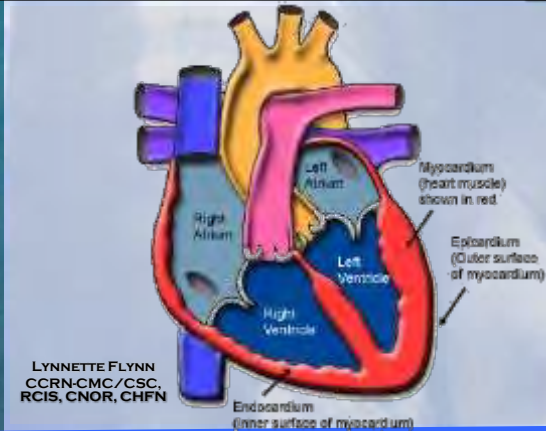


Cardiac Anatomy



1

1

Objectives

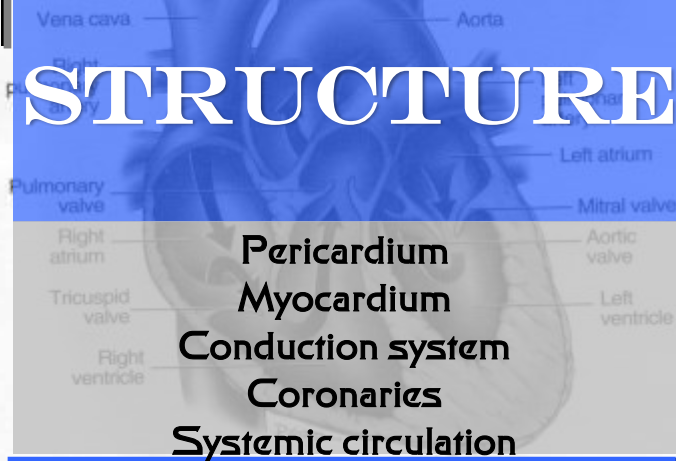
- Review of **basic anatomy**, cardiac cycle and conduction system
- Explore the various elements that control the CV system including intrinsic and extrinsic mechanisms.
 - Autonomic Nervous system
 - Sympathetic
 - Parasympathetic
 - Renin Angiotensin Aldosterone System
- Analysis of the **action potential** to understand the action of antiarrhythmics by class and Calcium effects
- Relate the mechanics of **myocardial contraction** to explain various medications effects



