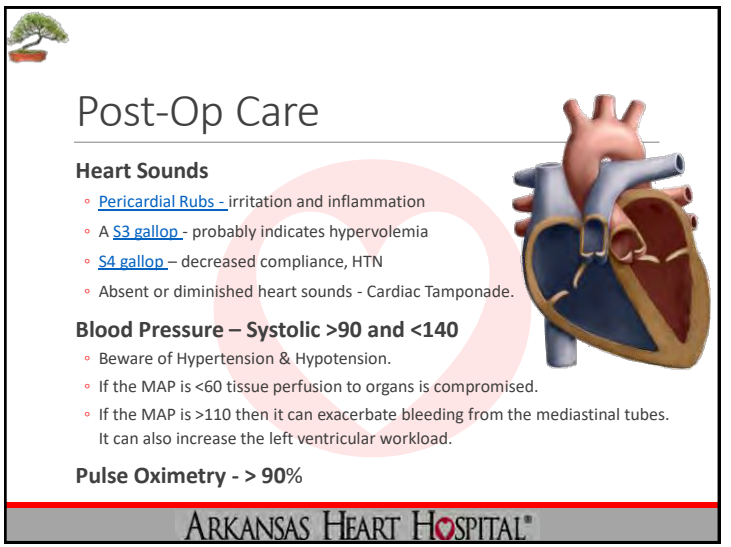

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Others?

Stem Cells

- Iron infused stem cells
- Antioxidants create abnormalities in stem cells

Electrophysiology

- Tilt test
- AV ablation
- Atrial fib ablation
- Accessory pathway ablation
- EP study

Implantables

- Loop recorder
- PA pressure recorder

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Ito, T., Kimura, M., Suzuki, T., **Impact of sirolimus-eluting stent fractures without early cardiac events on long-term clinical outcomes: A multislice computed tomography study,** Published 2014 in European Radiology, DOI:10.1007/s00330-014-3118-9

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Makhija et. Al, Three dimensional epicardial mapping and ablation of recurrent noon-ischaemic ventricular tachycardia, Indian Heart J. 2012 May 64(3) 324-328 , doi: 10.1016/S0019-4832(12)60092-3, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3860767/

Phillip Mumford, RCIS, Director Cath Lab 2012**The Correct Way to Pull a Sheath Issue Number Volume 20 - Issue 10 - October 2012** Palestine Regional Medical Center, Palestine, Texas October 5, **https://www.cathlabdigest.com/articles/Correct-Way-Pull-Sheath**

Saurav Chatterjee, Riyaz Bashir, Vladimir Lakhter, Brian O'Murchu, Brian O'Neill and Vikas Aggarwal, **JACC: Cardiovascular Interventions** Volume 10, Issue 12, June 2017DOI: 10.1016/j.jcin.2017.03.027 **PDF Article Case of Percutaneous Extracorporeal Femoro-Femoral Bypass for Acute Limb Ischemia From Large Bore Access, http://interventions.onlinejacc.org/content/10/12/e109**

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Pacemakers

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Clinical Educator
Central Arkansas Veterans Healthcare System
LRCCP Basic Critical Care Class
September 8, 2022

No conflicts of interest to disclose.

Learning Objectives

Identify the different types of pacemakers.
Recognize different rhythms associated with pacemaker malfunction.
Describe the pacemaker codes.

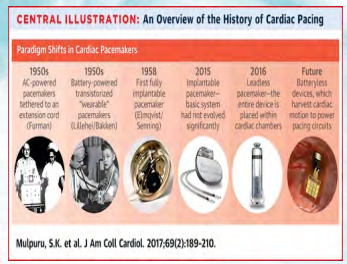
Pacemakers

A medical device that generates an electrical impulse to cause the heart muscle to contract. This process helps regulate the electrical conduction of the heart.

Pacemakers

If the patient cannot maintain an adequate cardiac output, use of a temporary or permanent pacemaker may be warranted.

Pacemaker History



Siva K. Mulpuru et al. *J Am Coll Cardiol* 2017; 69:189-210. JACC

Pacemaker History

The first implanted pacemaker therapy was introduced in 1958 in Stockholm. The pacemaker failed within just a few hours. The patient had another pacemaker placed in 1960. Additionally, he had 26 more pacemakers implanted in 43 years and outlived the surgeon that save his life. He died in 2001 of Melanoma at the age of 86 years old.

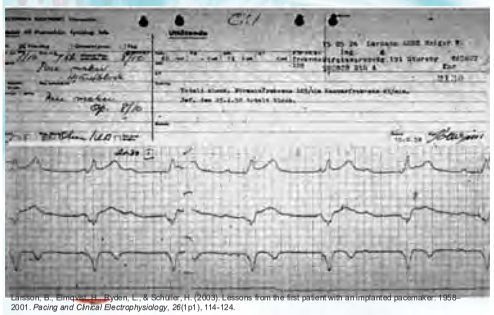
Jeffrey, K., Kay, G., & Furman, S. (1995). Cardiac pacing, 1960–1985: a quarter century of medical and industrial innovation. *Circulation*, *91*(7), 1978–1991.
Coombes, D. (2001). Pacemaker therapy 1: clinical indications, placement and complications. *Nursing Times*, *117*, 11-22.
Photograph: https://en.wikipedia.org/wiki/Arne_Larsson_(patient)

The First Pacemaker Implanted into a Patient

EKG before pacemaker

Larsson, B., Elmqvist, H., Ryden, L., & Schüller, H. (2003). Lessons from the first patient with an implanted pacemaker: 1958–2001. *Pacing and Clinical Electrophysiology*, *26*(1p1), 114-124.

EKG after pacemaker (Oct 8, 1958)



Larsson, B., Elmqvist, H., Ryden, L., & Schüller, H. (2003). Lessons from the first patient with an implanted pacemaker: 1958–2001. *Pacing and Clinical Electrophysiology*, *26*(1p1), 114-124.

EKG after pacemaker (Oct 15, 1958)

Larsson, B., Elmqvist, H., Ryden, L., & Schüller, H. (2003). Lessons from the first patient with an implanted pacemaker: 1958–2001. *Pacing and Clinical Electrophysiology*, *26*(1p1), 114-124.

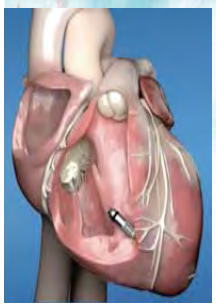
1960 Veterans Administration Pacemaker

On June 6, 1960, two years later the VA in Buffalo, NY carried out the first successful implantation of a battery-powered pacemaker with myocardial lead. The 77-year-old Veteran lived for 18 months after the surgery. There were 15 more Veterans with complete heart block that received a pacemaker that year.

In 1972, the first lithium battery was introduced.

Aquilina O. A brief history of cardiac acing. *Images Paediatr Cardiol*. 2006;8(2):17-81.
American Journal of Cardiology 2010 106810-818DOI: (10.1016/j.amjcard.2010.04.043)

New Pacemaker Technology



Single Chamber Leadless Pacemakers (20%)
- Medtronic Micra, first leadless pacemaker (FDA 2016)
- Abbott Aveir VR (March 2022)

New Trials
- Abbott Aveir DR i2i (2 chamber pacemaker) which communicates to each other to establish synchrony

Modes of Pacing

1. Fixed rate (asynchronous): Impulses are delivered at a predetermined rate.
2. Demand (synchronous): Impulses are delivered at a predetermine rate ONLY IF the patient's own heart rate is less than the pacemaker's set rate.

Pacemakers

- A mechanical device to stimulate the heart to contract
- Indications for Pacing:
  - Temporary
    - After heart surgery
    - Medication intoxication such as digoxin
    - Bridge to permanent pacing
    - Failure of permanent system
    - Overdrive pacing of tachycardia
    - Acute MI with CHJB or Mobitz II
  - Permanent
    - Congenital heart blocks
    - Acquired heart block (most often secondary to degeneration of the conduction system)
    - Sick Sinus Syndrome (SSS)
      - Brady-tachy Syndrome
      - Tachy-brady Syndrome

Pacemakers

- Methods of pacing
  - Temporary (generator external to the body)
    - Transvenous - through a vein
    - Epicardial – wires on epicardial surface of the heart
    - Transcutaneous – through electrodes placed on the chest wall
  - Permanent
    - All components of the system are inside the body

Pacemakers

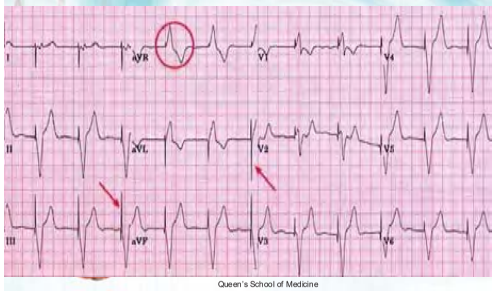
- Components
  - Pulse Generator – also called battery.
    - Contains battery (power source) and "brains" of system
  - Wire – Connects pulse generator to the electrode
  - Electrode – That part of system in contact with the heart muscle that transmits impulse and can detect electrical activity of heart.

Location of Pacemaker Leads

- **Temporary Pacing**
  - Transcutaneous: anterior and posterior of chest
  - Transvenous: right ventricle
  - Epicardial: right atrium and/or right ventricle
- **Permanent Pacing**
  - Right ventricle
  - Right atrium
  - Both right ventricle and right atrium
  - Bi-ventricular – right ventricle & coronary sinus
    - Usually implanted in Cardiac Cath Lab

Permanent Generator

SINGLE PACEMAKER SPIKES



Queen's School of Medicine

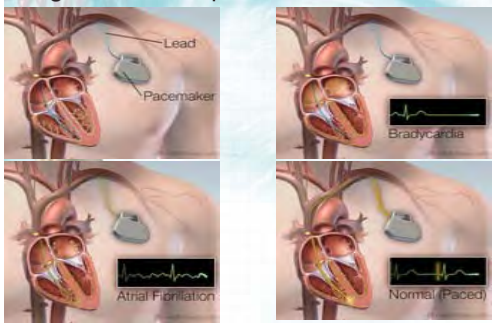
Pacemaker Terminology

- Dual chamber pacing: Two leads in the heart; one or both chambers being paced such as the right atrium or right ventricle
- Pacing the right atrium is called
  - Atrial pacing
- Pacing the right ventricle is called
  - Ventricular pacing
- Pacing of both atrium and ventricle
  - A-V sequential pacing

Pacemaker Terminology

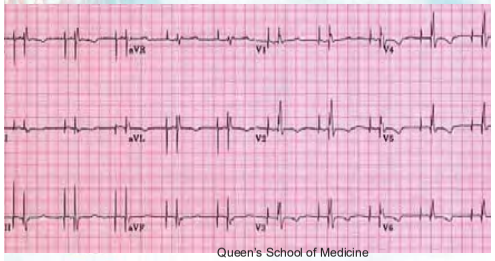
- Single chamber pacing: Only one lead in the heart; only one chamber being paced such as the right atrium or right ventricle.
- Pacing the right atrium is called
  - Atrial pacing
- Pacing the right ventricle is called
  - Ventricular pacing

Single Chamber pacemaker



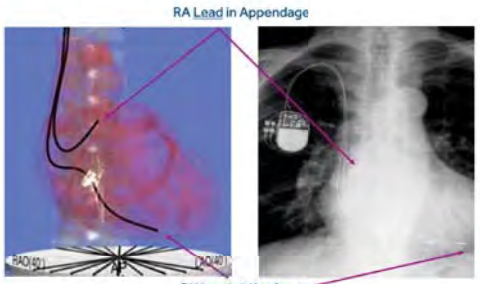
Dual Chamber pacemaker

DOUBLE PACEMAKER SPIKES



Queen's School of Medicine

DUAL CHAMBER PACEMAKER



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Pacemaker Terminology

- Bi-ventricular pacing: Two leads in the heart; one or both chambers being paced such as the right atrium or right ventricle
- Both ventricles and atria are paced
  - Of benefit to the Heart Failure patient who has dyssynchrony of the left and right ventricles

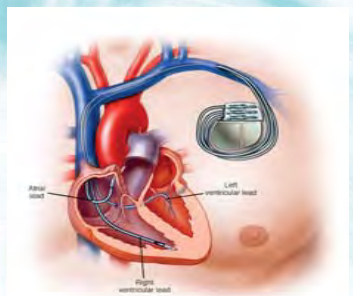
Temporary Pacemakers

Transcutaneous
Epicardial
Transvenous

Equipment needed for temporary pacing:

Generator
Leads
Patches

Biventricular Pacemaker

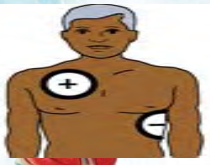


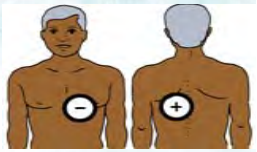
What type of pacemaker is this?

www.medtronicacademy.com

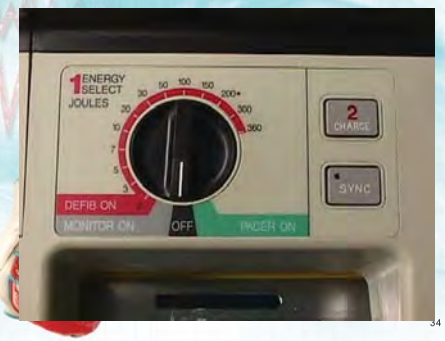
Transcutaneous Pacing

Transcutaneous Pacing





Sternum and posterior placement. In female patients, position the negative pacing electrode under the breast but above the diaphragm to prevent contraction of the diaphragm each time the pacer fires. (Modified from illustrations supplied by Physio-Control, Inc., Redmond, WA.)



Temporary Generator (Transvenous or Epicardial)

Single Chamber or Dual Chambers

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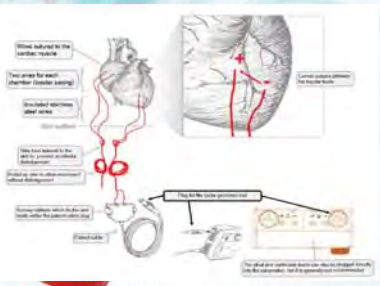
Pacer cables

Disposable

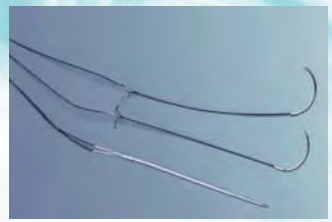
Non-disposable

Epicardial Wires



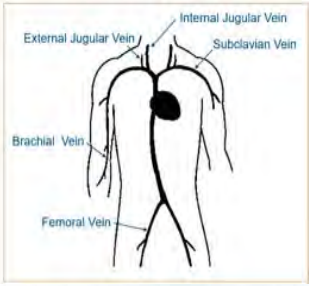
Anatomy of the temporary pacemaker circuit, Alex Yartsev

Epicardial Wires



Transvenous Pacing

Insertion Sites



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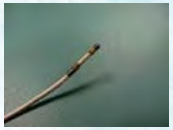
Transvenous wires



Universal Pacemaker Code

- A universal means of identifying the numerous functions of pacemakers by using a series of letters that represent a function.
- Need to know the first three positions (there are 5 or more).
- The code tells you the basic functioning of the generator.
- Not knowing how a pacemaker is programmed could result in misinterpretation of the rhythm.

Chamber(s) Paced	Chamber(s) Sensed	Response to Sensing	Rate Modulation	Multisite Pacing
O = None A = Atrium V = Ventricle D = Dual (A +V) S = Single (A or V)	O = None A = Atrium V = Ventricle D = Dual (A + V) S = Single (A or V)	O = None T = Triggered I = Inhibited D = Dual (T + I)	O = None R = Rate modulation	O = None A = Atrium V = Ventricle D = Dual (A + V)

Examples of pacing modes which are typically programmed:

DDD	VVI	DDIR
DDDR	VVIR	AAIR

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Universal Pacemaker Code

- Letters used in the code:
  - A = Atrium
  - V= Ventricle
  - I = Inhibited
  - D= Dual (A + V)
  - O = None
  - T = Triggered
- First three positions
  - Chamber paced
  - Chamber sensed
  - Response to a sensed event

Universal Pacemaker Code

- Examples of Code
  - V V I:
    - First letter V = Ventricular pacing
    - Second letter V = Ventricular sensing
    - Third letter I = Ventricular output inhibited if intrinsic event sensed
  - DDD:
    - First letter D = A + V paced
    - Second letter D = A + V sensed
    - Third letter D = T and I response to sensed event

Universal Pacemaker Code

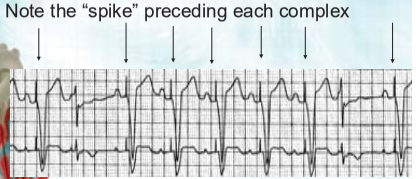
- Other examples include:
  - V O O
  - A A I
- Letters 4 and 5 of code indicate such functions as:
  - Multiprogrammability
  - Rate responsiveness
  - Communication possibilities

ECG Recognition of A Pacemaker Rhythm

- When the pacemaker "fires" or stimulates the heart to contract, an electronic spike is seen on the ECG.
- This "spike" is a vertical line either above, below or both the baseline.
- After the "spike" there will be a waveform which tells you which chamber was stimulated: a P wave or a QRS complex.

Pacemaker Identification

- Example of a Pacemaker Rhythm
- Note the "spike" preceding each complex



Pacemaker Identification

- After each pacemaker spike there should be evidence the heart depolarized. Either a P wave (depolarization of the atria) or a QRS complex (depolarization of the ventricles).
- Atrial Pacing: spike followed by a P wave
- Ventricular Pacing: spike followed by a QRS complex

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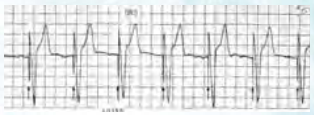
A Pacemaker Rhythm

- Pacemaker stimulus seen as a vertical spike
- Spike will precede the response of the heart



50

Terms used in the Interpretation of Pacemaker Rhythm

- Capture: pacemaker spike is followed by evidence of depolarization of the heart
  - Normal is "complete capture"
- Sensing: pacemaker detects intrinsic cardiac beats
  - Appropriate
  - Inappropriate: doesn't sense
    - Oversensing
    - Undersensing
- Rate: rate at which the pacemaker is firing

51

Interpreting a Pacemaker Rhythm

- Look for the "spike"
- Does spike precede a P wave or a QRS complex?
- Measure rate of pacemaker
  - Measure from spike to spike
- Is there appropriate function? Firing, Capture, Sensing
- Interpretation: Ventricular Pacing, rate with complete capture; Comment on sensing if sensing can be evaluated.

52

Pacemaker Malfunction

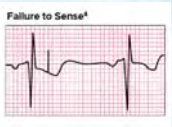
- **Failure to Capture**
  - Capture is the successful depolarization of the heart by the pacemaker
  - Capture is recognized by a pacer spike which is followed by the appropriate wave form
    - P wave if atrial pacing
    - QRS if ventricular pacing
  - Failure to capture (loss of capture) recognized as **pacer spikes not followed by a waveform**

Berberian JG. Pacing Rhythm Guide. 1st ed. Dallas, TX: Emergency Medicine Residents' Association; 2017.

53

Pacemaker Malfunction

- **Failure to Sense (Undersensing)**
  - Pacemaker spikes appear on the ECG when they should not; generator has failed to recognize intrinsic cardiac activity



Berberian JG. Pacing Rhythm Guide. 1st ed. Dallas, TX: Emergency Medicine Residents' Association; 2017.

54

Pacemaker Malfunction

- **Failure to Sense (Undersensing)**
  - Pacemaker spikes appear on the ECG when they should not; generator has failed to recognize intrinsic cardiac activity

Rounds, A. (2016). An uncommon cause of pacemaker-mediated ventricular tachycardia. *Journal of cardiovascular electrophysiology*, *27*(1), 107-109.

55

Pacemaker Malfunction

- **Failure to Sense (Oversensing)**
  - Results from generator sensing extraneous electrical signals (EMI) or misidentifies a T wave or P wave for the QRS and does not emit a stimulus.
  - Recognized by the absence of pacer spikes or pacing at a slower rate than preset interval. Can result in failure to fire….no pacer spikes seen.