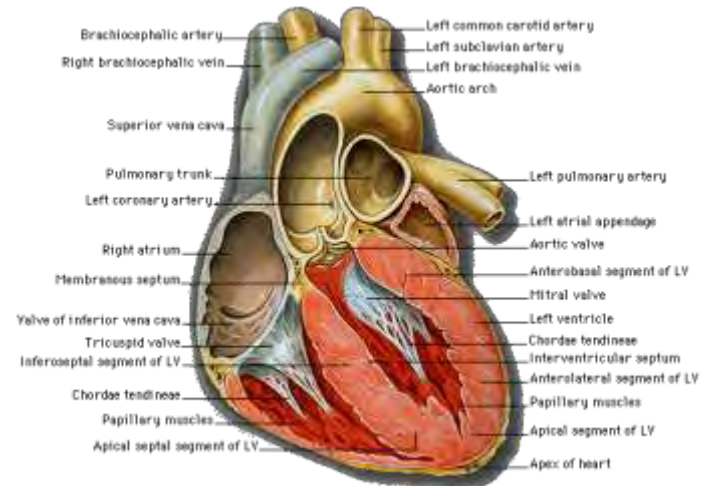
2

2

STRUCTURE

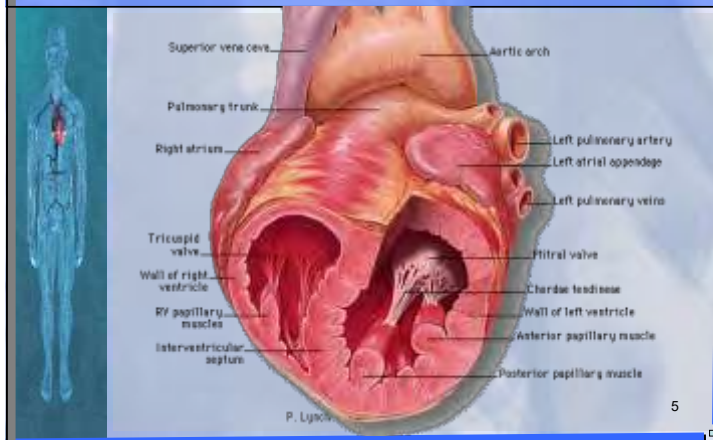


3



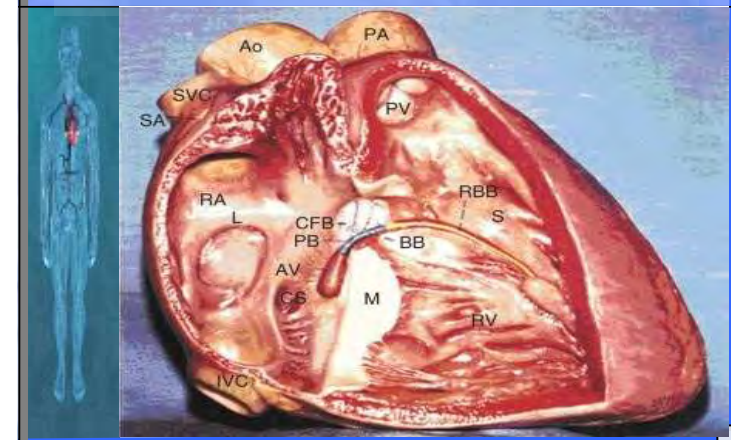
4

Papillary Muscles



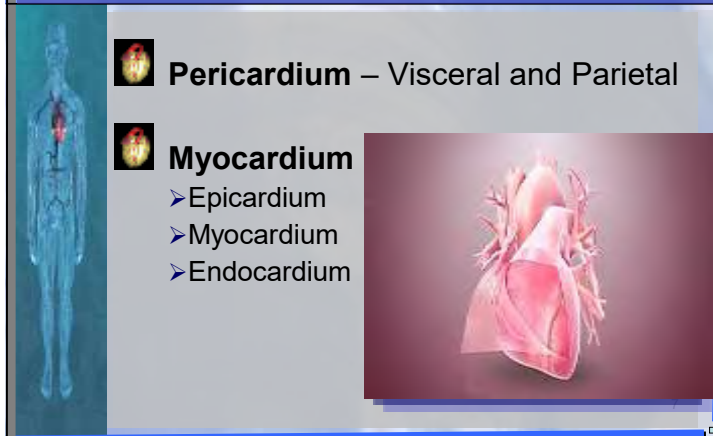
5

Myocardial Layers



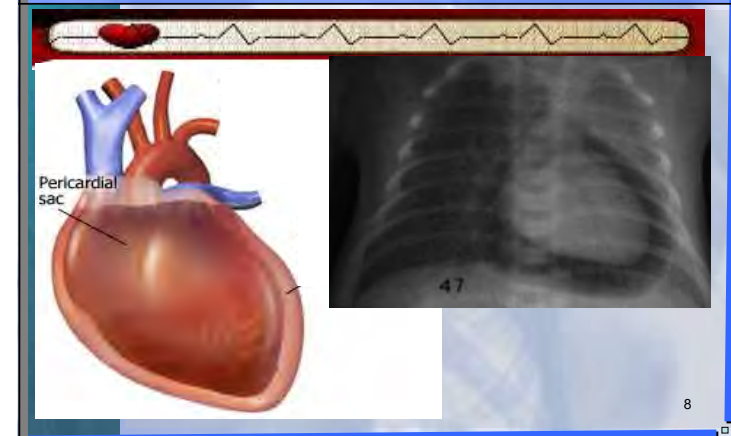
6

Heart Layers

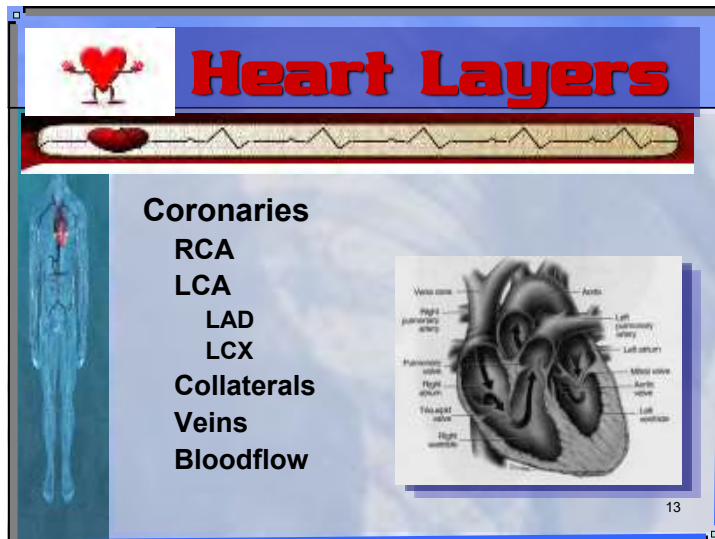


7

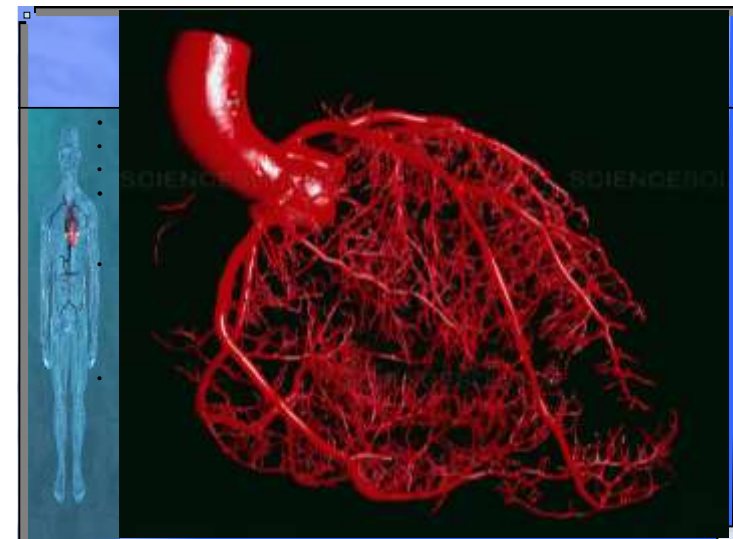
Effusion



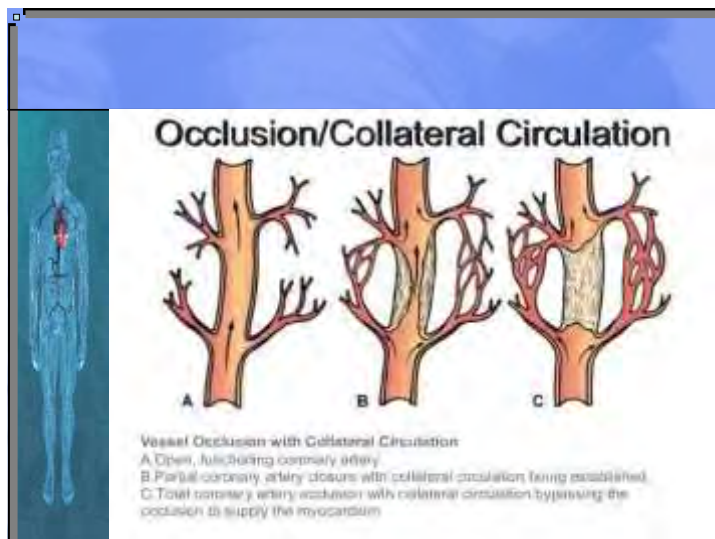
8



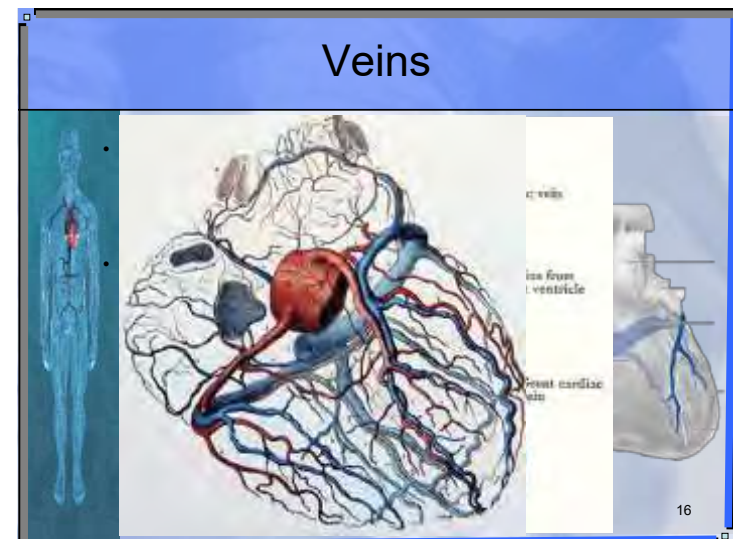
13



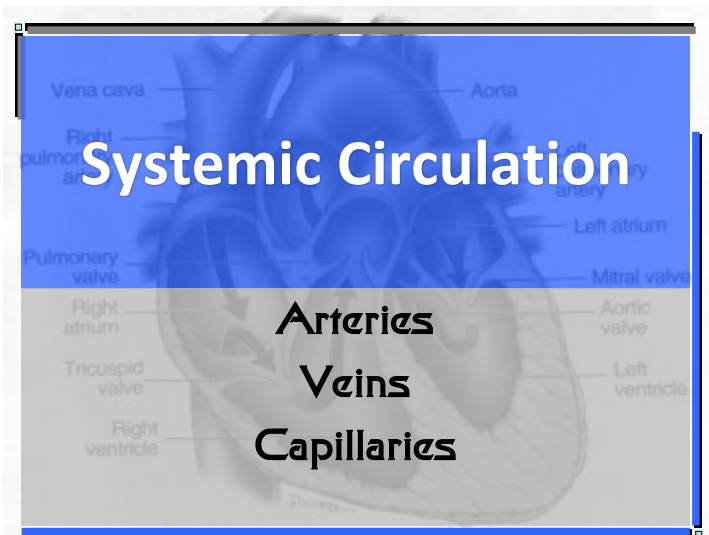
14



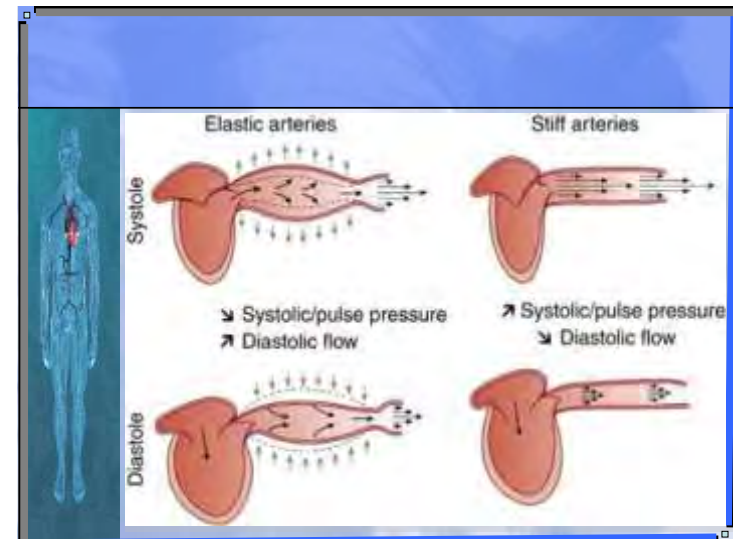
15



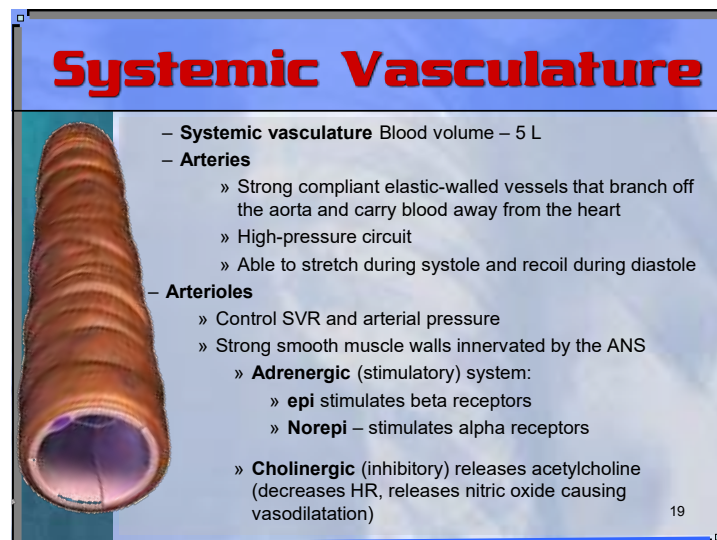
16



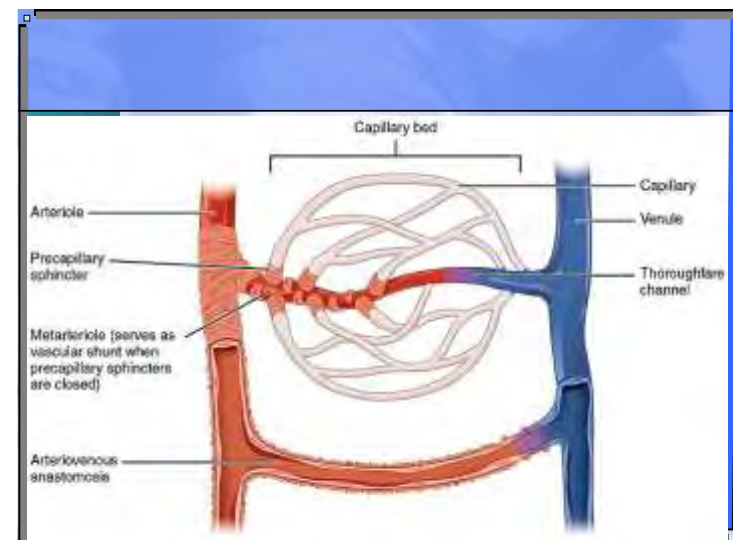
17



18



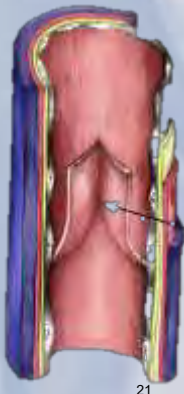
19



20

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



21

21

Capillaries

Venules

Capacitance vessels

Dilation = ↓ preload

Nitro
Morphine
Natreacor
Position
Volume



Arterioles

Resistance vessels

Dilation = ↓ afterload

Nipride
Natreacor
Catecholamines
Norepinephrine
Dopamine

Arterial constriction= increases afterload

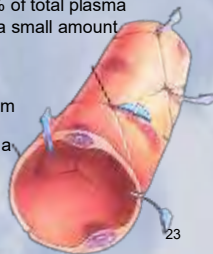
Venous Constriction= increase preload

22

22

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



23

23

FUNCTION

Extrinsic control

ANS – SNS & PNS

RAAS – Renal/Adrenal axis

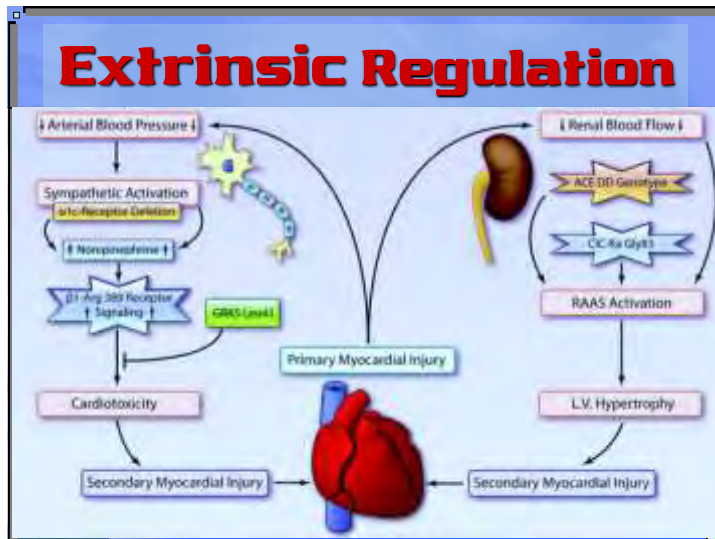
Intrinsic

Calcium

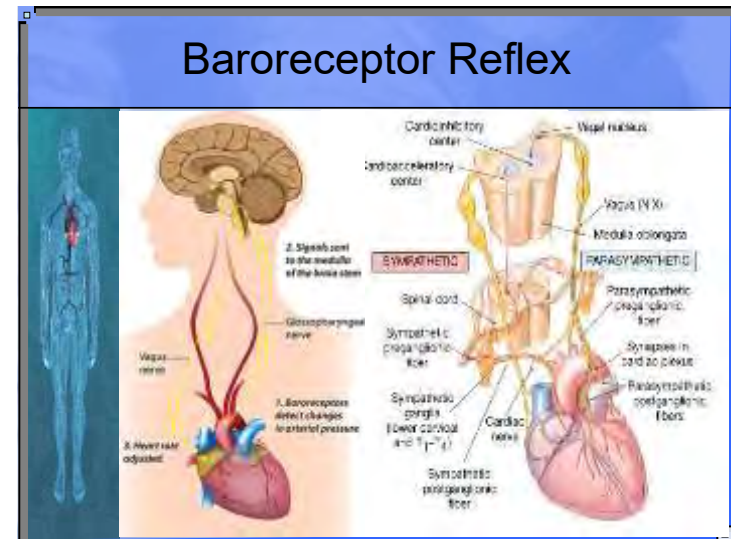
Action potential

Contractility

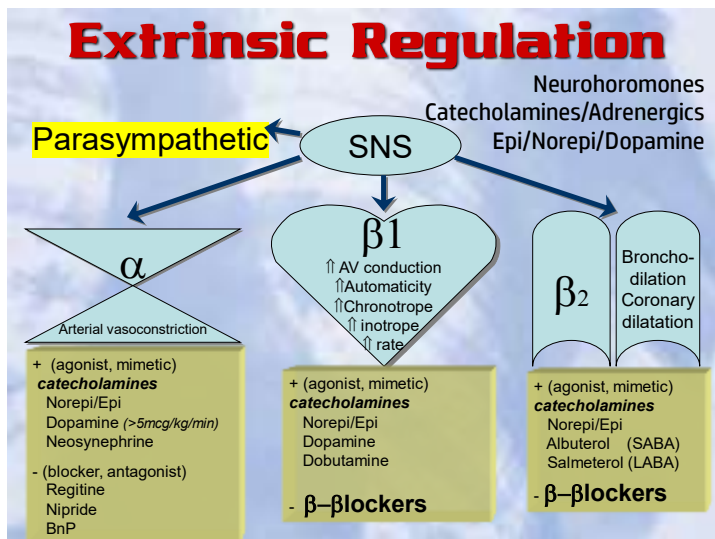
26



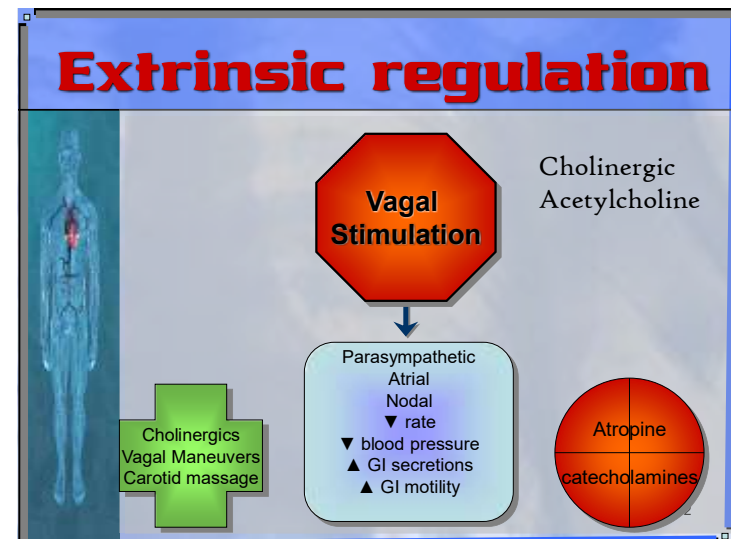
28



30

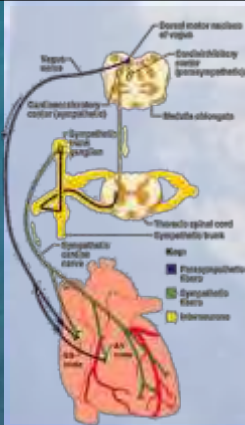


31



32

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

34

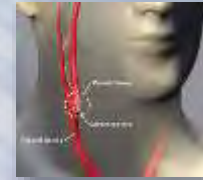
34

Decreased pressure



Decreased pressure:

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



35

35

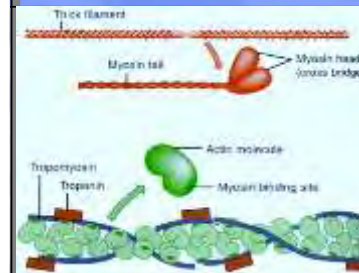
CALCIUM

Myocyte Contraction

36

36

Filament Proteins



• Thick Filament

–Myosin

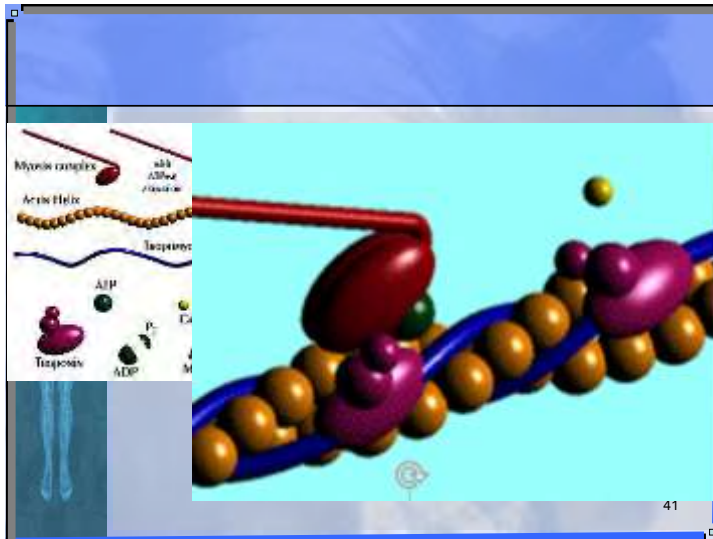
- head and tail regions
- 2 chains wrapped around each other (twisted golf clubs)
- hinge regions
- movement requires ATP
- filament has many Myosin units lined up
- bidirectional arrangement

• Thin Filament

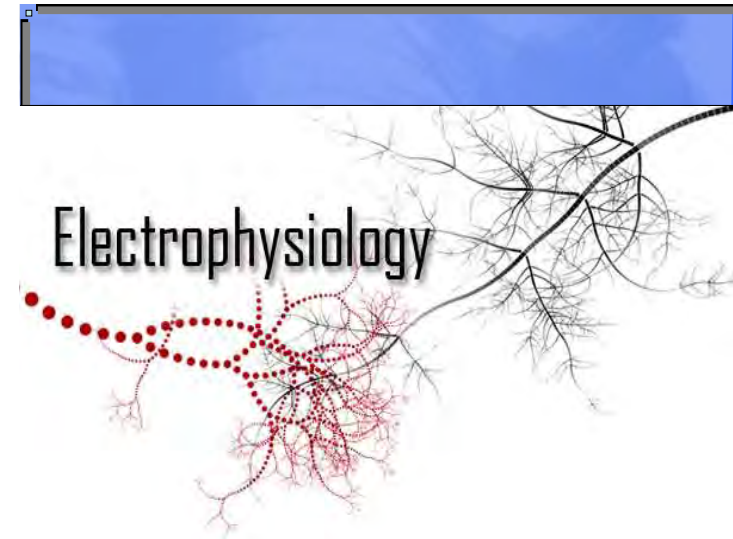
- Actin - many units in helix (Myosin binding sites)
- Tropomyosin - covers myosin binding site on actin
- Troponin - has Ca^{++} binding site

39

39



41



47

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

48

5 m/sec

1m/sec

0.3 m/sec

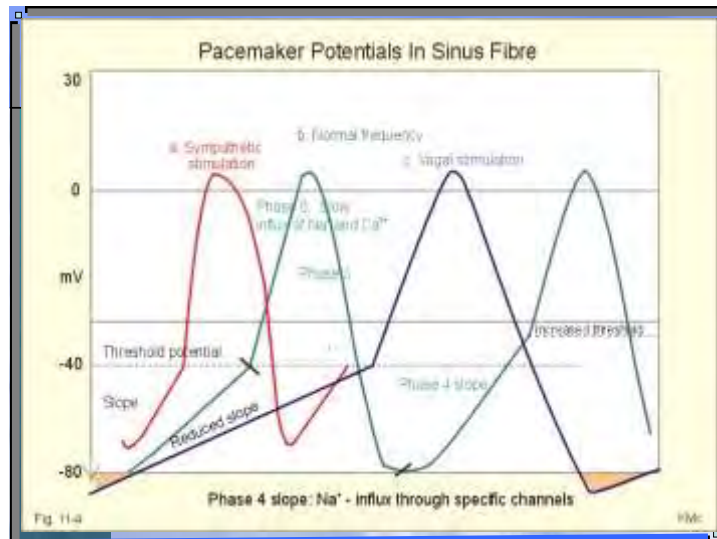
SA node	1.0 m/sec
Atria	0.3 m/sec
AV	0.03- .05 m/sec
Purkinje	1.5-5.0m/sec
Ventricular Muscle	0.3-0.5m/sec

Impulse direction

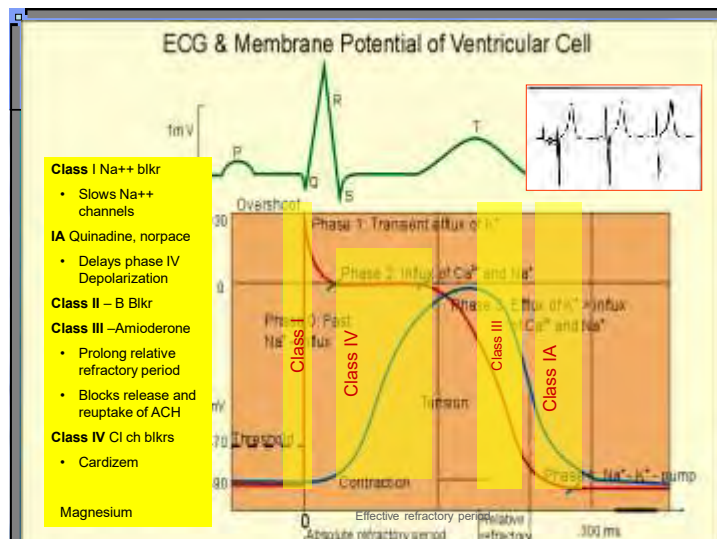
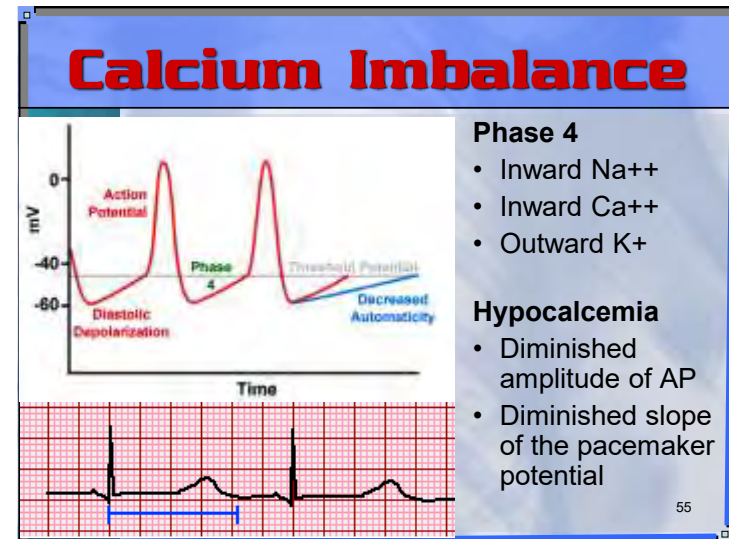
Tissue damaged, conducts in one direction only

P.04.02 - Re-entry pathway, caused by damaged tissue which only conducts impulses in one direction.

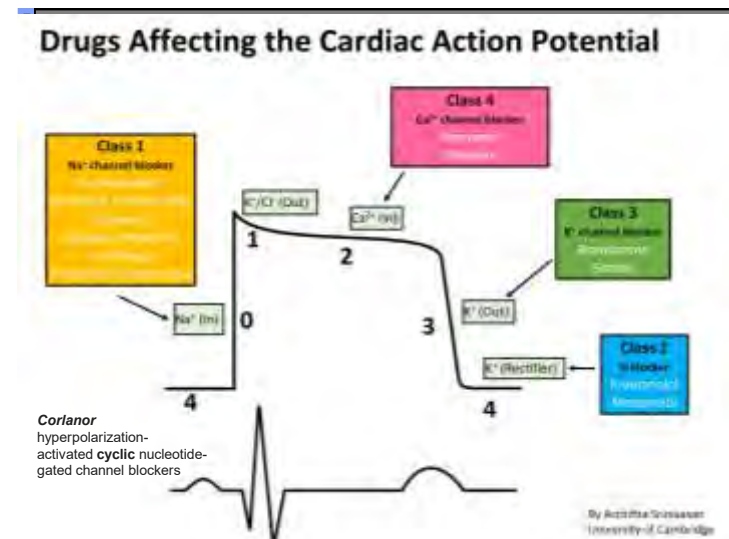
49



54

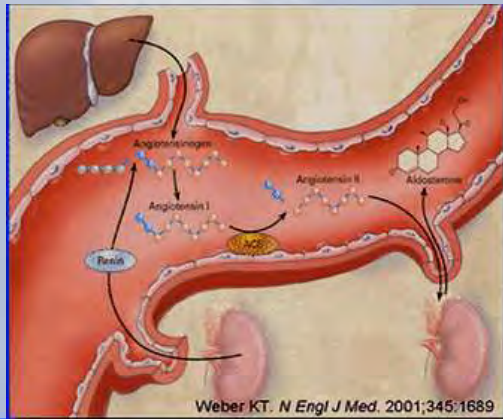


56



57

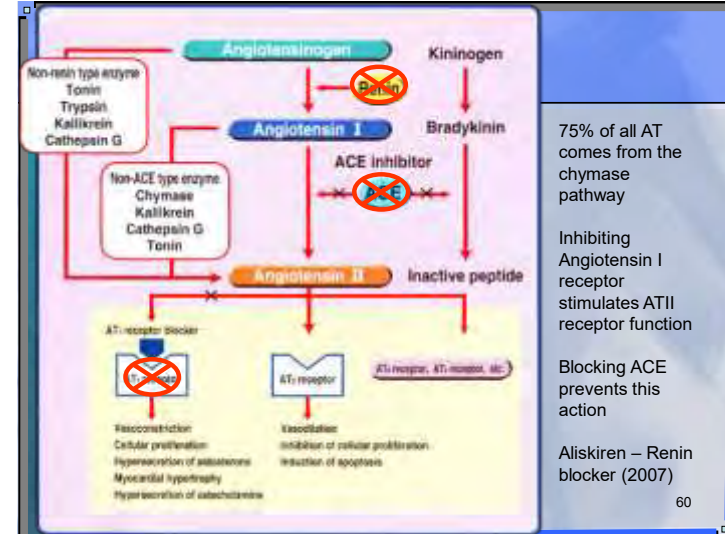
Renin-Angiotensin-Aldosterone System



Weber KT. N Engl J Med. 2001;345:1689

58

58



75% of all AT comes from the chymase pathway

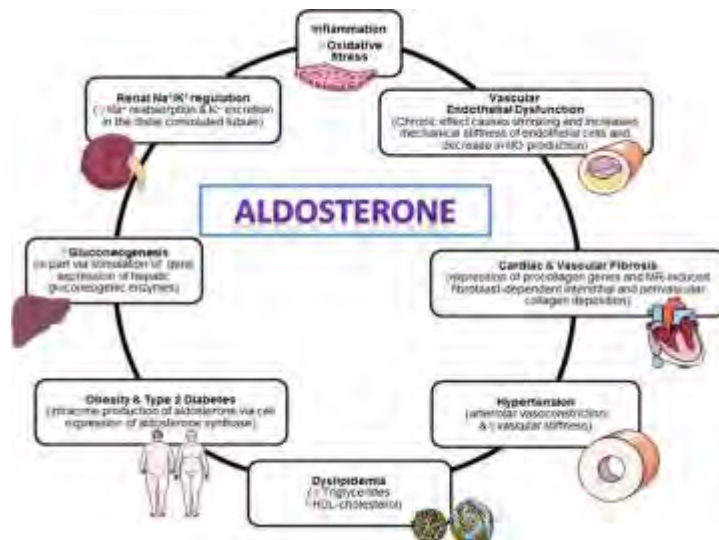
Inhibiting Angiotensin I receptor stimulates ATII receptor function

Blocking ACE prevents this action

Aliskiren – Renin blocker (2007)

60

60



61

When blood pressure drops

Decreased blood flow to the kidneys ($\uparrow$ BUN, eGFR)

Renin – angiotensin = $\uparrow$ BP/ SVR/ afterload

Inhibited with Meds:

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

Aldosterone = $\uparrow$ Na/water retention / preload

Inhibited with Meds:

Aldosterone blockers (spironolactone, Eplerenone)
Lasix to remove water
Natrecor to remove sodium and water

63

63

Application of Learning



66

APPLICATION

Preload
Afterload
Contractility
Heart Rate
Heart Sounds

68

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

69

69

Afterload

INCREASED BY

- HYPERTENSION
- AORTIC STENOSIS
- ATHEROSCLEROSIS
- NOREPINEPHRINE
- ANGIOTENSIN
- ENDOTHELIN

DRUGS

- DOPAMINE
- CATECHOLAMINES
- COCAINE/METH/CRACK
- EPI/NOREPI



LOWERED BY:

- SEPSIS
- NIPRIDE
- IABP
- BNP

70

70

Contractility

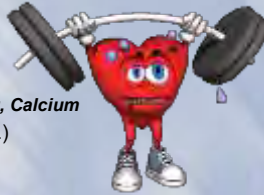
– Assessed indirectly through effects on CO or with noninvasive imaging

– **Effectors** include:

- » Positive inotropic drugs
 - » *Dobutamine, Primacore, Dig, Calcium*
- » Increased HR (Bowditch phen.)
- » SNS stimulation

– **Decreased contraction** is caused by:

- » Negative inotropes
 - » *IA antiarrhythmic, β blkrs, CaCH blkrs, barbs*
- » Hypoxia
- » Hypercapnia
- » Myocardial ischemia/infarction
- » Metabolic acidosis



71

71

Heart Rate

• **Heart rate:** Influenced by

- Blood volume status
- SNS and PNS tone
- Drugs
- Temp
- Resp
- Arrhythmias
- Peripheral vascular tone
- Emotions
- Metabolic status (hyperthyroidism)



73

73

Learning Objectives

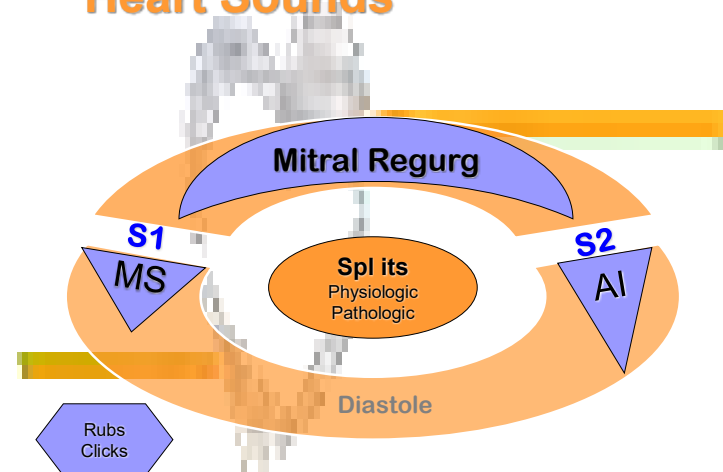
Can you now...

- 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

98

98

Heart Sounds



100

References

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 Anesthesiol.* 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/>

108

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

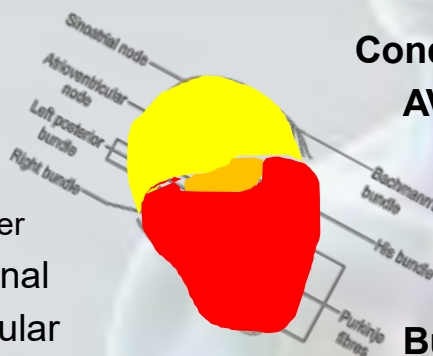
Conduction related

AV node

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

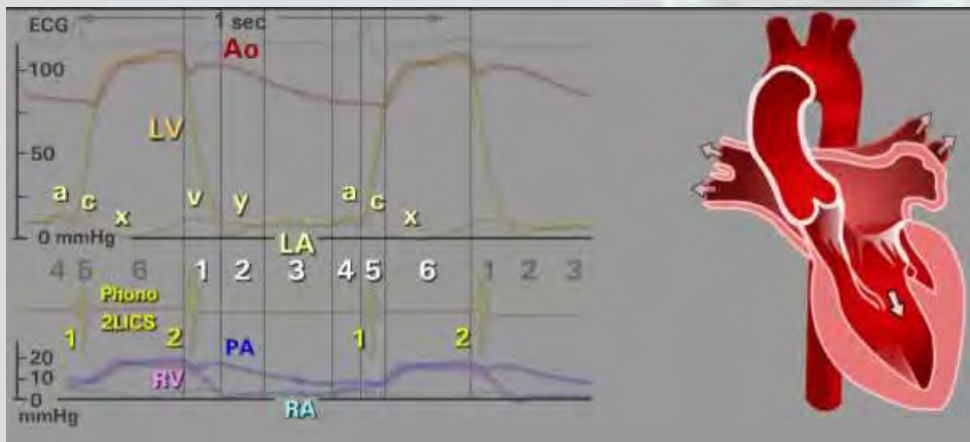
Bundle Branches

BBB



3

Cardiac cycle



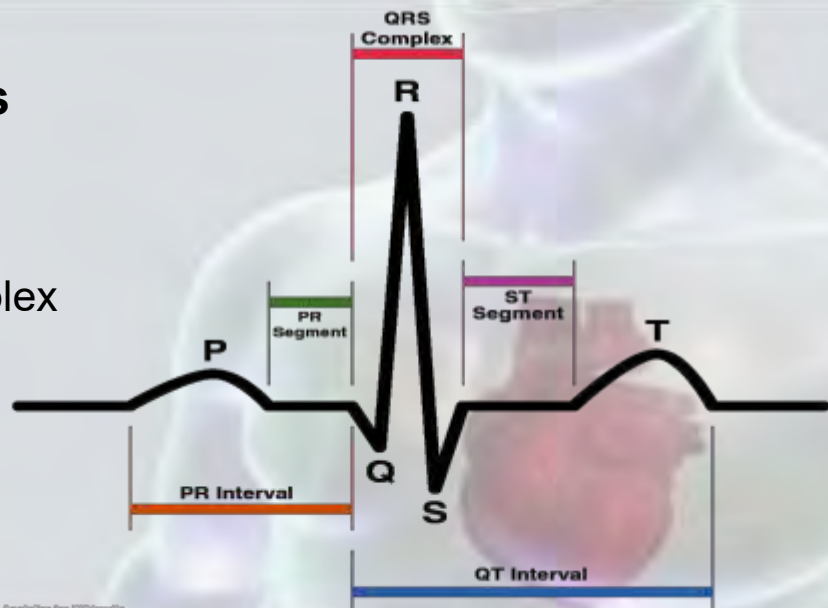
<http://blaufuss.org/tutorial/index1.html>