56

Pacemaker Malfunction

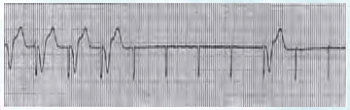
- **Failure to Fire**
  - Pacemaker fails to deliver an electrical stimulus or when it fails to deliver the correct number of stimuli per minute.
  - Recognized on ECG as **absence of pacemaker spikes** when they should be present
  - May result in bradycardia, syncope, chest pain, hypotension
  - Causes include battery depletion, electrode displacement, lead fracture, increased impedance, sensing problems

Berberian JG. Pacing Rhythm Guide. 1st ed. Dallas, TX: Emergency Medicine Residents' Association; 2017.

57

Pacemaker Rhythms

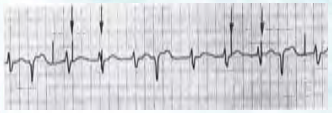
- Ventricular Pacing
  - Rate: 86 **with intermittent loss of capture** (pacer spikes not followed by a waveform)
  - Treatment: Report to MD Immediately; continue to monitor; anticipate TCP



58

Pacemaker Rhythms

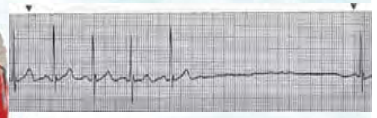
- Ventricular Pacing: Rate: ? with inappropriate sensing
  - Note pacemaker spikes in the QRS complexes)
- Treatment: Report Immediately to MD



59

Pacemaker Rhythms

- Situation: Lower Rate Limit of Ventricular Pacer: 62 bpm
- SR rate 79 with SNF (3.52 sec pause) with failure to fire
- Treatment: Report to MD immediately; anticipate TCP



60

Complications

- Temporary
  - Pain
  - Tissue damage if defibrillated
  - Failure to correctly ID pacer malfunction
  - Infection, bleeding
  - Pneumothorax
  - Perforation of ventricle
  - Instability of the system

61

Complications

- Permanent Pacing
  - Bleeding, Infection
  - Thrombosis at lead interface with heart
  - Electrode displacement, Lead fracture
  - Perforation of ventricle
  - Pacemaker Syndrome
  - Changes in impedance of lead
  - Battery depletion (life is 10 – 11 years with lithium battery)

62

Implantation

- Temporary
  - At bedside: transvenous, transcutaneous
  - Cardiac Cath Lab (CCL): transvenous
  - In surgery: epicardial
- Permanent
  - CCL
  - Surgery

63

Implantation

- Temporary
  - Local anesthesia
  - Battery external to body
- Permanent
  - Local/IVCS
  - Battery placed in pocket created beneath clavicle – R or L

64

Pacemakers

- Permanent
  - Subclavian vein to heart – RA &/or RV to position lead(s)
  - Pulse Generator positioned in a sub-clavicular pocket

65

Temporary

- Monitor site
- Frequent checking of connections
- Restriction of movement may be necessary
- Electrical precautions to prevent micro-shock
- Monitor ECG for appropriate pacer function
- Monitor patient response to pacing

66

Post-Implant

- Permanent
  - Initial period of bedrest (24 hrs) to allow lead to "settle in"
  - Initial restriction of movement of shoulder; wear sling; avoid heavy lifting; over head use of involved arm
  - Monitor site for hematoma, bleeding, infection
  - Monitor ECG for appropriate pacer function
  - Patient Teaching: follow-up; interference; ID card; Medic-Alert tag

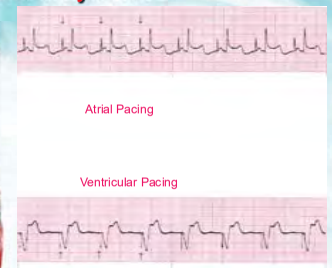
67

Post-Implant

- Follow-up: Check magnet rate; A decrease of 2 bpm indicates need for battery change
- Patient goes into VF and cardiac arrest.
  - Begin CPR; defibrillate as soon as defib available
  - Avoid placing electrodes/paddles over and/or near generator (hands-width away)
  - Pacemaker may malfunction post shock

68

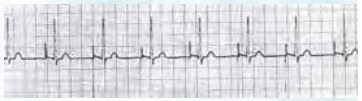
Pacemaker Rhythms



69

Pacemaker Rhythms

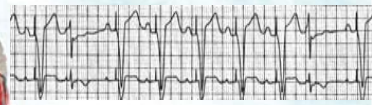
- Atrial Pacing
  - Rate: 58 with Complete Capture
  - Treatment: none; continue to monitor
  - Note: can't evaluate sensing-no intrinsic beats seen



70

Pacemaker Rhythms

- Interpretation: Ventricular Pacemaker
  - Rate: 79 with Complete Capture and appropriate sensing
  - Treatment: None; continue to monitor



71

Pacemaker Rhythms

- A-V Sequential Pacing (both atrial and ventricular spikes seen)
  - Rate: A = 56, V = 56
  - Complete Capture
  - Treatment: none; continue to monitor

72

Pacemaker Rhythms

- Interpretation:
  - A - V Sequential Pacing Rate: 63 with complete capture
- Treatment: none; continue to monitor

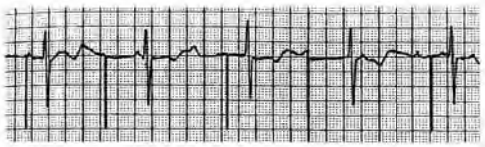
73

Pacer Malfunction

- Failure to Capture
  - Lead displacement or fracture
  - Sensing problems
  - Battery depletion

CASE STUDY: ER VISIT

A patient presented to the ER with the complaint that he felt just the way he did when he first received his pacemaker. What is your interpretation?



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ORDER A CHEST X-RAY

The chest x-ray revealed a dislodged lead

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Gather Information and Identify the Problem

Electrical stimuli delivered by the pacemaker does not initiate depolarization of the ventricle

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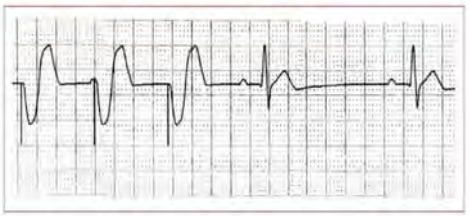
Gather Information and Identify the Problem



Pacemaker fails to emit stimuli at the programmed intervals

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ATRIAL ELECTROGRAM

Indications:

- When atrial activity id not clearly visible on the ECG.
- Trying to determine the relationship between the atrial and ventricular activity.
- Differentiate wide-complex rhythms (Vtach vs SVT)/
- Differentiate Narrow-complex SVT (Sinus Tach/Atrial Tach, PSVT, A Flutter, A Fib, Junctional Tach)

http://booksite.elsevier.com/9780323376624.

Atrial Electrocardiogram

- Take V lead with electrode and place over end of epicardial wire
- Then print out a strip with a limb lead and the V lead simultaneously
- Helpful with diagnosis of rhythm origin - atrial vs. junctional or ventricular

Figure 44-3 The tip of the atrial epicardial wire in direct contact with the conductive gel on the adhesive side of the electrode. (*Kern LS, McRae ME, Funk M. ECG monitoring after cardiac surgery: Postoperative atrial fibrillation and the atrial electrogram, AACN Adv Crit Care 18[3]:296, 2007.*)

Figure 44-4 An electrode wrapped around the tip of the atrial epicardial wire. (*Kern LS, McRae ME, Funk M. ECG monitoring after cardiac surgery: Postoperative atrial fibrillation and the atrial electrogram, AACN Adv Crit Care 18[3]:299, 2007.*)

Atrial ECG

References

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- Rounds, A. (2014). An uncommon cause of pacemaker-mediated ventricular tachycardia. *Journal of cardiovascular electrophysiology*, *25*(1), 107-109.
- Sternum-apex pacing electrode placement. In female patients, position the negative pacing electrode under the breast but above the diaphragm to prevent contraction of the diaphragm each time the pacer fires. (Modified from illustrations supplied by Physio-Control, Inc., Redmond, WA.)
- **Wesley: Huszar - Basic Dysrhythmias: Interpretation & Management, 4th ed.,** Copyright © 2011

Hemodynamic Monitoring

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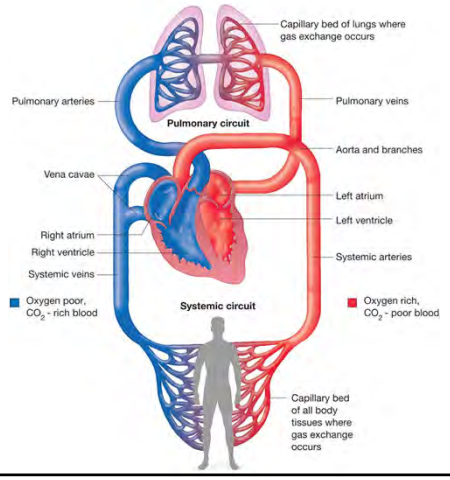
University of Arkansas for Medical Sciences

1

4 Factors that Affect the Hemodynamic Conditions

- Myocardial Contraction
- Heart Rate
- Intravascular Volume
- Vasoactivity



2

The main goal of Hemodynamic Optimization

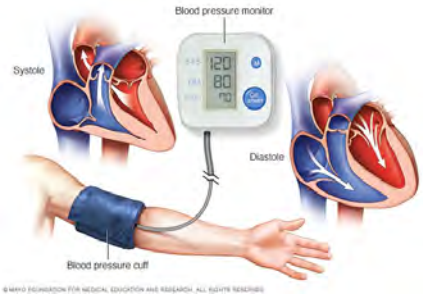
- Squeeze
- Resistance
- Volume
- Delivery
- Consumption

Perfusion

3

Noninvasive Hemodynamic Monitoring

- Noninvasive Blood Pressure
- Heart Rate
- Pulses
- Mental Status
- Skin Temperature
- Capillary Refill
- Urine Output



4

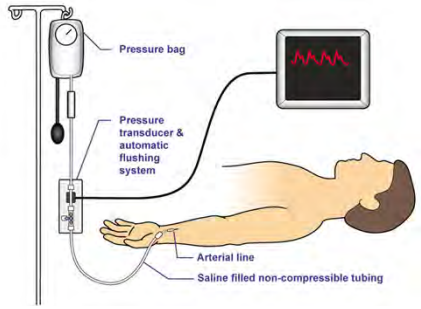
Invasive Hemodynamic Monitoring

Arterial Blood Pressure
Central Venous Pressure
Pulmonary Artery Pressure

5

Pressure System Set-Up

Pressure Bag
Fluids
Pressure Transducer



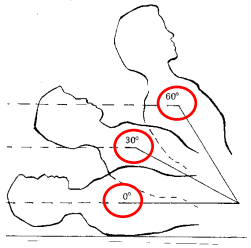
6

Phlebostatic Axis

4th Intercostal space, mid-axillary line
Level of the atria

7

Referencing the "zeroing" stopcock to Phlebostatic Axis



The phlebostatic axis is the approximate level of the left atrium. It is located midway between the anterior-posterior chest wall at the 4th intercostal space.
The patient need not be flat, but must be supine.