4

Waveforms

• Waveforms

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



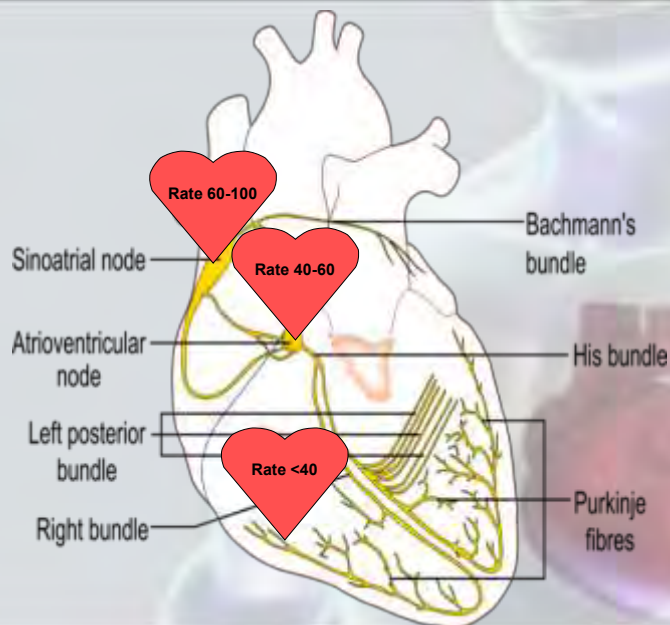
5

Paper



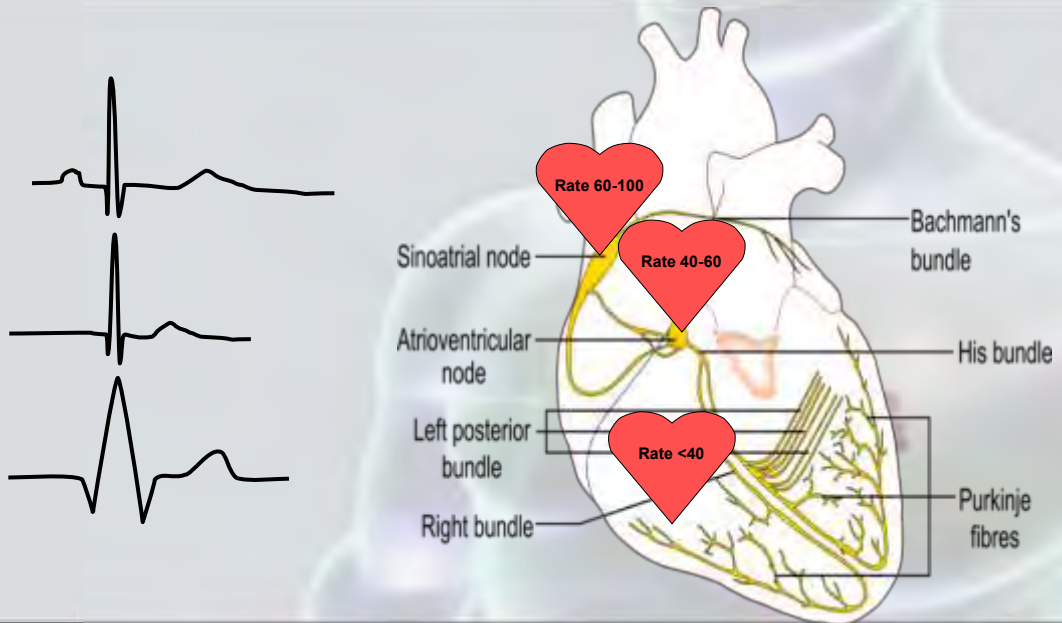
6

Anatomy



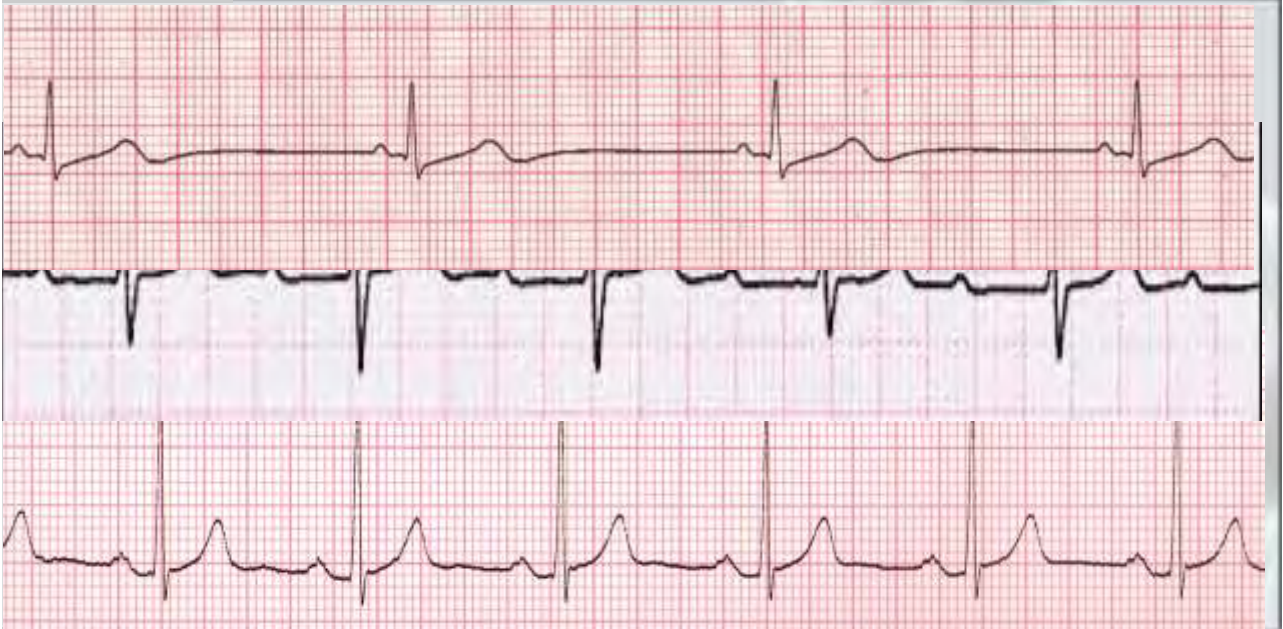
7

Conduction system

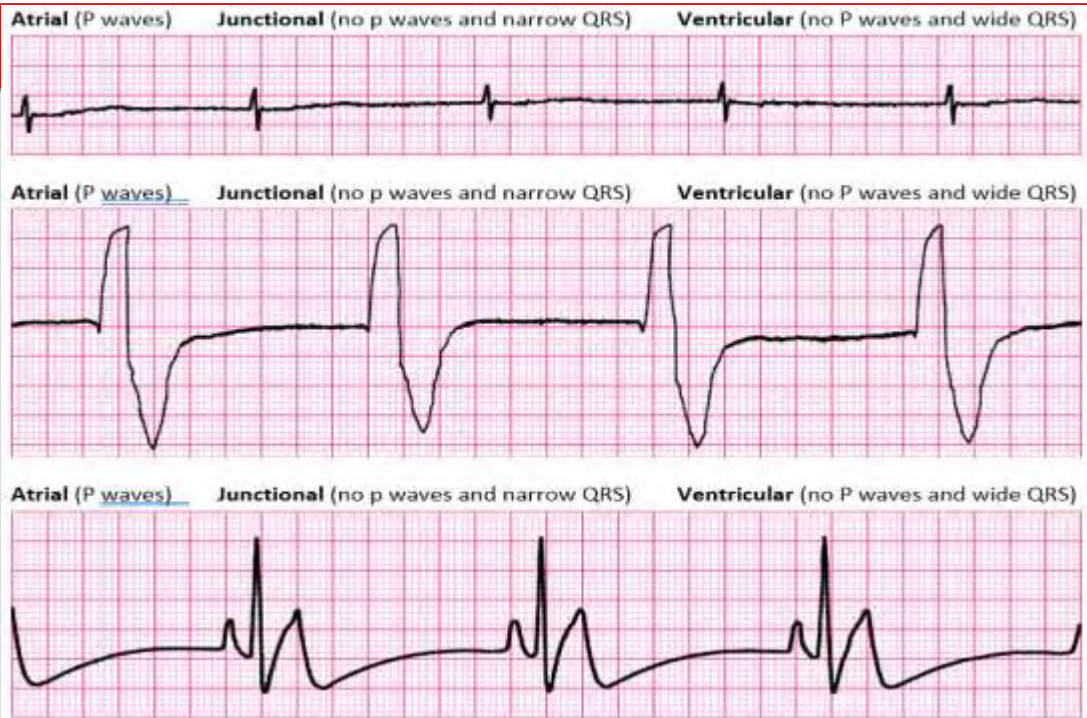


8

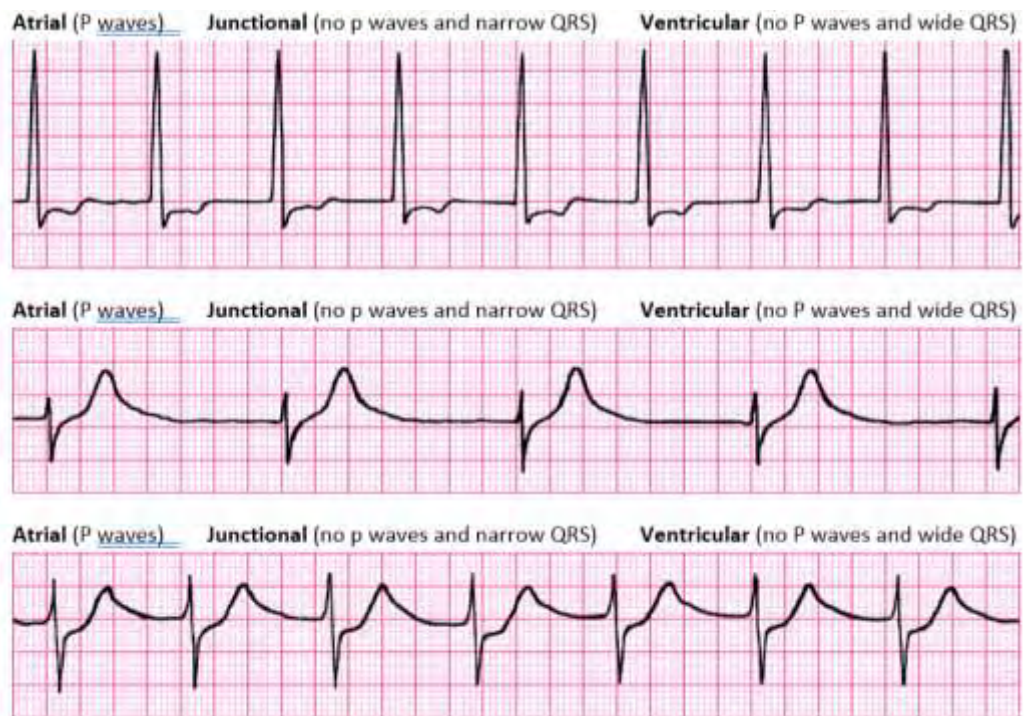
Not in Handout – View Screen



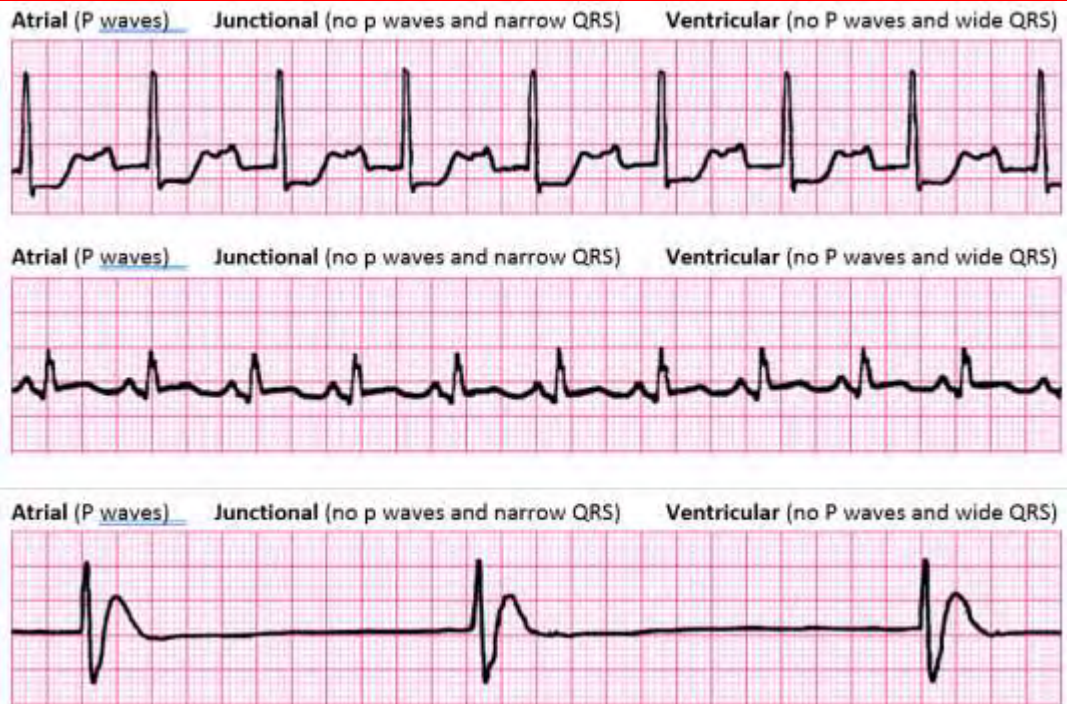
9



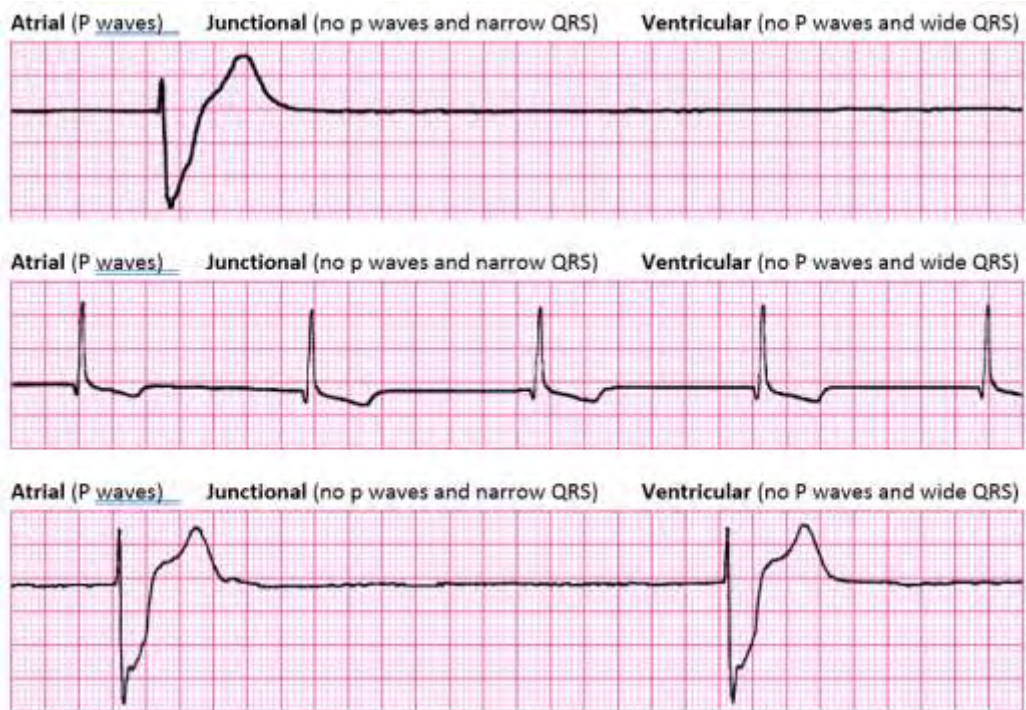
10



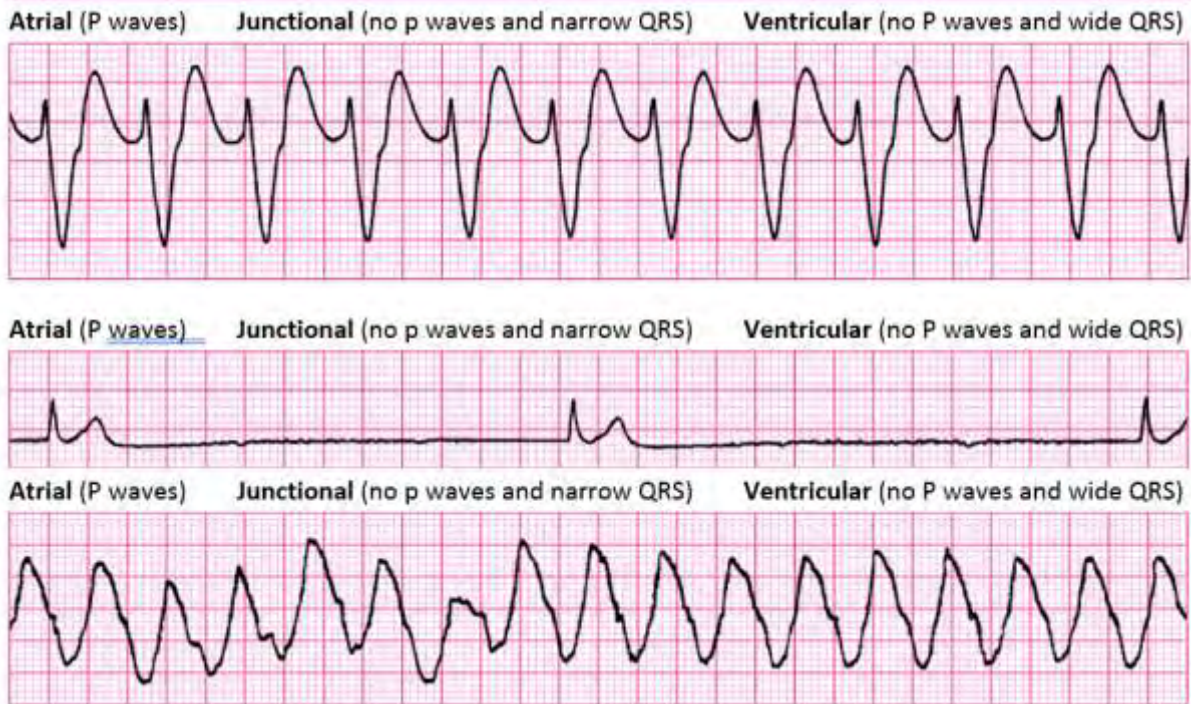
11



15



16



17

Premature atrial contraction

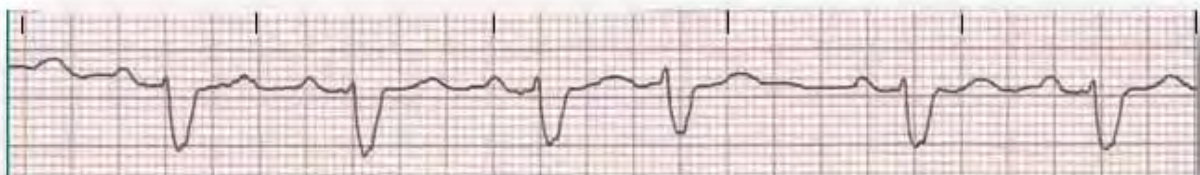
Atrial Rhythms:

PAC

Cause: irritability, atrial enlargement, caffeine, stress

Causative Medications: inhalers,

Treatment: none



Atrial rate: any

Ventricular Rate: any

P waves: upright, one to one, - **early beat may look different**

PR Interval: normal or long

QRS Complex: any

Rhythm: any – early beat causes irregularity

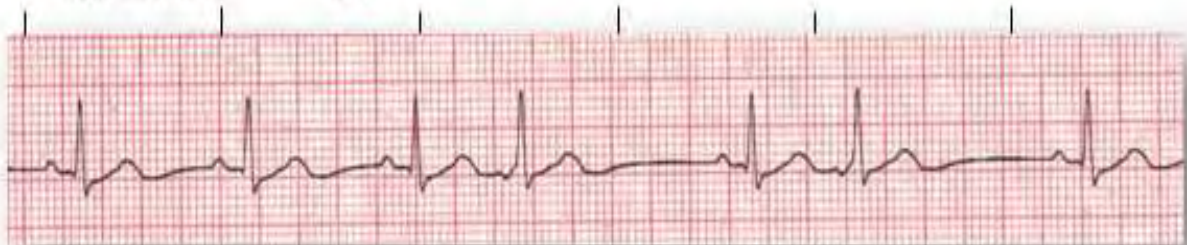
Rhythm: any – early beat causes irregularity

18

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	



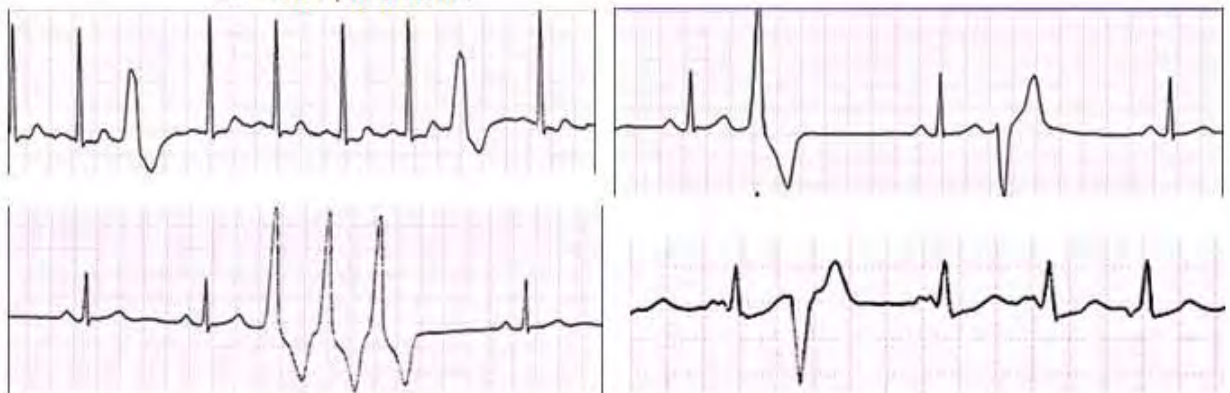
19

Premature ventricular contraction

PVC - (Premature Ventricular Contraction)

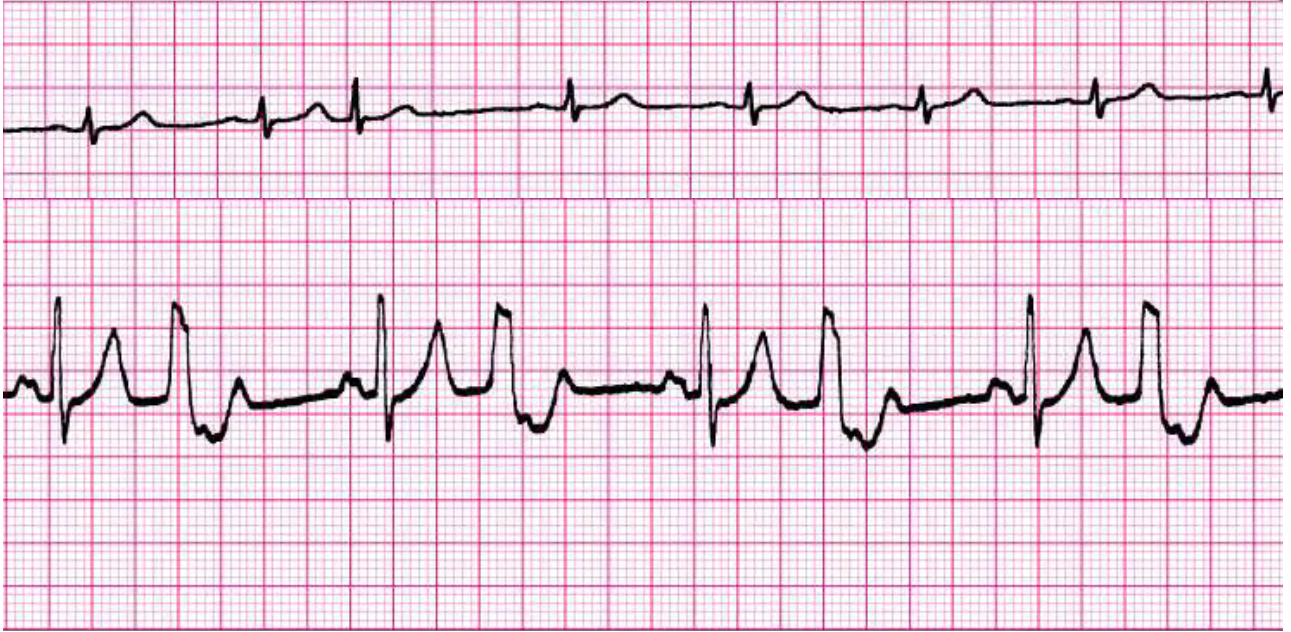
Reportable types:

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. Ron T phenomena



20

Premature Contractions



21



23



24



25

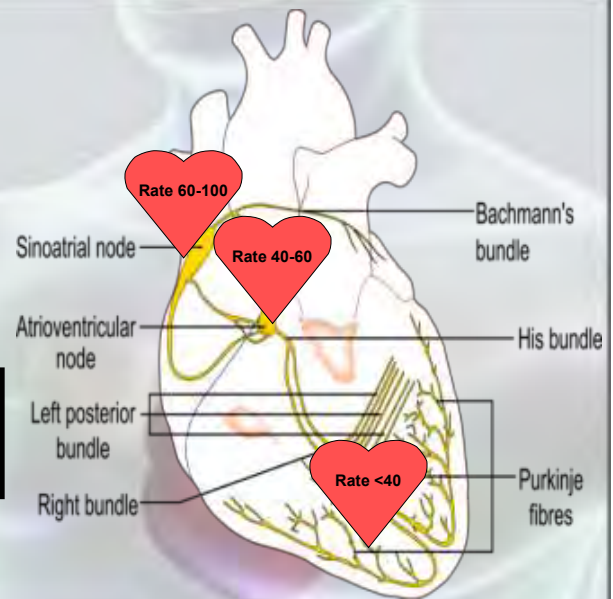


26



27

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)	

39

Sinus Brady

Sinus Brady

Cause: normal for athletes - PNS (Vagal response, bearing down, straining) Hypoxia, age related degeneration **Causative Medications** (BBkrs, 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

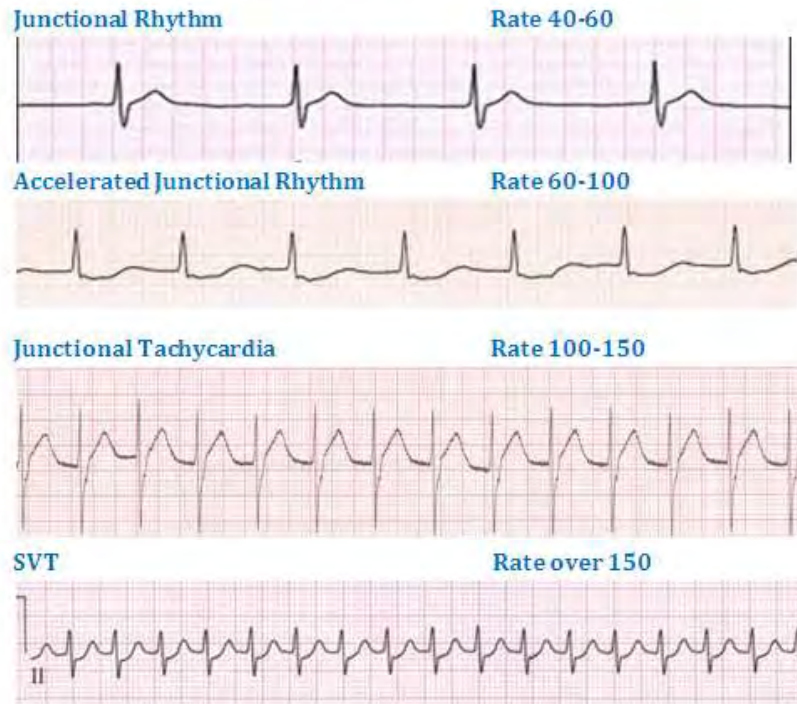
Sinus Tachycardia

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

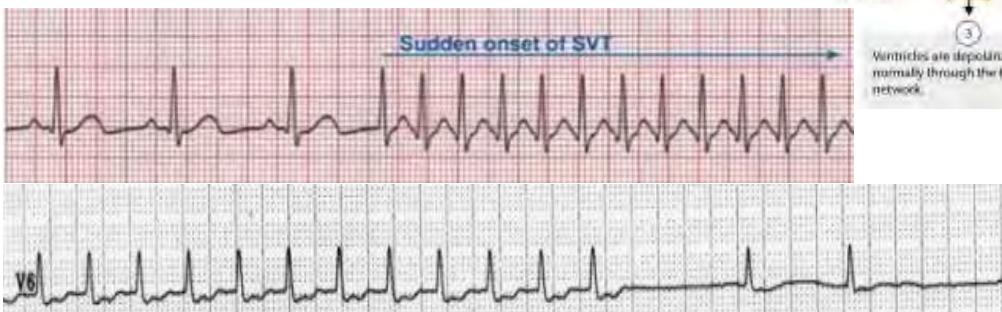
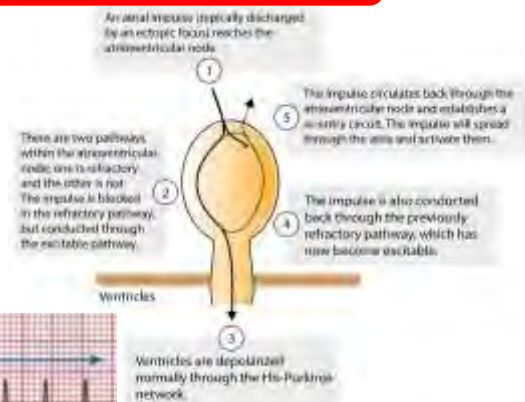


42

SVT — Supraventricular Tachycardia

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

Treatment
Vagals
Adenosine
Cardiovert if unstable



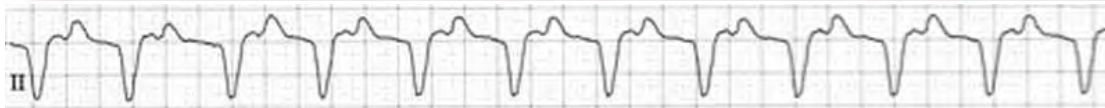
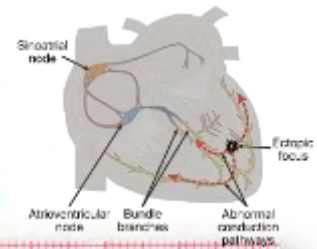
43

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)	

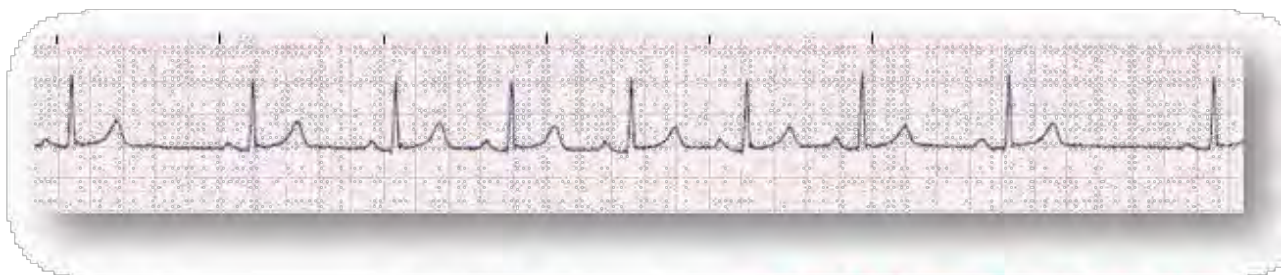
Rate less than 40

Rate 40-100

IVR – Idioventricular Rhythm**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 BBIs, CaChBIs, 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	

48

Atrial flutter

Atrial Flutter

Cause: irritability, atrial enlargement, Heart failure

Causative Medications: Digoxin toxicity

Treatment: chemical or electrical cardioversion. Digoxin, Amiodarone, BBIs, CaChBIs

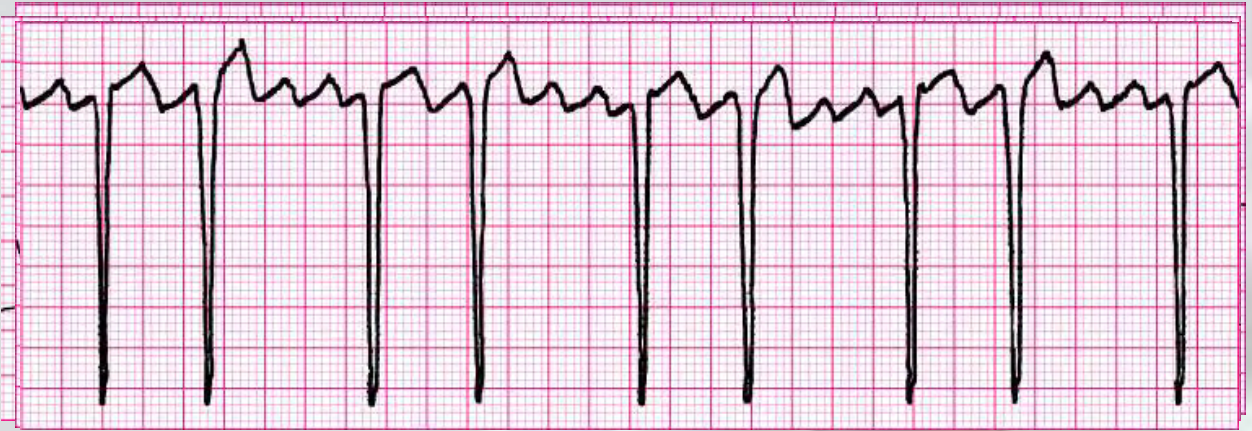


Atrial rate	over 250	Rhythm: Regular
Ventricular Rate	over 250	Rhythm: Regular or irregular
P waves:	replaced with saw-toothed flutter waves	
PR Interval:	none	
QRS Complex:	any – normal or wide	

49

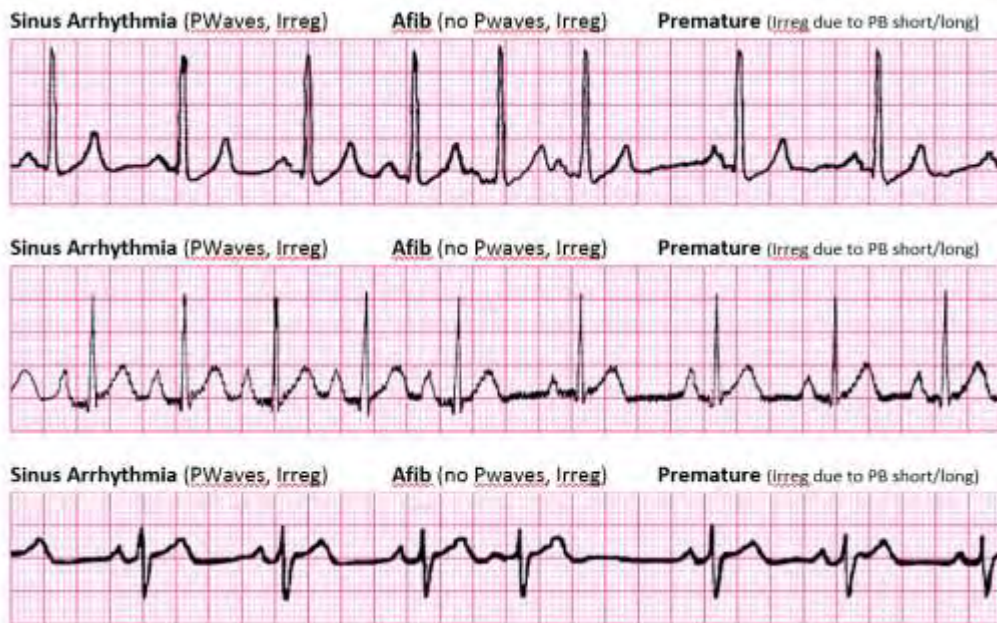
Irregular rhythm check

Not in Handout – View Screen



50

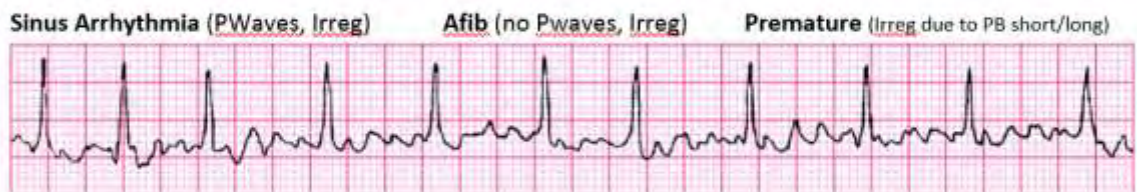
Worksheets



51



52



53



54

Disorders of Conduction



55

First Degree AV Block

First Degree AV Block

Cause: PNS (vagal, straining), age-related degeneration,

Causative Medications (BBIs, CaChBIs, Dig)

Treatment: none – watch for advancing block



Atrial rate

normal tachy or brady

Rhythm: Regular or irregular

Ventricular Rate

normal 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)

56

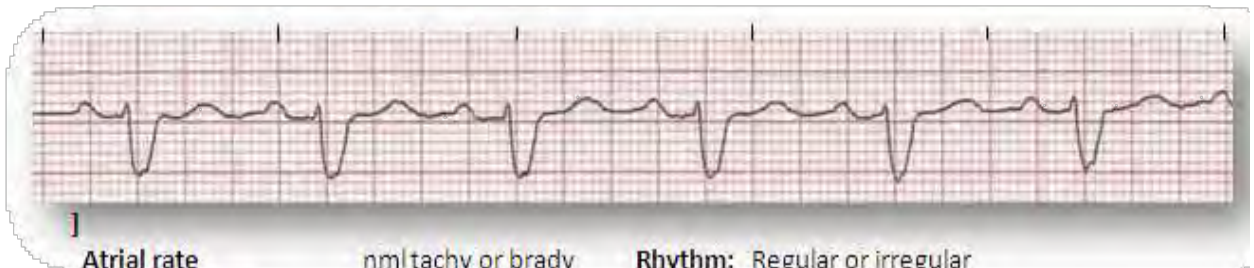
Bundle Branch Block

Bundle Branch Block

Cause: PNS (vagal, straining), age-related degeneration, MI

Causative Medications (BBIs, CaChBIs, Dig)

Treatment: none – watch for advancing block



Atrial rate

normal tachy or brady

Rhythm: Regular or irregular

Ventricular Rate

normal tachy or brady

Rhythm: Regular or irregular

P waves:

upright, one to one, and all look the same

PR Interval:

normal, short or long

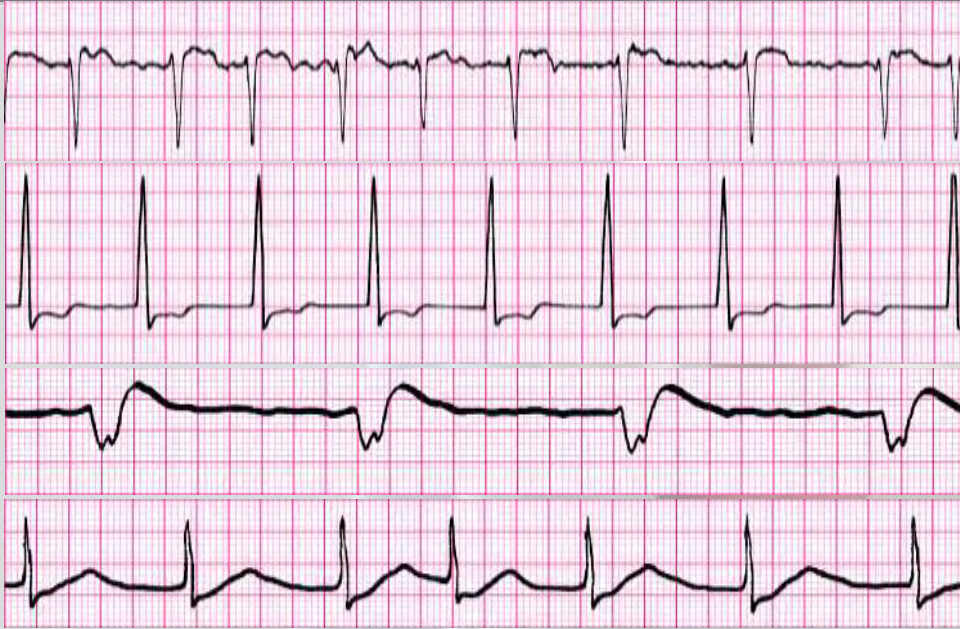
QRS Complex:

greater than 0.12 milliseconds (less than 3 small blocks)

57

No Pwaves

Afib, Junctional, Ventricular



85



86

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

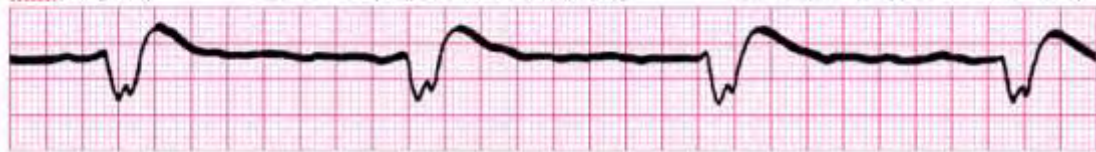
Afib (Irregular) **Junctional** (Regular with Narrow QRS) **Ventricular** (Regular with wide QRS)



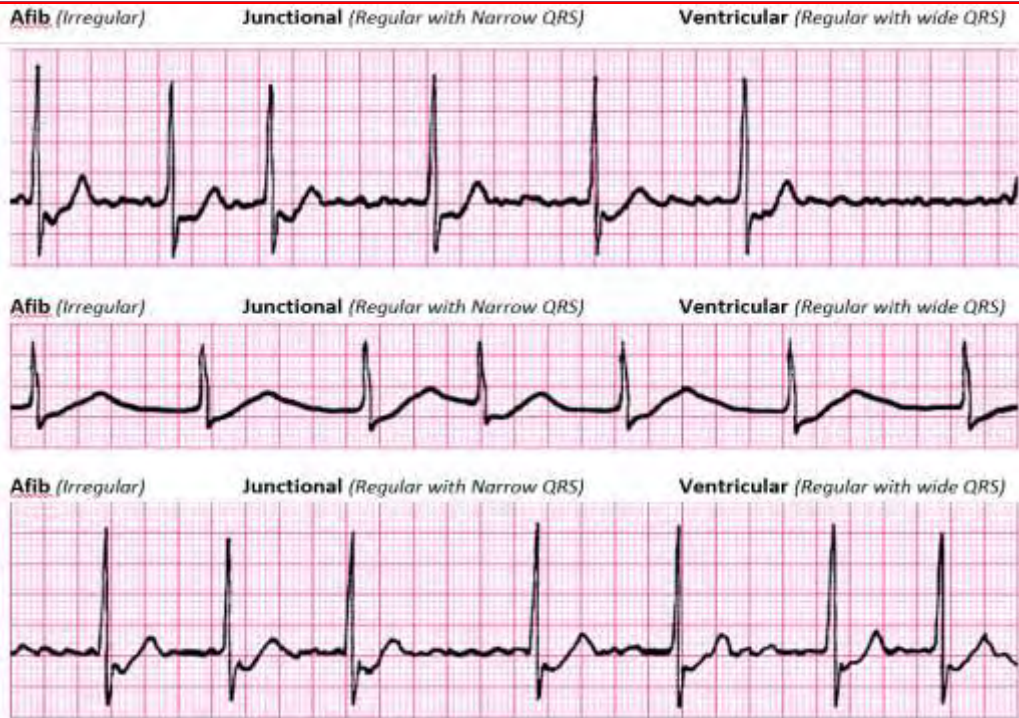
Afib (Irregular) **Junctional** (Regular with Narrow QRS) **Ventricular** (Regular with wide QRS)