8

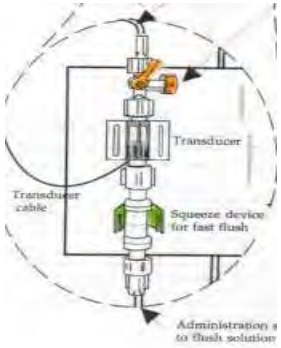
Accuracy

Zeroing transducer system to
negate atmospheric pressure
- Open air-reference port on transducer
- Push appropriate "button" on bedside monitor

Calibration to avoid electronic drift
- Rechecked q shift

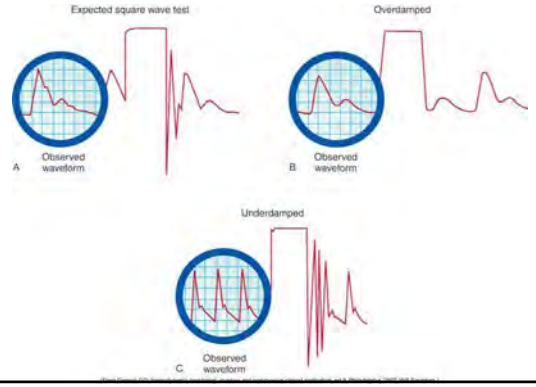
Maintaining continuous flush
- Fluid in flush bag
- Pressure bag at 300 mmHg

- **All readings are taken at end of expiration**



9

The Square Wave Test: 1.5-2 Oscillations

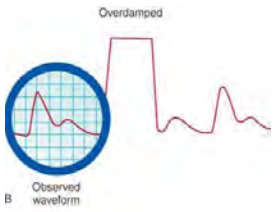


10

Overdampened: < 1.5 oscillations

Results in erroneously low SBP and high DBP

Causes:
- large air bubbles
- Loose/open connections
- Low fluid level in flush bag
- Pressure bag less than 300 mmHg

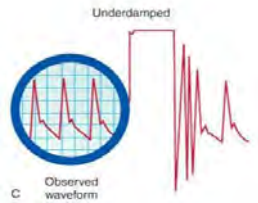


11

Under Dampened: > 2 oscillations

Results in erroneously high SBP and low DBP

Causes:
- Small air bubbles
- Tubing too long
- Defective transducer



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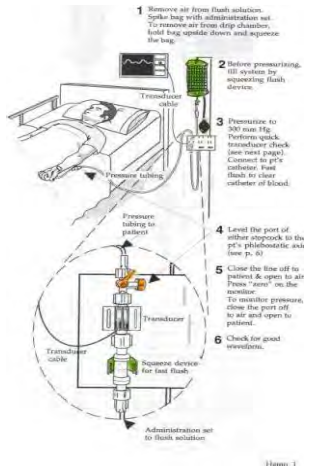

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Arterial Blood Pressure

Measuring Blood Pressure
- Direct: A-line
- Indirect: Cuff

Purpose
- to obtain direct arterial pressure readings of the systolic, diastolic, and mean pressures

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Arterial Line Indications

Closely Monitor Blood Pressure
- Vasoactive drips (dopamine, nipride, etc)

Frequent B/P measurements are needed

Frequent blood sampling indicated

Cardiac Output and other hemodynamic measurements. (Vigileo/Flotrac)

15

Components of Arterial Waveform

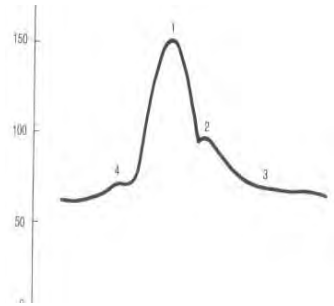
Peak Systolic Pressure

Dicrotic Notch

Diastolic Pressure

Anacrotic Notch

Pulse Pressure



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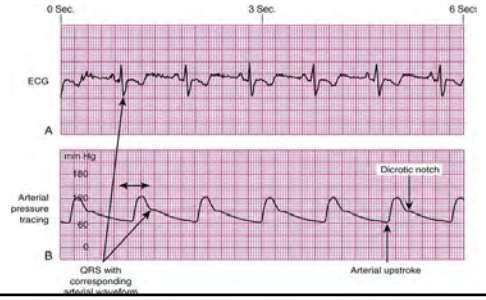
A-line Waveforms

A-line pressure waveform represents the ejection phase of left-ventricular systole



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Normal Pressures

Systolic: 90-140 mmHg

Diastolic: 60-90 mm Hg

Mean: 80-100 mm Hg

MAP = $\frac{S + 2D}{3}$ or D + PP

Example: 120/80 = 120 + 160 ÷ 3 = 93

Compared to indirect (Cuff Pressures)

18

Compare Aline Pressures to Cuff Pressures

A line Systolic----------130
Cuff Systolic----------125
Cuff Diastolic----------75

A line Diastolic ---------- 70

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General Nursing Care

Check adequacy of circulation q 2 hrs

Reference point: Phlebostatic Axis
Research: Tip of Catheter

Site care, tubing and solution change

Patient teaching

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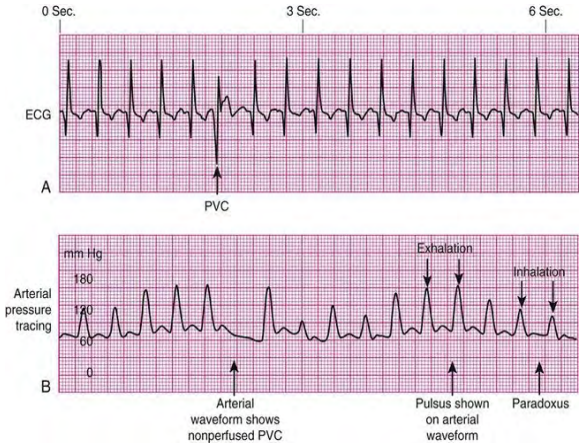
Complications

Absent or dimenished distal pulse
Hematoma after D/C
Local Infection
Sepsis
Dampened pressure tracing
Bleedback into the tubing

21



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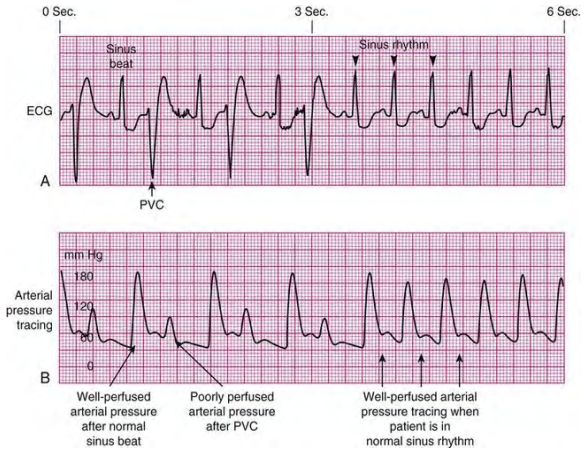

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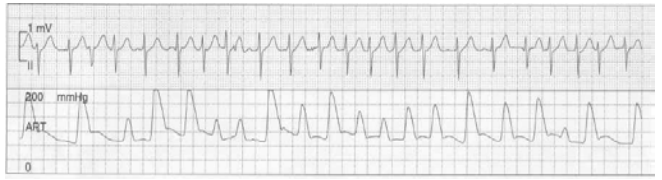

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Pulsus Paradoxus

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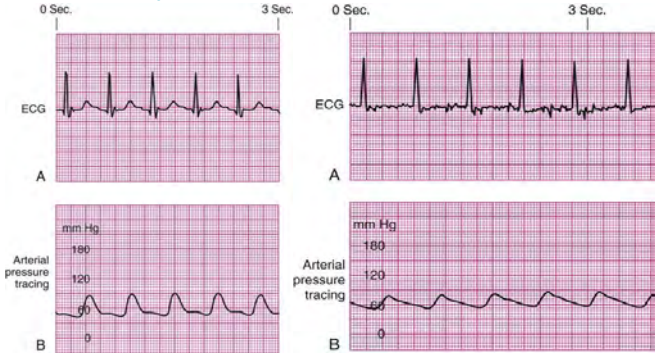
Low Pulse Amplitude or Dampened Wave Form



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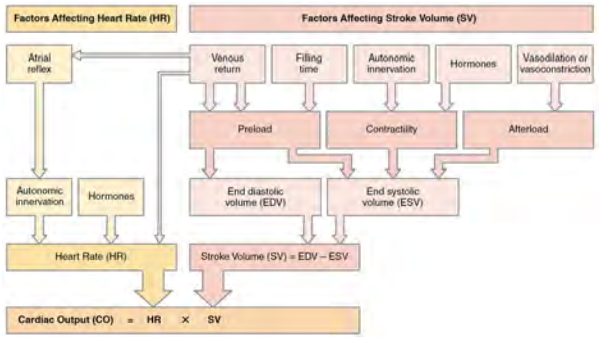
Cardiac Output