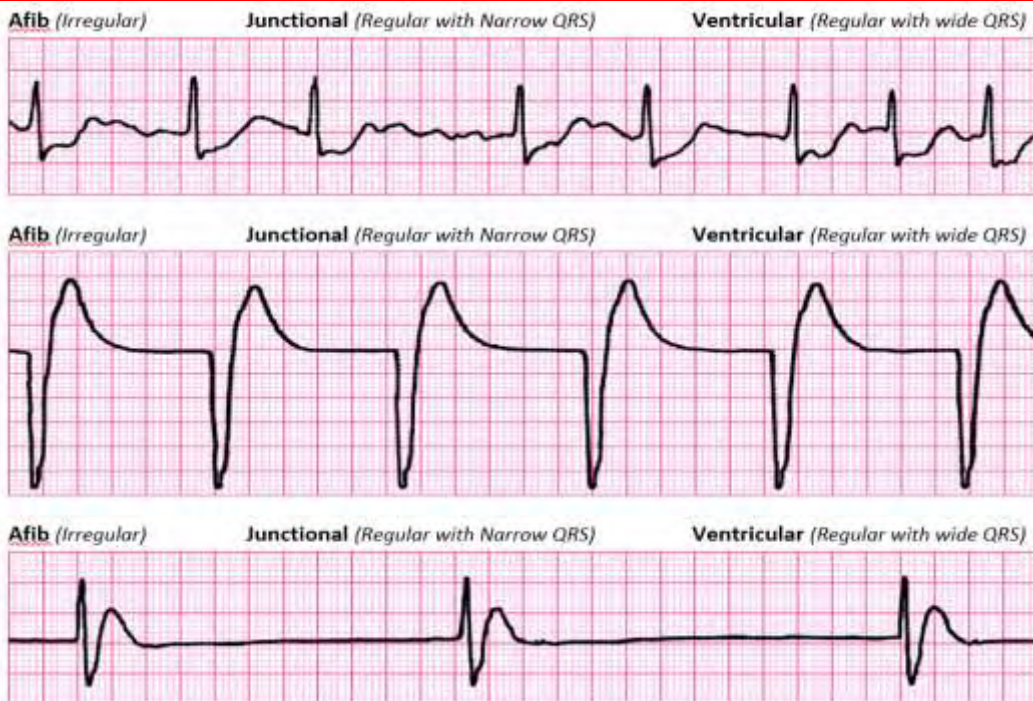
Afib (Irregular) **Junctional** (Regular with Narrow QRS) **Ventricular** (Regular with wide QRS)



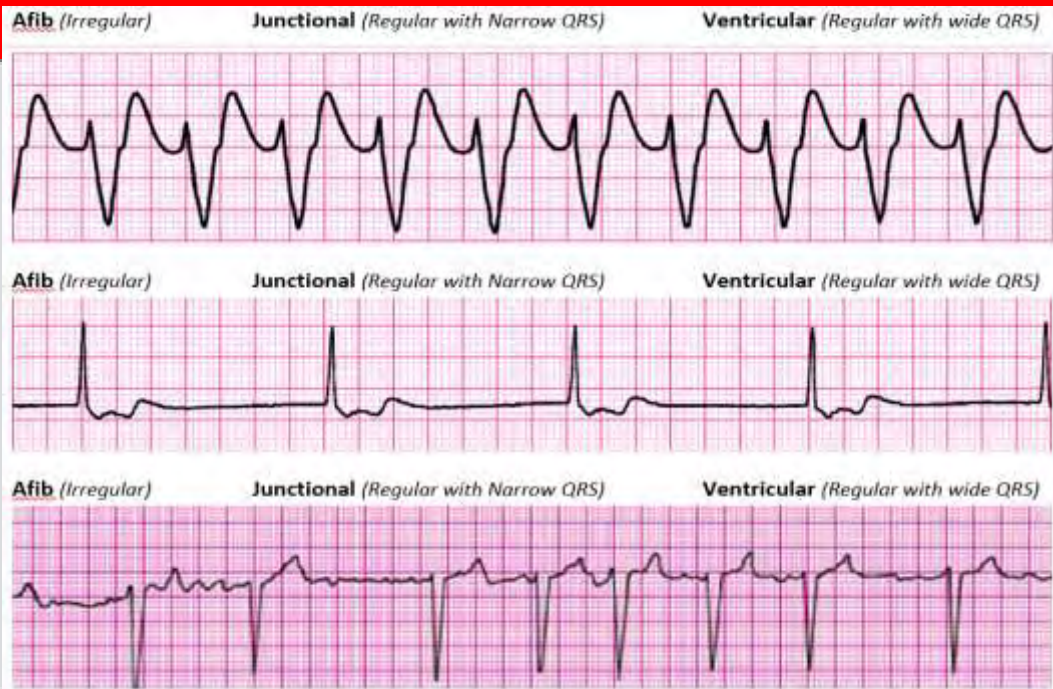
88



89



90



91

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
 Ventricular Rate: none
 P waves: none
 PR Interval: none
 QRS Complex: none

Rhythm: NA
 Rhythm: chaos

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?

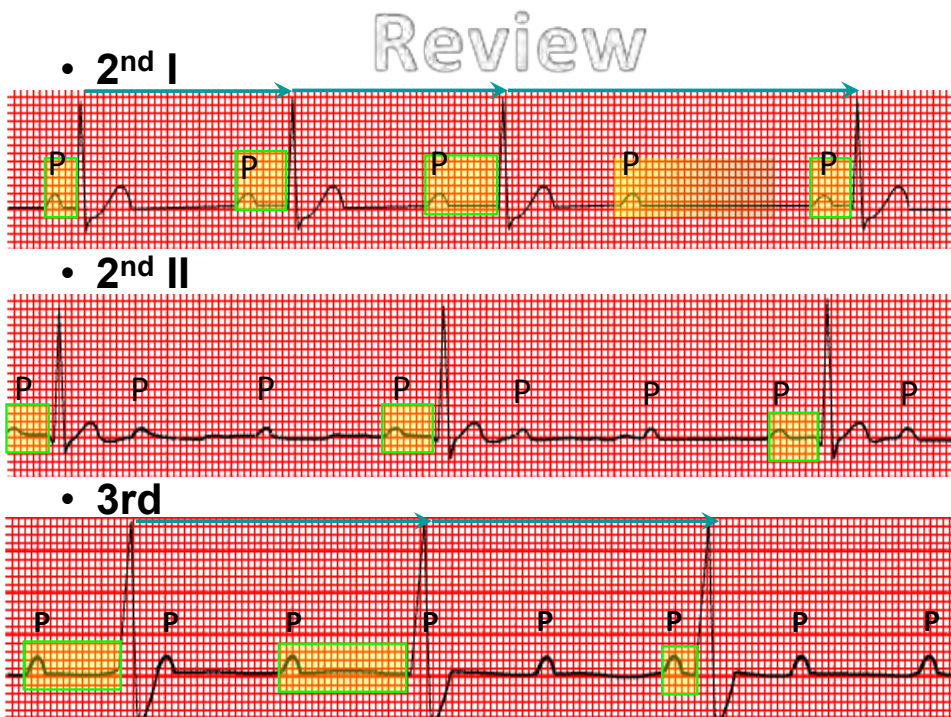
If irregular – **2nd I**



If regular – **3rd**



100



101

Heart Block Practice



105

Worksheets

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

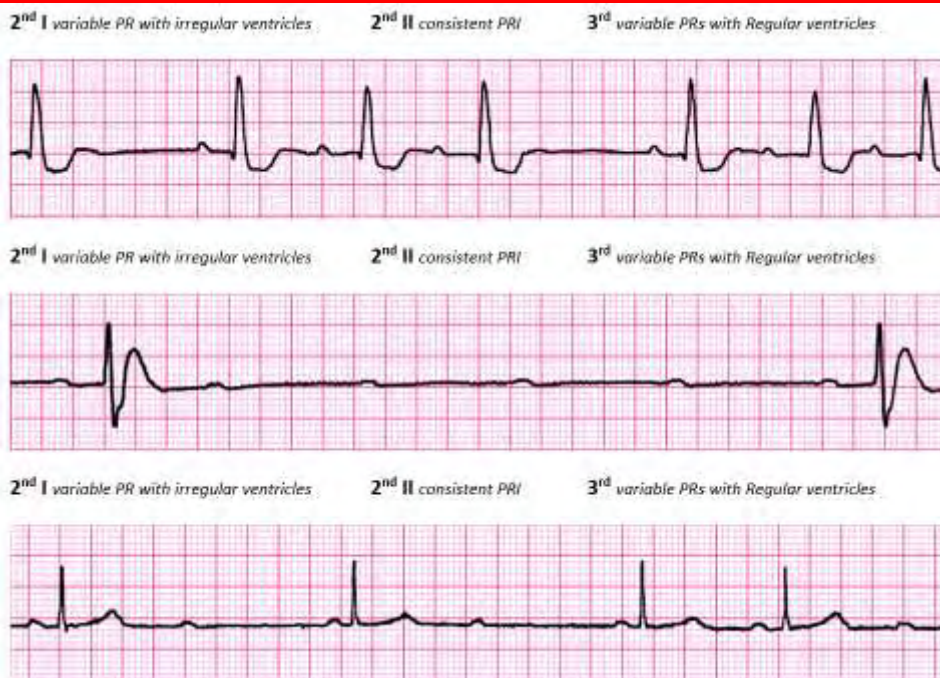
3rd variable PRs with Regular ventricles



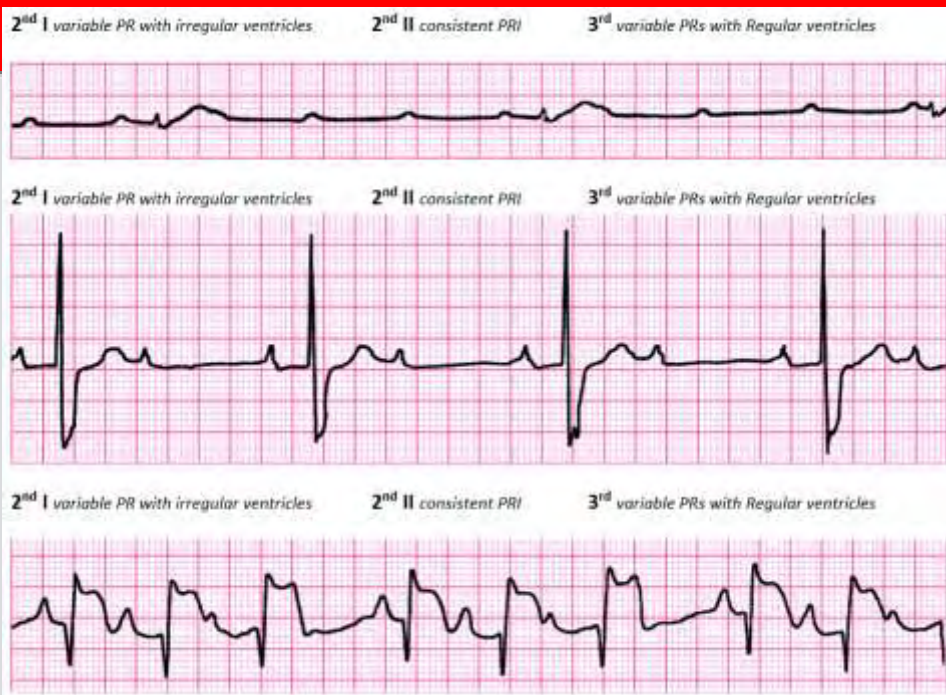
106



107

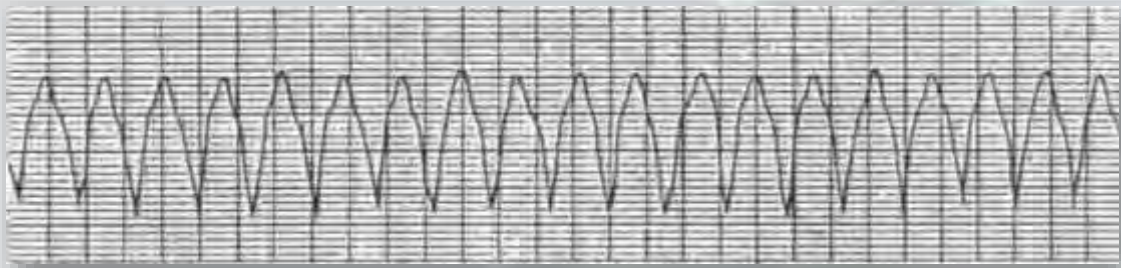


108



109

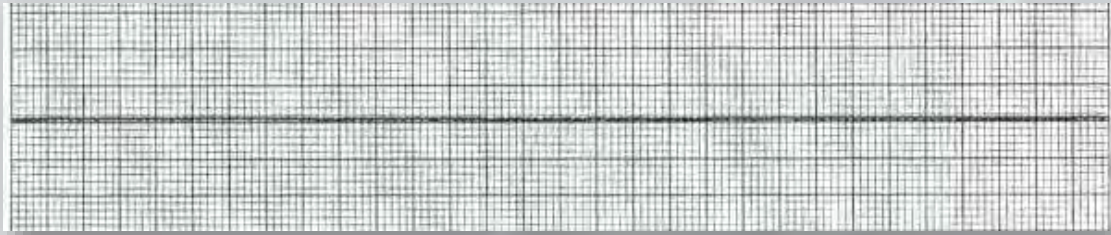
Question 1



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

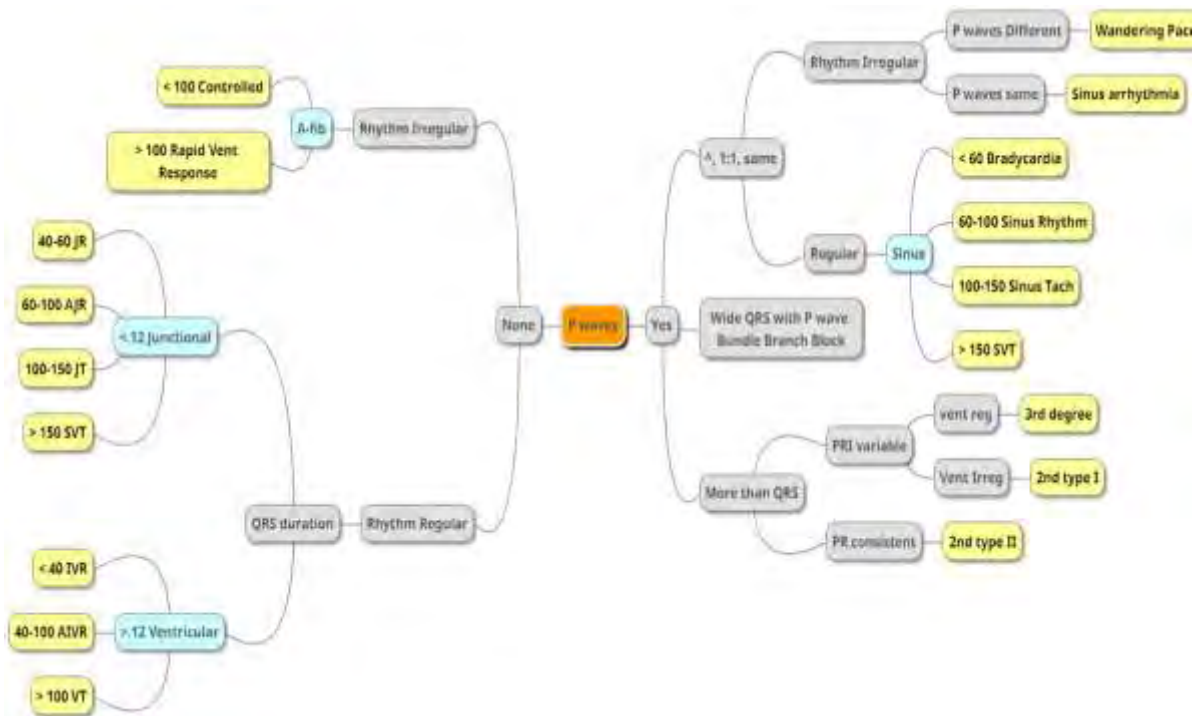
143



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





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



ARKANSAS HEART HOSPITAL®

2



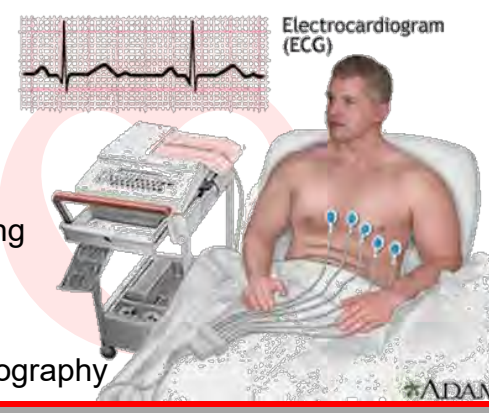
ECG

12 LEAD
STRESS TESTING
AMBULATORY ECG

3



12-Lead ECG



Electrocardiogram (ECG)

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

ADAM

4



7



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

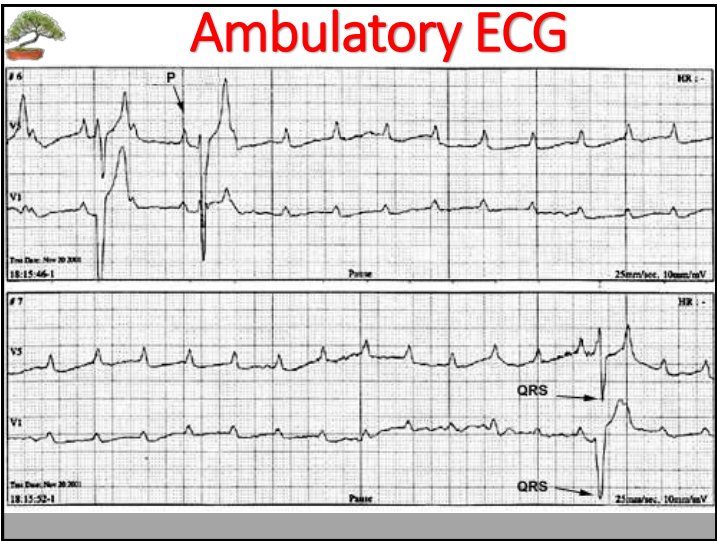
When to Stop the Stress

- 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

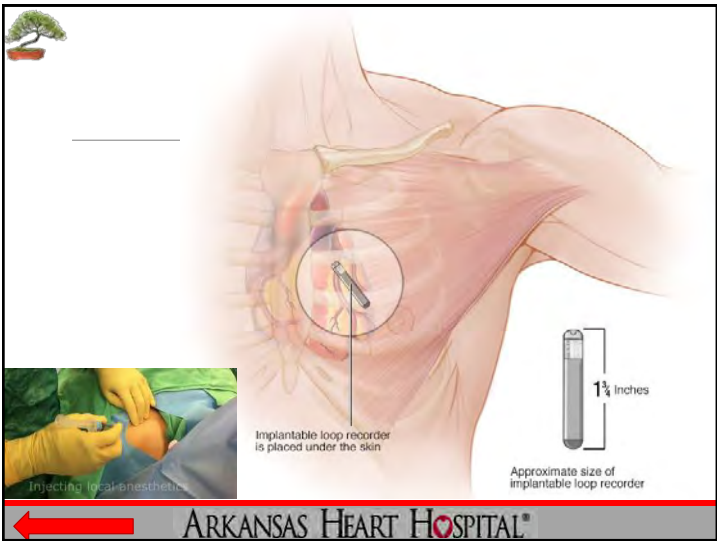


17

Ambulatory ECG



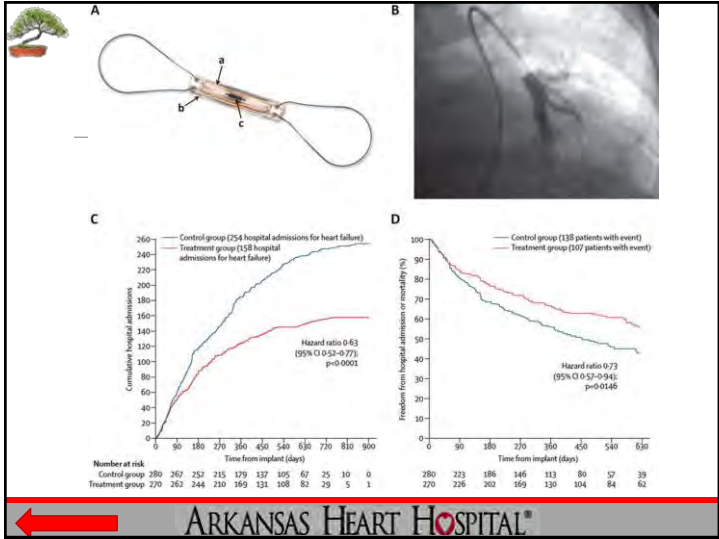
18



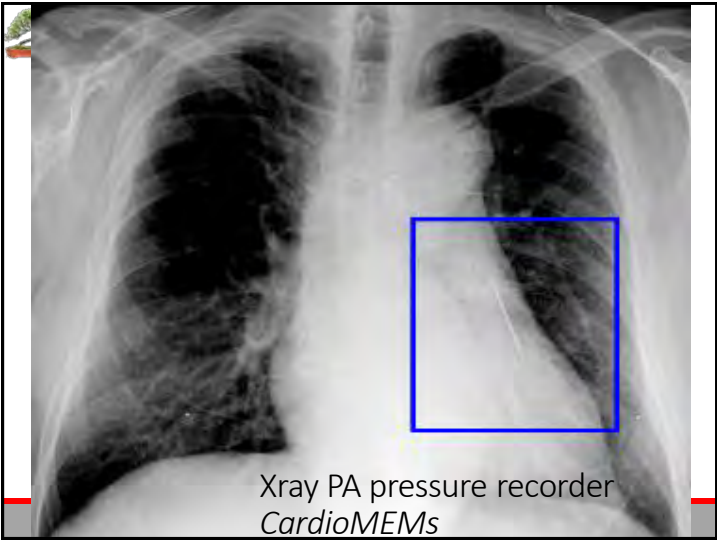
19



20



21



22

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

Imaging

23

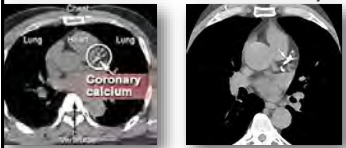
CAC – Calcium Scoring

Calcium is measured using a noncontrast limited CT

3-5 second breath hold

Scored 0 to 500

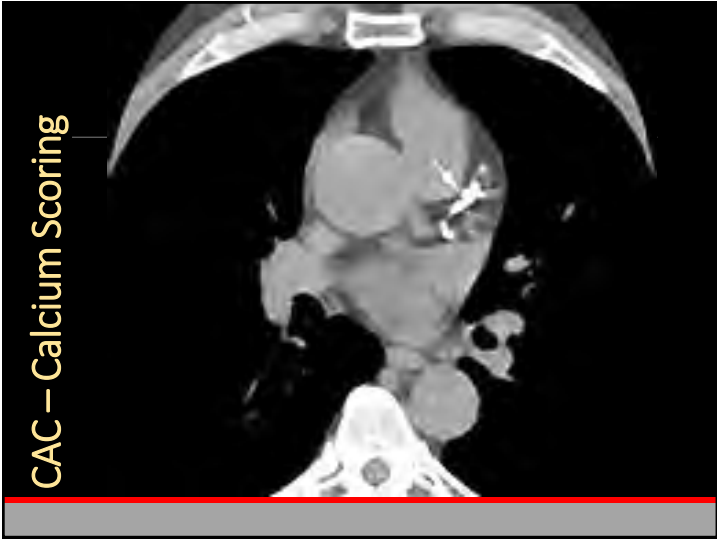
Not recommended < 40y



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



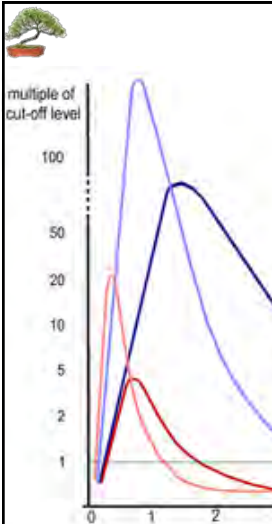
25



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

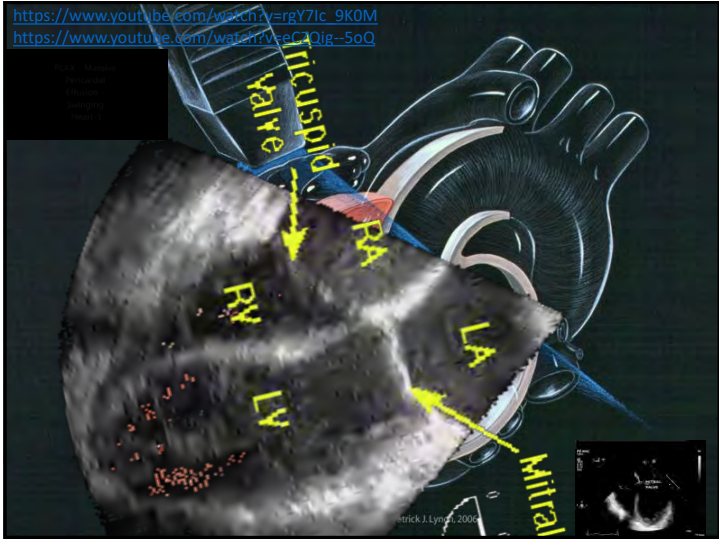
Noninvasive ultrasonic imaging to visualize heart

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



ARKANSAS HEART HOSPITAL®

31



32

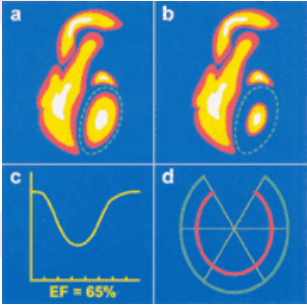


Nuclear Medicine

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

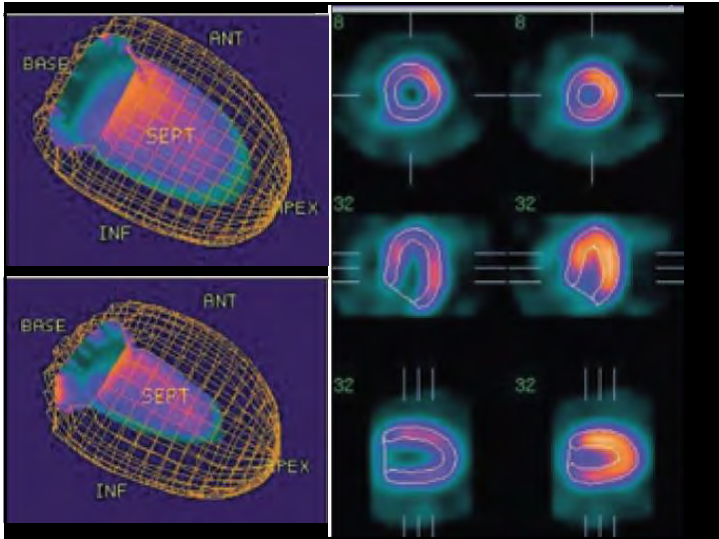
2 main varieties:

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

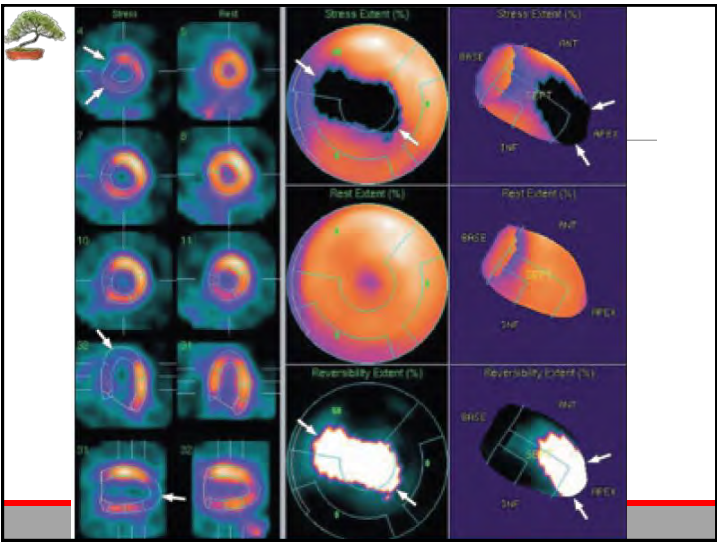


ARKANSAS HEART HOSPITAL®

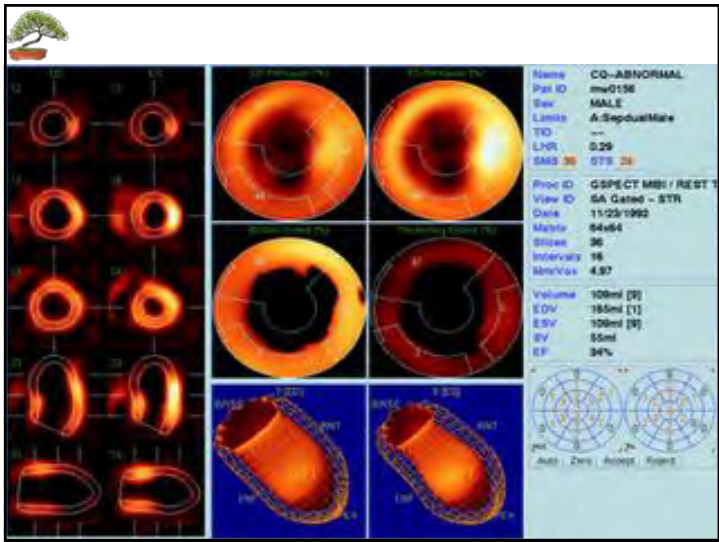
41



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.)



ARKANSAS HEART HOSPITAL®

45

Non-contrast MRA

Anatomy is Key

Great for patients with renal insufficiency

- New FDA warning on Gadolinium

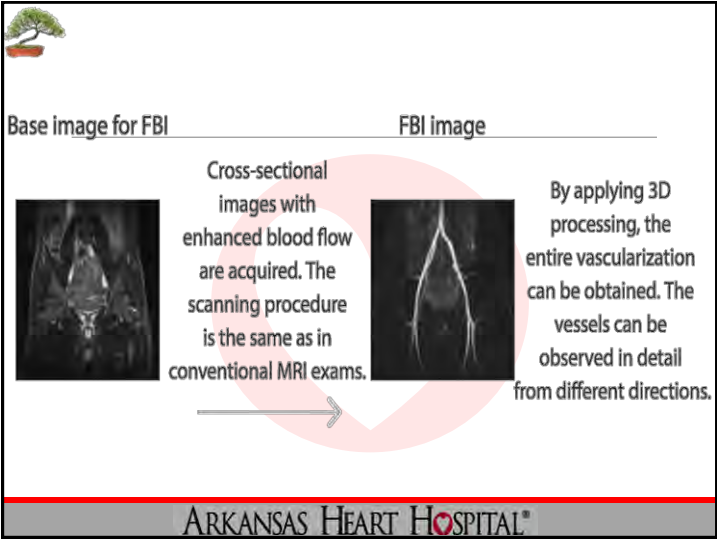
Time of Flight (TOF) MRA (technique to visualize flow within vessels, without the need to administer contrast.)

Phase Contrast MRA (technique that can be used to visualize moving fluid.)

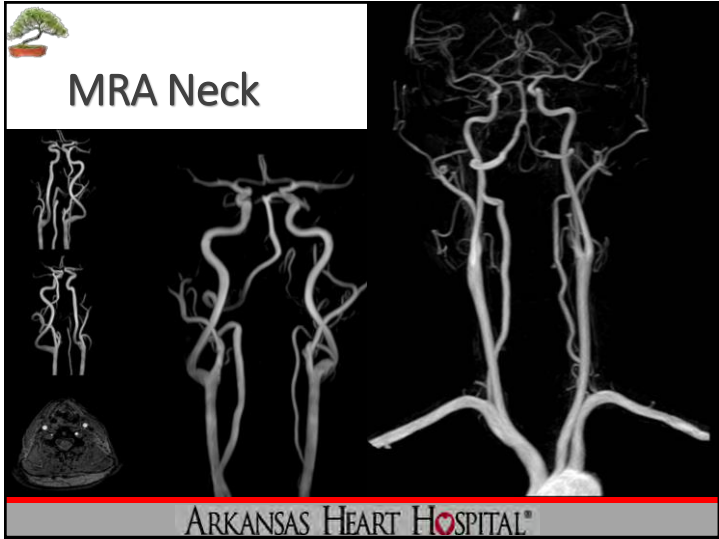
Fresh blood imaging technique (FBI)

- Fast
- STIR (fat suppression)
- ECG gated
- Arterial and Venous flow
 - Subtract venous from arterial