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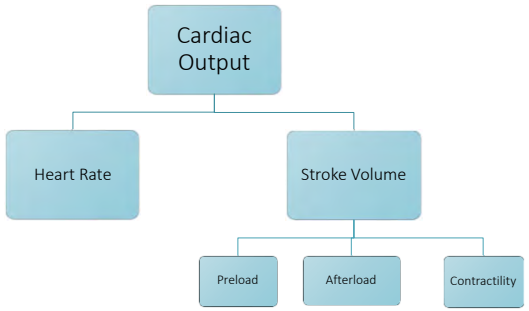
Cardiac Output



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Cardiac Output Terminology

Cardiac Output (CO):
- Amount of blood ejected from the ventricle in 1 minute
- Measured in Liters/minute
- CO = Heart Rate (HR) x Stroke Volume (SV)
- Normal CO: 4-8 L/min

Cardiac Index (CI):
- CO/BSA (body surface area)
- Normal CI: 2.5 – 4 L/min/M^2

Hemodynamic Definition of Shock: CI ≤ 2

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Cardiac Output Terminology

Stroke Volume:
- Difference between the end-diastolic volume (amount of blood in the ventricle at the end of diastole) and end-systolic volume (blood volume in the ventricle at the end of systole)
- Normal SV: 60 – 100 ml

Ejection Fraction:
- Stroke Volume expressed as a percentage of end-diastolic volume.
- Normal EF: 60-75%

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Preload

Amount of myocardial fiber stretch at the end of diastole

Central Venous Pressure (CVP)
- *Used to assess right ventricular preload*
- *Normal CVP: 2-6 mmHg*

Pulmonary Artery Wedge Pressure (PAWP)
- *Used to assess left ventricular preload*
- *Normal PAWP: 6-15 mmHg*

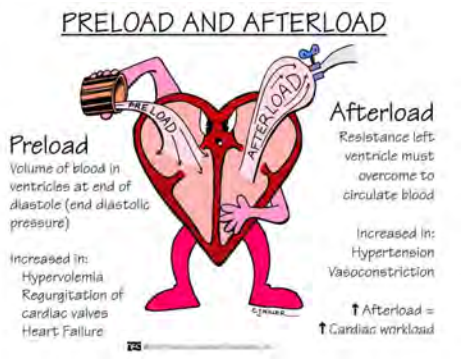
Frank-Starling Law:
- The more the diastolic volume or fiber stretches at the end of diastole, the stronger the next contraction during systole to a physiologic limit (up to a point)

32

Preload

CVP: Right Ventricular Filling Pressure reflects
- *Volume*
- *Ventricular Compliance*
- *Valve Integrity (Tricuspid)*

PAOP: Left Ventricular Filling Pressure reflects
- *Volume*
- *Ventricular Compliance*
- *Valve Integrity (Mitral)*



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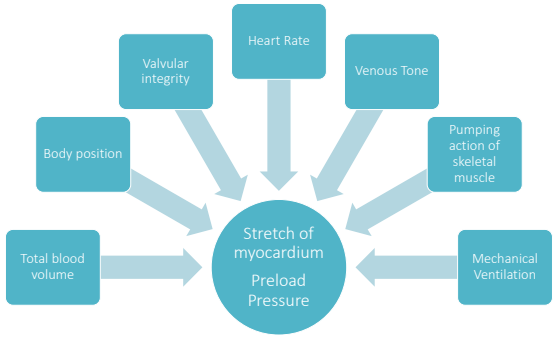
Preload – Filling the Ventricles Depends on



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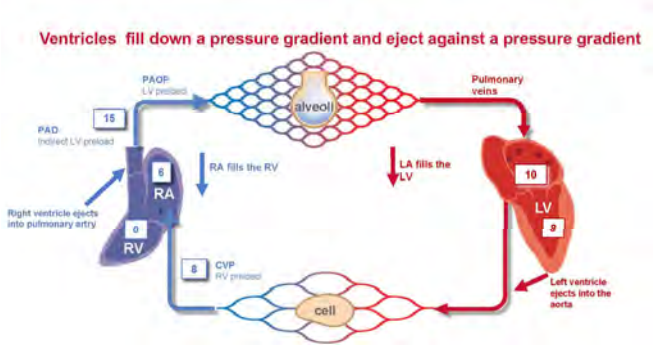
Preload



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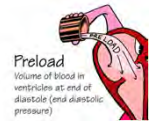
Preload



VOLUME

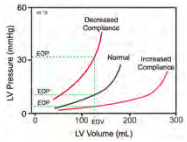
↑ filling volume = ↑ preload = ↑ stroke volume

↓ filling volume = ↓ preload = ↓ stroke volume

VENTRICULAR COMPLIANCE

Normal Compliance: easy to fill
Large ↑ volume = small ↑ pressure

Less-Compliant: harder to fill
Small ↑ volume = large ↑ pressure



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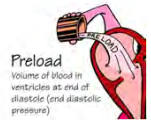
Preload can be affected by



Anything that changes circulating blood volume
(dehydration, hemorrhage, hypervolemia, etc)

Anything that changes the amount of blood returning to the heart
(vasoconstriction, vasodilation, etc)

Anything that changes the ventricular filling time or volume
(Heart Failure, cardiac tamponade, ↑ or ↓ heart rate)

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Decreased Preload

If preload is too low, causes include: dehydration, hemorrhage, hypovolemia, vasodilation, tachycardia (decreased filling time)

Symptoms include:

- **Tachycardia**
- **Decreased UO**
- **Chest Pain**
- **Cool, clammy skin**
- **Decreased BP**
- **Dizziness**

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Treatment for ↓ Preload

Fluids (most common)

Tx to slow heart rate and increase filling time
For SVT or VT

Vasoconstrictor (nor-epi, epi drips) If and only if the tank is full
Vasodilation due to
sepsis/ anaphylaxis, etc

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Increased Preload

POSSIBLE CAUSES:

- Fluid overload
- Hypervolemia
- Vasoconstriction
- Heart failure
- Possibly bradycardia, exercise

SIGNS/SYMPTOMS:

- CVP > 8
- Decreased CO
- Dist. Neck Veins
- Hepatojugular Reflux
- Weight gain
- Peripheral edema

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Hepato-jugular Reflex

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Preload Right Side of Heart: CVP (normal 2-6)

LOW	HIGH
Hypovolemia	Right Ventricular Dysfunction
Tachycardia	Pulmonary Hypertension
	Tricuspid Valve Dysfunction
	Volume Overload

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Preload Left Side of Heart: PAOP (normal 6-15)

LOW	HIGH
Hypovolemia	Left Ventricular Dysfunction
Tachycardia	Increase in Pulmonary Venous Blood Volume
	Decrease in Left Ventricular Compliance
	AV or Semilunar Valve Stenosis/Regurgitation

PA Diastolic reflects the PAOP

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↑ Preload: PAWP & CVP

↑ PRELOAD: PAOP & CVP

- Right sided failure, ↑ CVP
  - CVP 18
- Left sided failure, ↑ PAWP
  - PAWP 25
- Arterial Vasoconstriction
  - Hypertension, ↑ SVR

↑ PRELOAD: PAOP

- Blood pools in lungs
  - Pulm. Edema
  - SOB
  - Pink, frothy sputum
- ↓ Urine Output
- Tachycardia: Compensation
- Chest pain: ↑ Work

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Treatment of ↑ Left Preload

Diuretic to ↓ circulating volume

Medication to vasodilate to trap circulating volume in the periphery (NTG)

Positive Inotrope: increase strength of contraction (dobutamine)

Stop negative Inotropes (stop Ca++ Channel blocker, Beta Blockers, etc)

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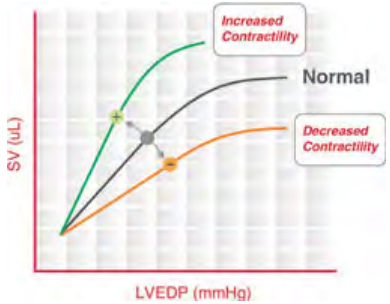
Preload Responsiveness

The bigger the stretch,
the bigger the
contraction,
up to a point.



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Stroke Volume and Preload Responsiveness

300 -500 ml of saline=

Stroke Volume Variation > 15% = Preload Responsive
SV = 60 ml; after Fluid bolus SV = 90 ml

SVV % < 10% ≠ Preload Responsive
SV = 60 ml; after Fluid bolus, SV = 64 ml

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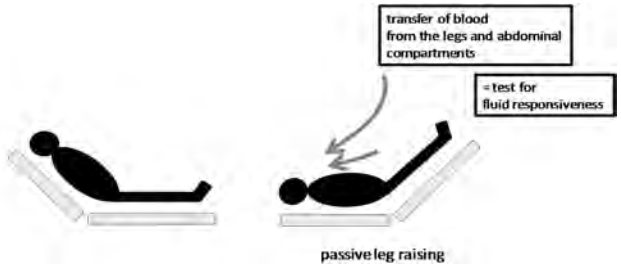
Approx. 300 ml challenge = ↑ SV in 90 sec



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Not measuring Stroke Volume?

Look at pulse pressure

Systolic – diastolic = Pulse Pressure

Normal is 30-40 mm Hg

↑ in Pulse Pressure = ↑ in stroke volume

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SVV Normal < 13%

Limitations

- Currently, only supported by literature if
- Pts are 100% mechanically ventilated in Control Mode with tidal volumes of more than 8 ml/kg and fixed respiratory rates, in sinus rhythm
- Not supported on patients with spontaneous ventilation
- Arrhythmias and PEEP can dramatically affect SVV values

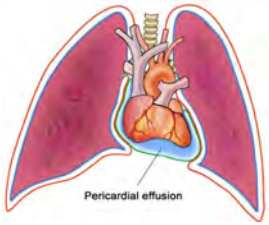
50

Ventricular Distensibility

Ability of heart to stretch and accommodate a volume of blood

Decreased by anything that

- Makes a ventricle stiff
  MI, hypertrophy, Old Age, Contusion
- Cardiac Tamponade
- Pericardial Constriction



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Afterload

Tension developed by the myocardial muscle fibers during ventricular systolic ejection

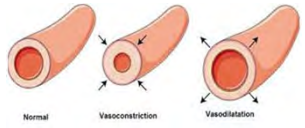
Described as resistance, impedance, or pressure that the ventricle must overcome to eject its blood volume

Systemic Vascular Resistance (SVR)

- *Most sensitive measure of afterload for the left ventricle*
- *Normal: 800-1200 dynes*

Pulmonary Vascular Resistance (PVR)

- *Most sensitive measure of afterload for the right ventricle*
- *Normal: 100-250 dynes*



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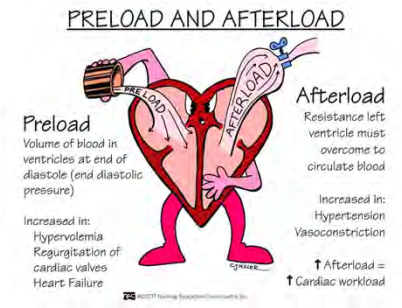
Afterload

Afterload has an inverse relationship to ventricular function

As resistance ↑, the force of contraction ↓ = ↓ stroke volume.

As resistance ↑, myocardial oxygen consumption ↑



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SVR & PVR

SVR

$$\frac{MAP - CVP}{CO} \times 80$$

PVR

$$\frac{MPAP - PAWP}{CO} \times 80$$

MAP: Mean Arterial Pressure

MPAP: Mean Pulmonary Artery Pressure

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Afterload Affected By

Anything that ↑ or ↓ vascular resistance
- Vasoconstriction, vasodilation, IABP

Anything that affects
- the aortic valve or aorta on the left
  - *Aortic stenosis*
  - *Aneurysm*
  - *Miss-timed IABP*
- Pulmonic valve or pulmonary artery on the right

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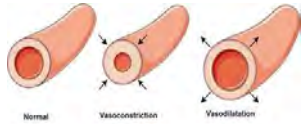
Afterload



HIGH – VASOCONSTRICTION
- Hypertension
- Vasopressors
- Aortic Stenosis
- Hypothermia
- Pulmonary Hypertension
- Hypoxia
- PE
- Pulmonary Stenosis

LOW - VASODILATION
- Distributive Shock states
- Vasodilators
- IABP

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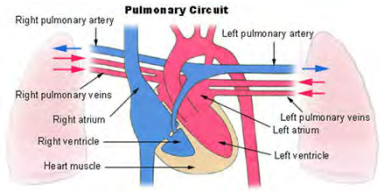
Right Ventricular Afterload (PVR)

HIGH

- Pulmonary Embolism
- Pulmonary hypertension
- Left ventricular failure

LOW

- Lysis of Pulmonary Embolism
- Treatment of ↑ PVR



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Left Ventricular Afterload (SVR)

HIGH

Vasoconstriction
- Vasopressors
- Hypertension
- Compensatory mechanism for ↓ CO states (hypovolemia/cardiogenic shock)

Aortic Valve Stenosis/ aortic aneurysm

Miss-timed IABP

LOW

Vasodilation
- Nipride /NTG drips

Distributive Shock States
- Anaphylaxis
- Sepsis
- Neurogenic

Intra-Aortic Balloon Pump

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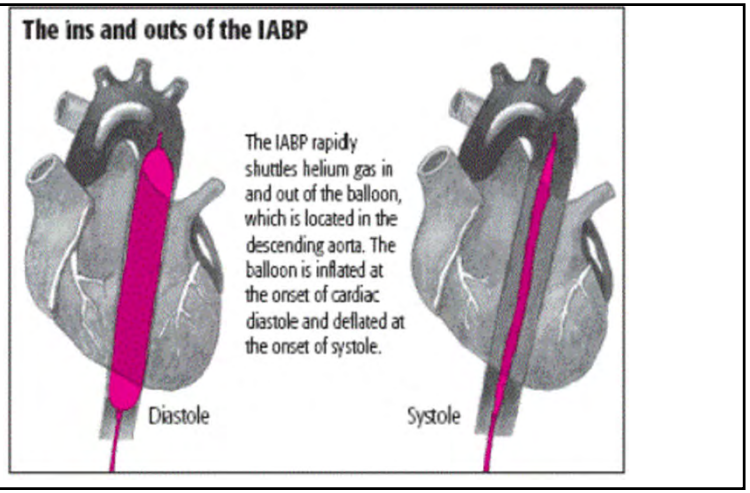

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Treatment of High Afterload

Medication to vasodilate

Surgery to correct aortic stenosis/ aneurysm

↑ is due to ↓ CO, treatment is aimed at ↑ CO (positive inotrope, etc)

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Treatment of Low Afterload

Medications for vasoconstriction

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Contractility

Inotropic state of the myocardium

The velocity and extent of myocardial fiber shortening

Parameters that reflect contractility include

- *Stroke Volume*
- *Stroke Volume Index*
- *Left ventricular stroke work index*
- *Right ventricular stroke work index*

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Contractility

Pumping ability of the heart

Estimated by Stroke Volume

SV = 60-130 ml

SV = CO ÷ HR

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Contractility

HIGH

- Positive inotropic drugs
  - Dobutamine
  - Dopamine
  - Digoxin
- Calcium
- Increased preload
  - The greater the muscle stretch, the greater the contraction, up to a point: Frank-Starling Law

LOW

- Negative inotropic drugs
  - Ca++ Channel Blockers
  - Beta Blockers
- Acid/base imbalance
- Hypoxemia
- Electrolyte imbalance

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Signs & Symptoms

Too much contractility: chest pain, tachycardia

Too little: Heart Failure, Pulmonary Edema, decreased perfusion

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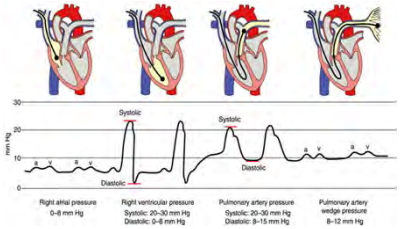
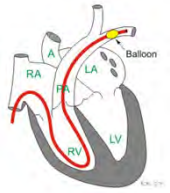
Pulmonary Artery Pressure Monitoring

Provides information about vascular capacity, blood volume, pump effectiveness, and tissue perfusion





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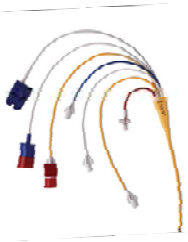
Pulmonary Artery Pressure Monitoring

Pulmonary Artery Pressure (PAP) is measured by a pulmonary artery catheter

Indirectly reflects left ventricular end-diastolic pressure (LVEDP), which indicates left ventricular function

Location	Color	Function
Distal	Yellow	Monitors PA pressures
Proximal	Blue	Monitors RA pressures (with manual C.O. monitoring, it is used for the C.O. injectate fluid)
Balloon Gate Valve	Red	Syringe used to inflate balloon for placement and obtaining wedge values
Thermistor Connector	White/Red	Measures blood temperature 4cm from the distal tip
Venous (VIP)	White	Additional RA lumen for fluid infusion
	Blue	Measures the SvO_2



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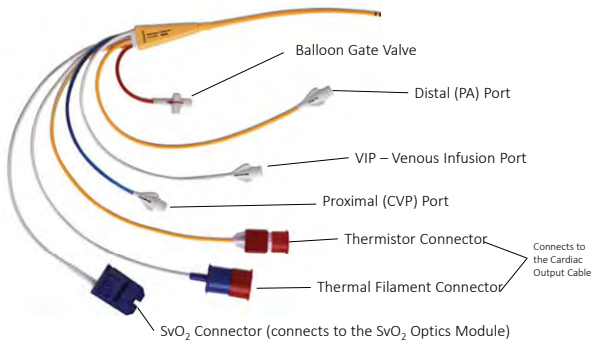
Pulmonary Artery Catheter Components



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Indications for PA catheter use

- Intra-abdominal hypertension
- Patients at risk for acute right ventricular dysfunction
- ARDS
- Extensive burns
- Cardiac surgery
- Significant cardiac tamponade
- Significant cardiomyopathy
- Significant constrictive pericarditis
- Drug intoxication
- Severe eclampsia
- Significant intra- or extra-vascular fluid shifts
- At risk for hemorrhage
- Intra- and post-op high risk surgery management
- Patient on intra-aortic balloon counterpulsation
- Complex liver resections
- Liver transplantation
- Complex lung resection
- Complex myocardial infarctions
- Pulmonary edema
- Pulmonary hypertension
- Acute renal failure
- Severe sepsis
- Presence of or at risk for: cardiogenic, distributive, hemorrhagic, or obstructive shock
- Shock of unknown etiology
- Shock unresponsive to attempts at resuscitation
- Severe trauma
- Ventilator effects on hemodynamics

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Relative Contraindications for PA catheter use

Left bundle branch block

Patients with tricuspid or pulmonic heart valve replacements

Lack of appropriate clinical skills or infrastructure to insert and/or support the use of a pulmonary artery catheter

Heparin coated catheters in patients with known sensitivity to heparin (HIT) - Ensure catheter is Heparin-free for such patients

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Pulmonary Artery Pressure Monitoring

Swan – Ganz Catheter

Distal Tip

Measures PAP & PAWP

SVO2 blood specimens can be obtained from this port (when obtaining SVO2 sample, be sure to draw the sample SLOWLY)

Do NOT administer medications in this port!

Proximal Port

Continuous infusions of fluids or medications

CVP measurements

May use stopcock, and use line for both medication administration and CVP measurement

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Pulmonary Artery Pressure Monitoring

Normal Readings

CVP

2-6 mmHg (or 3-8 cm H2O)

PAP

Systolic 20-30 mmHg

Diastolic 10-20 mmHg

Mean 10-15 mmHg

PAWP

6-15 mmHg (should be 2-5 mmHg less than PA diastolic pressures)

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Preparation for Insertion of Pulmonary Artery Catheter

Prepare Equipment
- Turn on Vigilance monitor (10-15 min)
- Get appropriate PA catheter (fiberoptic, thermodilution, etc) & cables
- Introducer kit (venous sheath)
- Set up pressure monitoring system (for PA and CVP lumens)
  - *500 ml Normal Saline x 2*
    - Remove air from bag
  - *Pressure Tubing and transducer x 2*
  - *Pressure Bag x 2*
  - *Pressure Cable x 2*

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Preparation for Insertion of Pulmonary Artery Catheter

Zero & Calibrate bedside monitor

Check balloon integrity

Assist MD with introducer insertion

Calibrate fiberoptics (SvO_2) – In Vitro

PA Catheter – distal port is attached to pressure line

The MD inserts the PA Catheter thru introducer into vena cava. As it enters right atrium, the CVP waveform and pressures will appear in the PA pressure area on the monitor. The MD requests that the balloon be inflated. The balloon acts like a sail to "float" the tip of the catheter down stream to the pulmonary artery.

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Preparation for Insertion of Pulmonary Artery Catheter

Catheter advancement to the pulmonary artery should be rapid, since prolonged manipulation can result in loss of catheter stiffness

Common sites for percutaneous approach include:
- Internal jugular
- Subclavian vein
- Femoral vein

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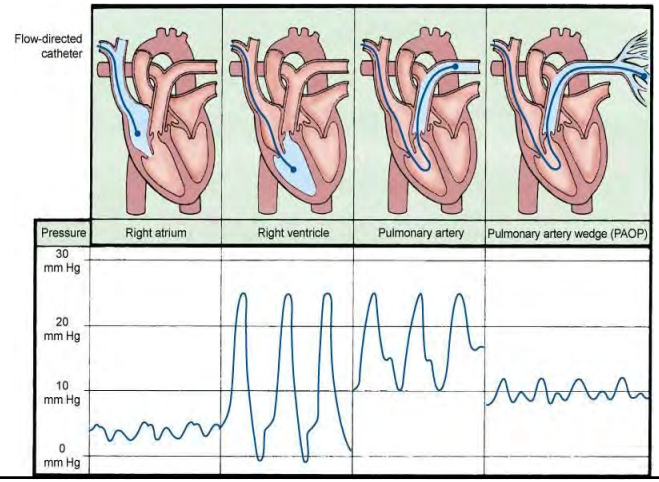

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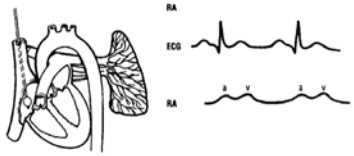
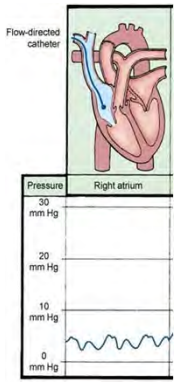
Insertion of Pulmonary Artery Catheter: Right Atrium

The first chamber reached is the right atrium

Pressures are usually low and will produce 2 small upright waves.



CVP: 2-6 mmHg



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Insertion of Pulmonary Artery Catheter: Right Ventricle

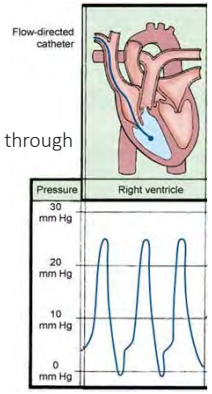
The next chamber is the right ventricle

Waveforms show taller, sharp uprises and low diastolic dips

Special attention must be paid to the ECG once the catheter passes through the tricuspid valve

Ventricular ectopy may occur

Right Ventricle:
Systolic 20-30 mmHg
Diastolic 0-5 mmHg
Mean 2-6 mmHg



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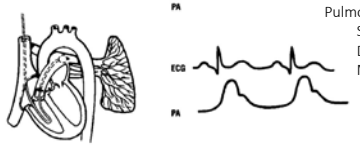
Insertion of Pulmonary Artery Catheter: Pulmonary Artery

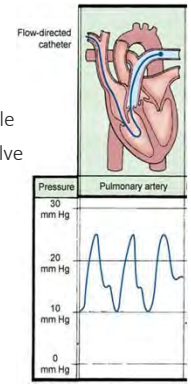
As the catheter floats into the pulmonary artery, characteristic waveforms can again be noted

There is a rise in pressure in the pulmonary artery, especially diastole

A dicrotic notch should be visible due to closure of the pulmonic valve



Pulmonary Artery:
Systolic 20-30 mmHg
Diastolic 10-20 mmHg
Mean 10-15 mmHg



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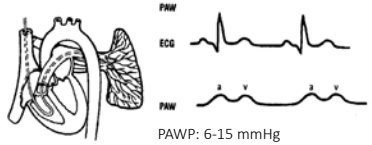
Insertion of Pulmonary Artery Catheter: Wedge

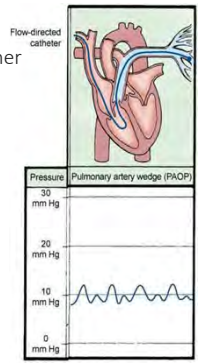
The catheter (with the balloon still inflated) is now advanced further until it finally wedges in a central branch of the pulmonary artery.

Waveform seen is a reflection of the left atrium

The waveform will have 2 small rounded excusions from left atrial systole and diastole



PAWP: 6-15 mmHg



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Insertion of Pulmonary Artery Catheter

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Pulmonary Artery Catheter Insertion Follow-up

Chest X-ray for placement

Secured by MD with sutures

Sterile dressing

Daily chest x-ray to assess placement

82

Caring for the patient with a PA catheter

Read pressures at the end of expiration (intrathoracic pressure changes from breathing, mechanical ventilation, or PEEP/PS will alter PAP and PAWP)

Keep in mind that right ventricular pressure readings are obtained only during catheter insertion

Normal:

Systolic: 20-30 mmHg

Diastolic: 0-5 mmHg

Mean: 2-6 mmHg

Level the transducer with the phlebostatic axis

83

Caring for the patient with a PA catheter

Remove all air from the tubing and transducer while setting up and monitoring

Air will dampen or distort waveform and can cause an air embolus

Use pressurized normal saline flush solution (500 ml bag) to maintain patency and normal waveform transmission

Inflate the balloon slowly while observing waveforms for only to the point that occlusion (wedge) is obtained.

Do not exceed 1.5 ml when inflating balloon

Keep the balloon inflated no longer than necessary (less than 15 seconds or 2-3 respiratory cycles)

Keeping balloon inflated too long could result in pulmonary infarction

84

Caring for the patient with a PA catheter

Allow the balloon to passively deflate to increase balloon longevity and avoid balloon rupture
- Aspirating air to deflate the balloon can cause the balloon to lose elasticity

Avoid using the PA port for routine blood sampling
- Only use for mixed-venous sampling
- Routinely using port may increase the risk of clotting

Never infuse vasoactive drugs through the proximal port of the PA catheter
- Inconsistency with the infusion due to frequent flushing and placing on hold for CVP measurements

85

Caring for the patient with a PA catheter

Never hep lock the proximal port of the PA catheter

Notify the MD if waveform shows a permanent PAWP reading, right ventricular pressure tracing, or PAWP outside given parameters

When inflating the balloon, you should feel slight resistance
- If you feel marked resistance or no resistance, stop inflating immediately (lack of resistance suggests balloon rupture)

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Complications

Arrhythmias: (especially ventricular irritability) may result from the catheter irritating the ventricular endocardium or the heart valves
- Treatment includes administering medications (Lidocaine, amiodarone) as ordered by MD; also keep defibrillator nearby
- The MD can prevent this during insertion by keeping the balloon inflated when advancing the catheter through the heart

Hemorrhage (bleeding at the insertion site):
- Treatment includes applying firm pressure until bleeding stops
- To prevent, maintain pressure on the site during catheter withdrawal and for at least 10 minutes afterward, and apply pressure dressing over the site

Infection:
- To prevent, maintain sterile technique, apply topical antibiotic ointment, as policy permits
- Catheter should not exceed 72 hours if possible (catheter may be changed over wire one time only)
- If catheter will be indwelling for > 48 hours, prophylactic antibiotics are recommended

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Complications

Thrombi, emboli (air, blood): a thrombus migrates from the catheter into the pulmonary circulation or a catheter tip clotted from inadequate flushing
- To prevent, use a continuous flush system
- Signs/symptoms include: sharp and stabbing chest pain, anxiety, cyanosis, dyspnea, tachypnea, and diaphoresis
- Try to aspirate blood, but do NOT irrigate if you suspect embolus. If you can't aspirate blood, a P.E. may be obstructing the line. Notify the MD at ONCE
- If suspect emboli (air), turn patient to his/her left with head down and administer oxygen

Cardiac Perforation

Electrical Microshocks: the patient is electrically sensitive with the catheter in place; a catheter entering the heart increases the risk of micro-shock, producing ventricular fibrillation
- To prevent, make sure that all electrical equipment is grounded. Do not touch the patient and a piece of electrical equipment at the same time.

Complete Heart Block

88

Complications

Pulmonary Infarction: tip migration with spontaneous wedging, air embolism, thromboembolism, and prolonged balloon inflation can lead to this.

- Signs/Symptoms: chest pain, hemoptysis, fever, pleural friction rub, low arterial oxygen levels, and dyspnea
- Do NOT flush catheter
- Monitor PAP continuously and notify MD

Pulmonary Artery Rupture: can be contributed to pulmonary hypertension, thrombus, catheter migration into the peripheral branch of the artery, or improper inflation or prolonged wedging

- Signs/Symptoms include: restlessness, tachycardia, hypotension, hemoptysis, and dyspnea
- Notify the MD immediately
- Preventive measures:
  - *slowly inflate balloon and let balloon deflate passively*
  - *NEVER over inflate balloon*
- Other associated factors: advanced age, cardiac surgery with hypothermia and anticoagulation

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Complications

Heparin-induced Thrombocytopenia: if patient has Heparin antibody, then the patient will develop heparin-induced thrombocytopenia.

- The catheters are available with or without the Heparin coating. (To tell which one the catheter is, look at the number on the box; If there is an H in the number, then it has heparin coating.)

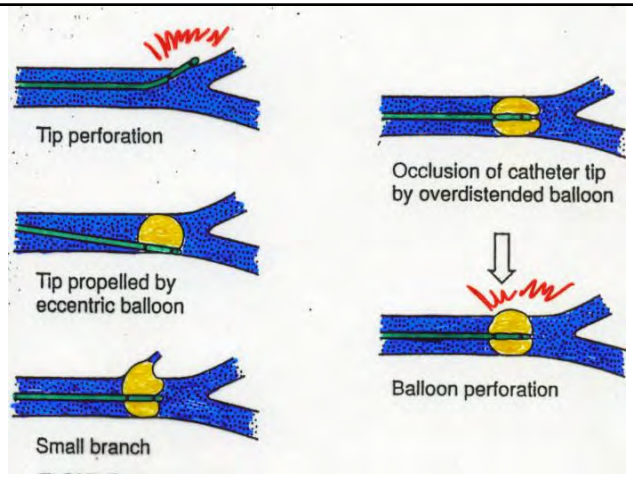
Bleedbacks: caused by leaks in the PA catheter apparatus or a pressure bag that is inflated below 300mm Hg

- Preventive measures include returning stopcocks to their proper position after use and keeping the pressure bag adequately inflated

Kinking, Looping, and Knotting

Tricuspid and Pulmonic Valve Damage

90



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Abnormal Waveforms

No Waveform on monitor:

- Reasons include: transducer is not open to the catheter, the transducer or monitor has been set up improperly, or a catheter is clotted
- Corrective measures: checking the stop-cock, calibration, or scale mechanisms; tightening connections; re-zeroing the setup; or replacing the transducer

Overdamped waveform:

- Causes can include air bubbles, blood clots, or a catheter tip lodged in the vessel wall
- Corrective measures: removing air bubbles; restore patency to a clotted catheter by aspirating clot with a syringe; try moving a lodged catheter by repositioning the patient or by having him cough and breathe deeply.
- NEVER irrigate the line as a 1st step!!

Changed waveform configuration: (noisy or erratic waveforms)

- May result from incorrectly positioned catheter, loose connections in the setup, or faulty electrical circuitry
- Corrective measures: reposition the patient, arrange for a CXR to verify catheter location, or check and tighten connections in the catheter and transducer apparatus

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Abnormal Waveforms

Catheter fling: (erratic waveform reflecting excessive catheter movement)
- May result from an arrhythmia or excessive respiratory effort

False pressure readings:
- Pressure readings that are either inaccurately too high or too low
- To correct, reposition the transducer –make level with the phlebostatic axis, or re-zero the monitor setup

Right ventricular waveform: (pulm. artery catheter migrating into the right ventricle)
- Inflate the balloon with 1.5 cc of air to move the catheter back to the pulm. artery (MD)

Continuous PAWP waveform: (catheter may migrate or the balloon may remain inflated)
- Reposition the patient, have patient cough and breathe deeply

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Abnormal Waveforms

Missing PAWP waveform:
- Mal-positioned catheter, insufficient air in the balloon, or a ruptured balloon could be the causes
- Treatment includes:
  - *Reposition the patient (don't aspirate the balloon)*
  - *Reinflate the balloon adequately (1st remove the syringe and let the balloon deflate passively)*
  - *Assess the balloon's competence (resistance??—lack of resistance could be balloon rupture)*
  - *If the balloon has ruptured, turn the patient onto his/her left side, tape the balloon inflation port, and notify the MD*

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Online Resources

https://education.edwards.com/series/icu#

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Hemodynamic Applications

CVP 1 PAWP 6 BP 90/60 SVR 1400

Possible DX; Treatment:

CVP 1 PAWP 6 BP 90/60 SVR 400

CVP 18 PAWP 25 CI 2.1 SVR 1500
BP 160/95

CVP 8 PAWP 10 CI 5 SVR 400

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Hemodynamic Applications

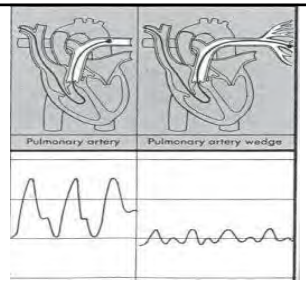
You would expect a Dobutrex (dobutamine) drip to increase_____

You would expect a Nipride drip to decrease _____

How can you judge if the CO is adequate for your patient?

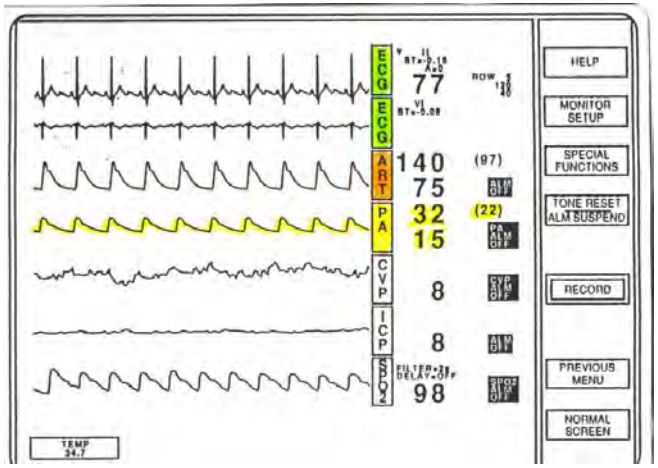
97

What pressure usually estimates the PAWP?



What landmark estimates the level of the right atrium?

98



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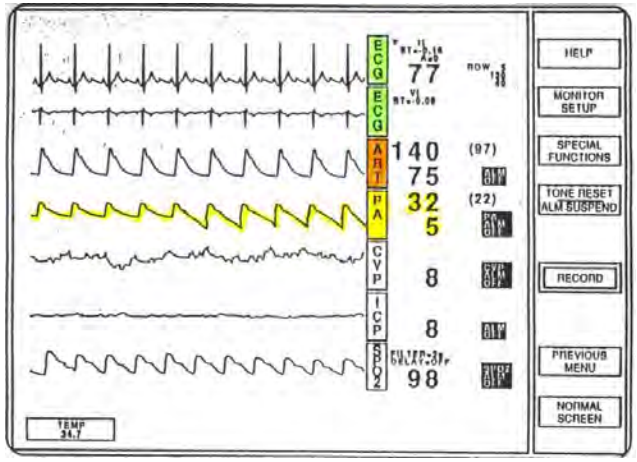

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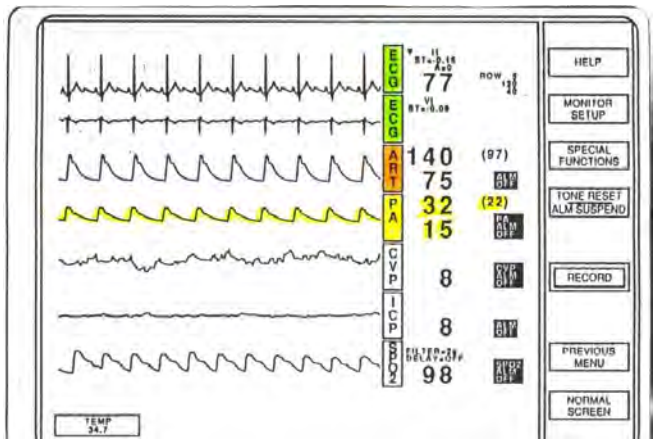

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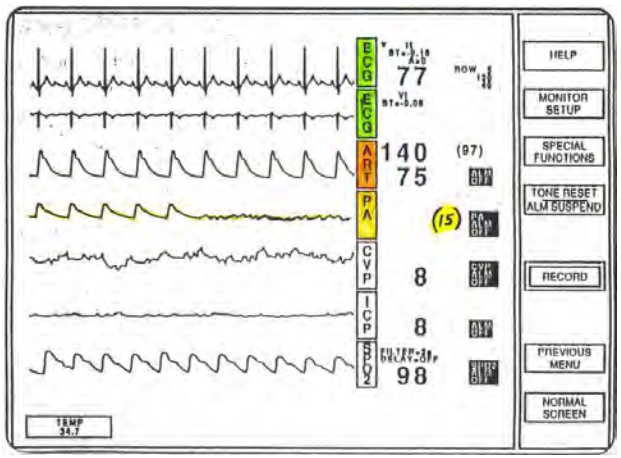

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Review

Hemodynamic definition of Shock?

103

Review

Hemodynamic definition of Shock?

Cardiac Index (CI) ≤ 2

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Review

Increase Preload with:

105

Review

Increase Preload with:

↓ circulating volume: fluids

Vasodilitation: fluids, vasoconstrictor drip

106

Review

Decrease Preload with:

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Review

Decrease Preload with:

Diruretic (Lasix), Vasodilator (NTG), + Inotrope

108

Review

Hemodynamic definition of Heart Failure?

109

Review

Hemodynamic definition of Heart Failure?

PAWP 25

CVP 18

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Review

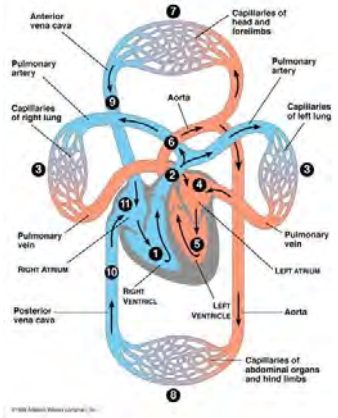
What happens to CO (and/or CI) with Sepsis/ SIRS?

Why is morphine the drug of choice for pain relief for MI?

- Preload?
- Afterload?
- Work of Heart?
- Oxygen consumption of heart?
- Anxiety (sympathetic nervous system)?

111

Questions?



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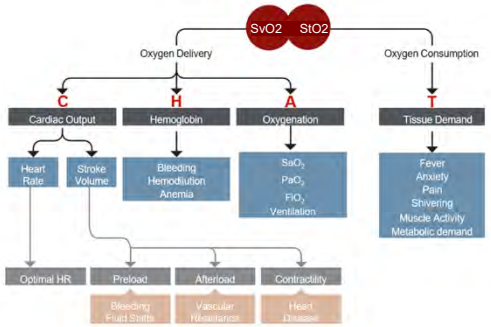
The Balance of Oxygen Delivery & Oxygen Consumption

Let's CHAT

C - CO
H - Hgb
A - Arterial Sats
T - Tissue Demand

Traditional vital signs may be late indicators of compromised or inadequate oxygen delivery to the tissues in the critically ill. The goal of continuous SvO2/StO2 monitoring is to bring into balance the relationship between oxygen consumption and oxygen delivery to improve the care of high-acuity patients.



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