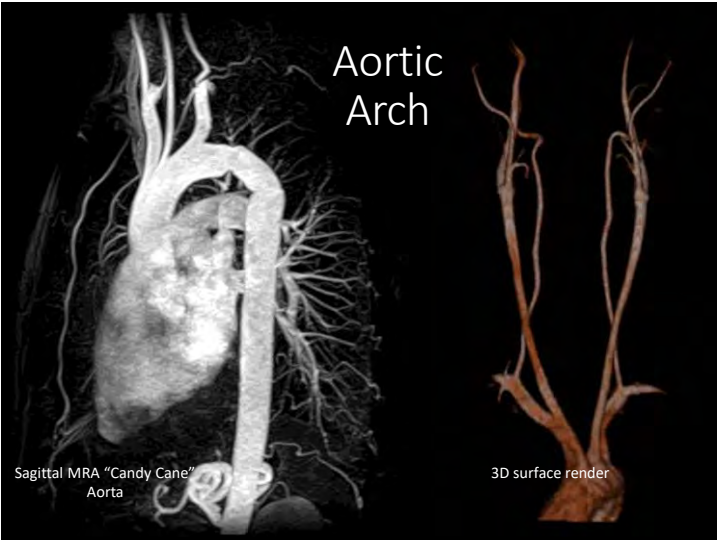
48



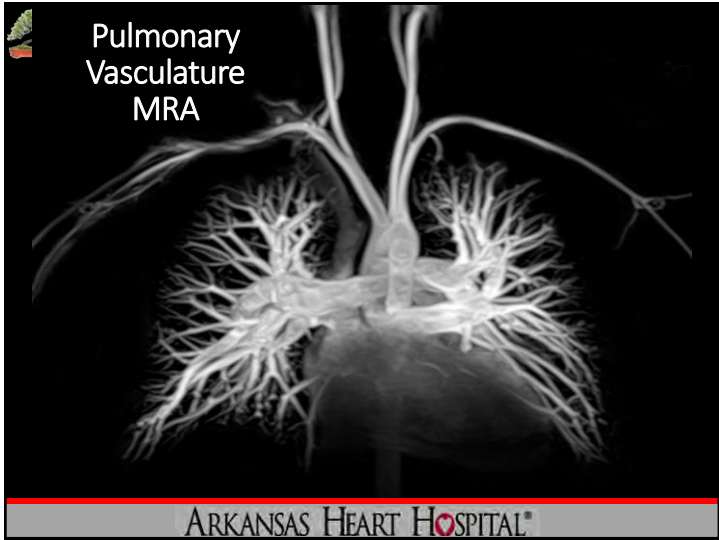
49



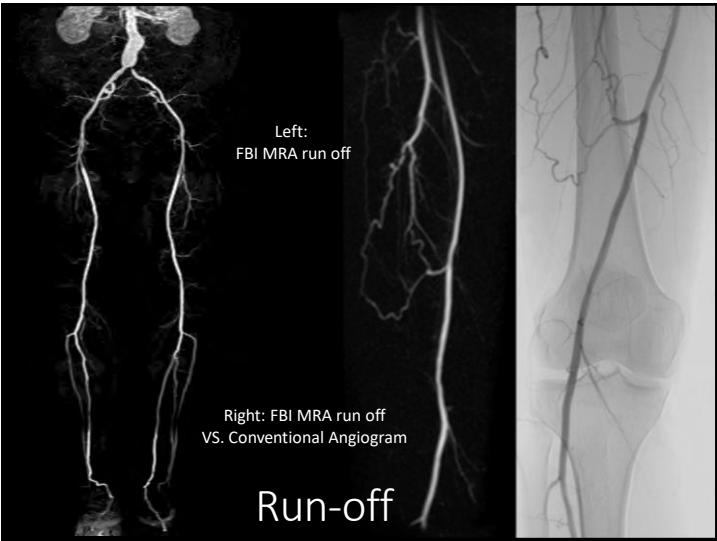
50



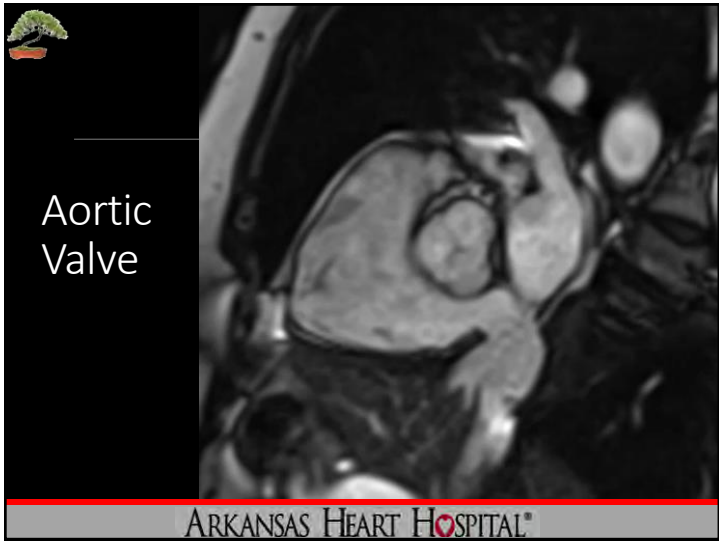
51



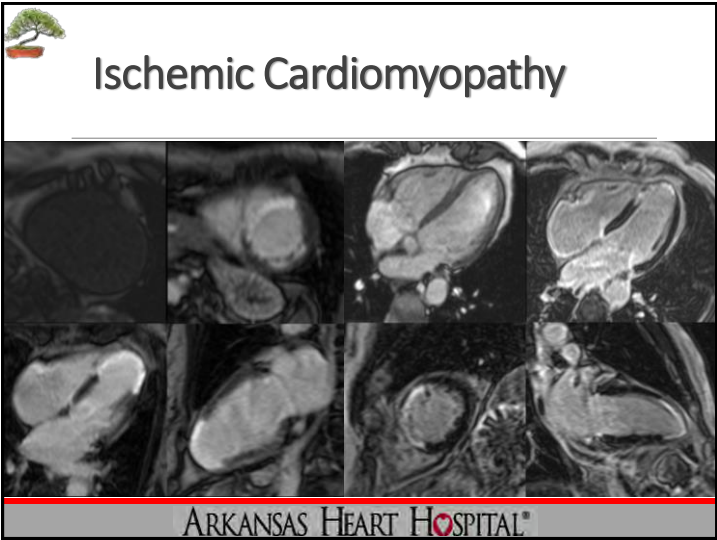
52



54



62



64

Cardiac MRI Indications

Anatomy ◦ ASD ◦ VSD ◦ Tri vs. Bicuspid delineation ◦ Myocardial wall thickness	Excellent tool for functional imaging ◦ EF calculation ◦ Shunt fractions using flow quantification ◦ Myocardial motion
Cardiac masses ◦ Myxomas ◦ Thrombus ◦ Tumors	Valvular pathology ◦ Stenosis ◦ Regurgitation ◦ Valvular vegetation
DCE ◦ Ischemic vs. Non-Ischemic Cardiomyopathy	

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

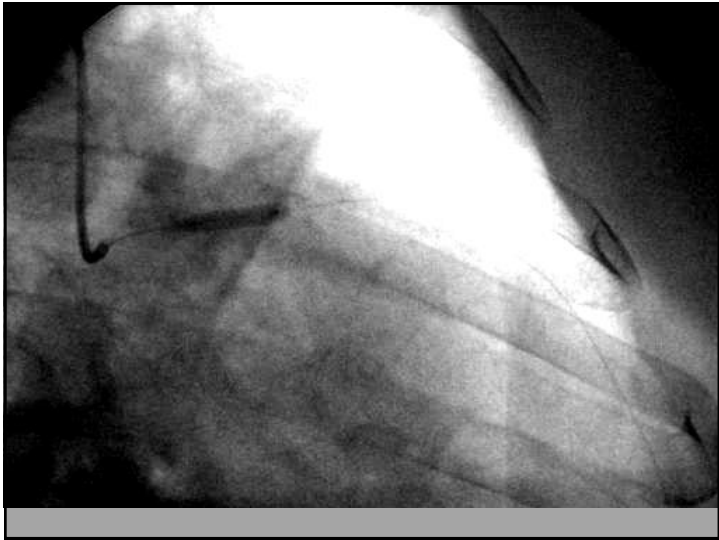
68



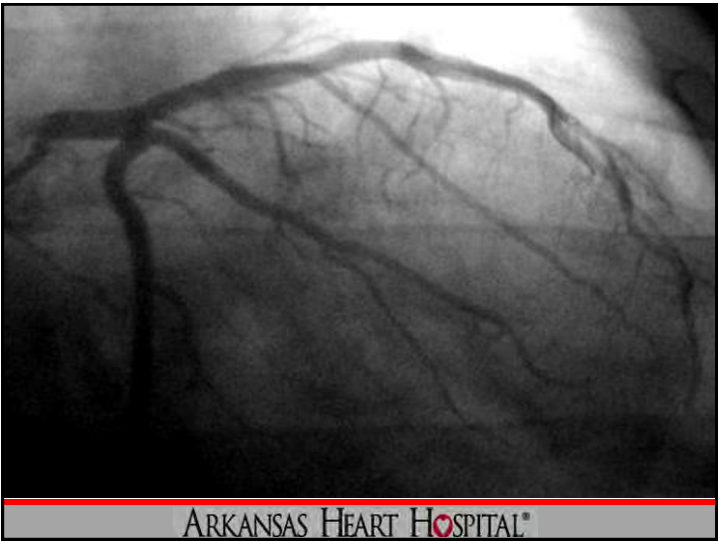
69



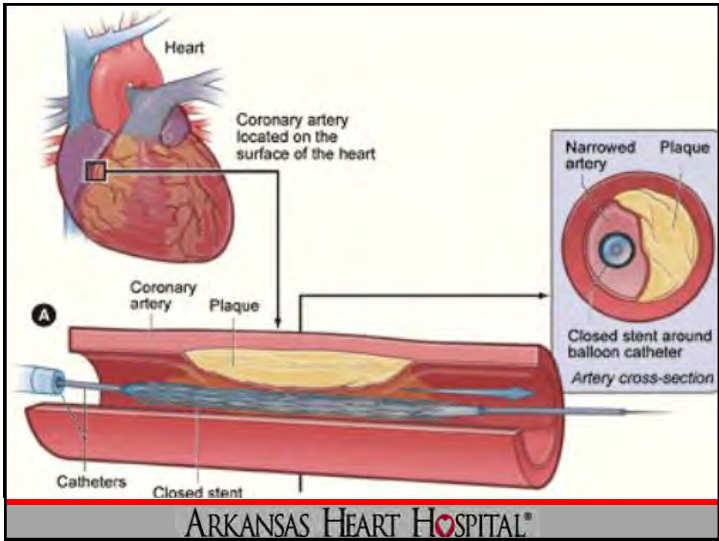
70



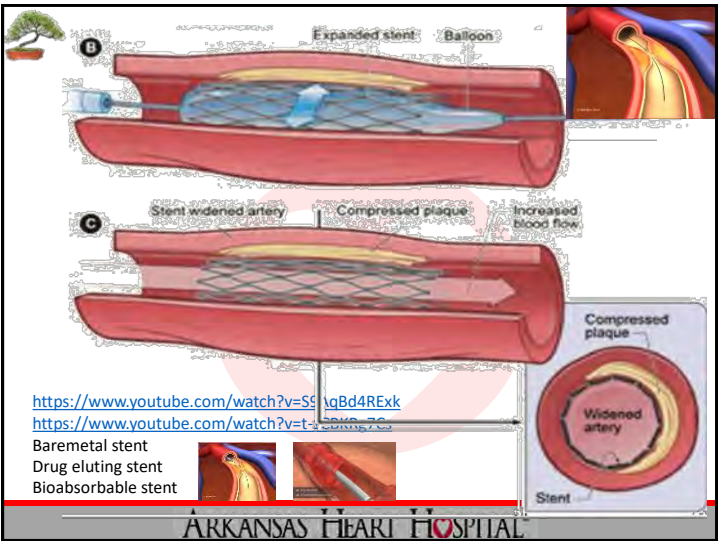
71



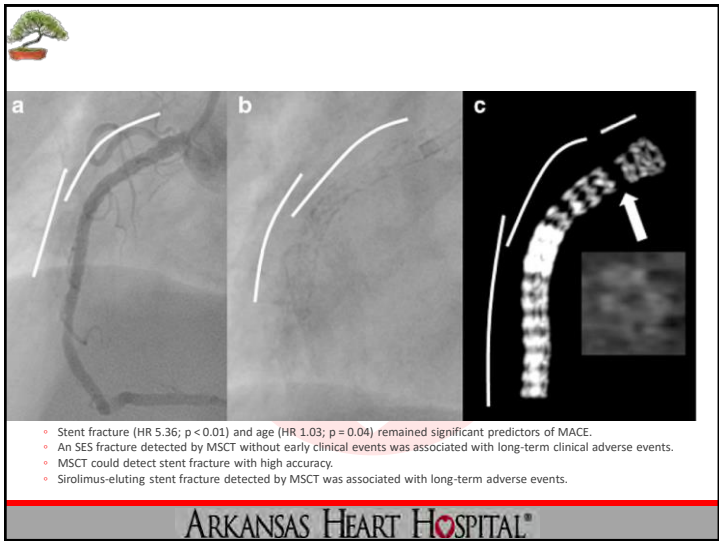
72



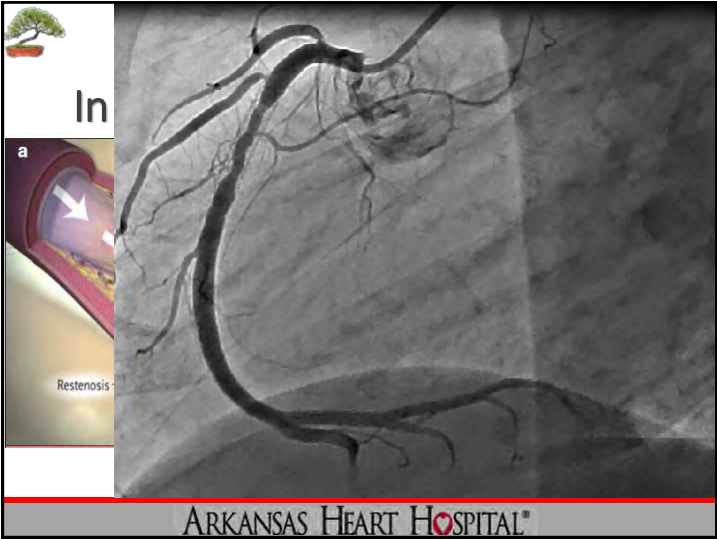
74



75



79



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

81

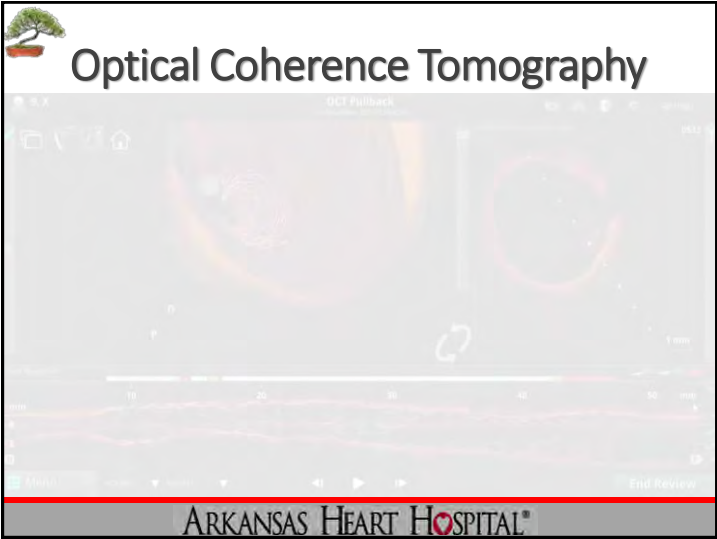
	DAPT coronary stent	TRIPLE THERAPY AF + stent*	DAPT cerebrovascular
Phase I: Initial Therapy The specialist will select the intended duration of therapy, & will specify if therapy is to be extended. Initial prescription is usually for:	Clopidogrel + ASA or Prasugrel + ASA or Ticagrelor + ASA x 12 months	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 note	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 ¹	 CHADS2 CHF, Hypertension Age ≥75 years Diabetes mellitus Stroke x2 HASBLED	ISCHEMIC STROKE 21 days of DAPT ↓ risk of stroke in a Chinese population ² DAPT >90 days ↑ risk of major bleeds & all-cause mortality ³

82

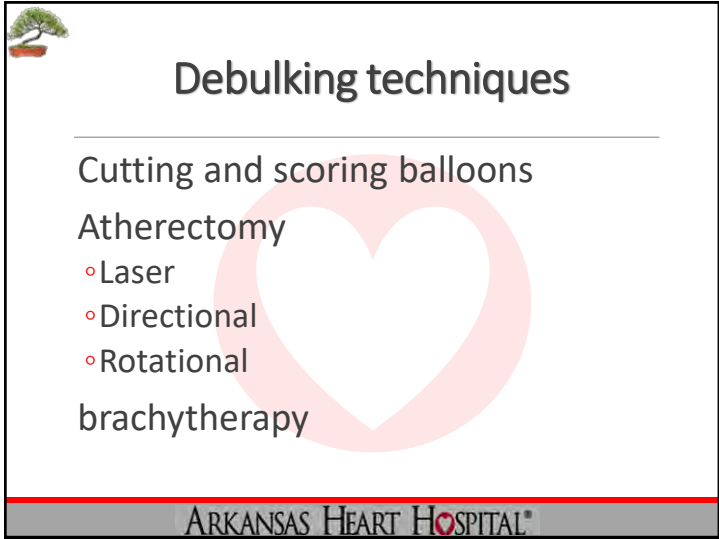
83

Section 8

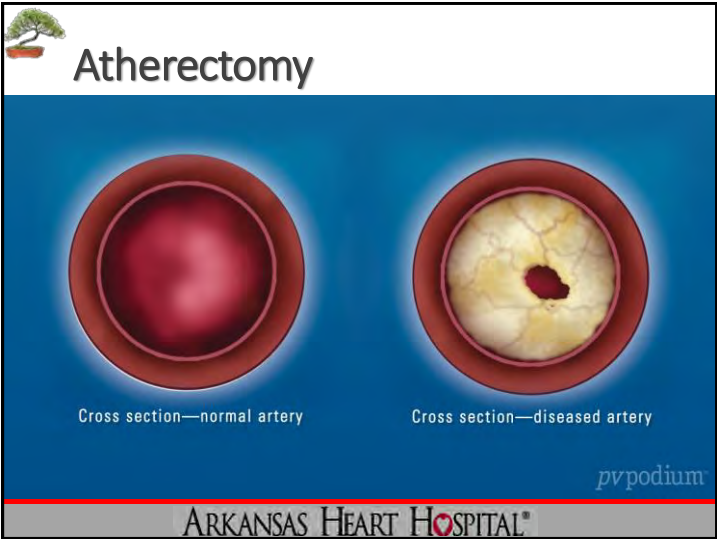
12



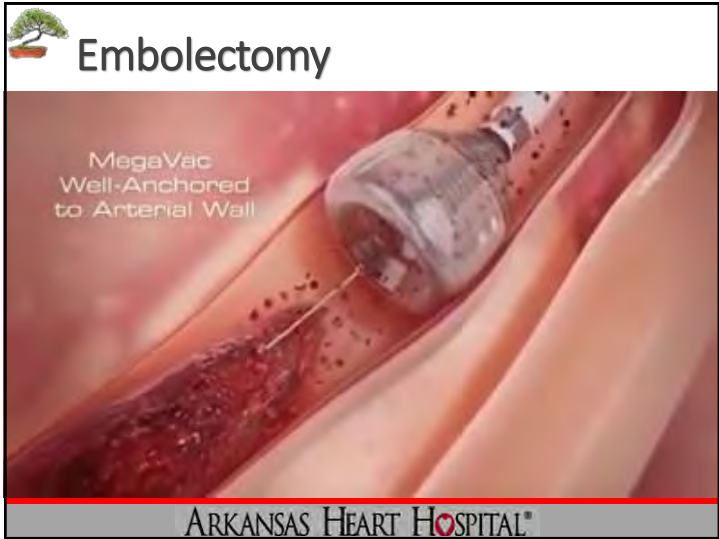
84



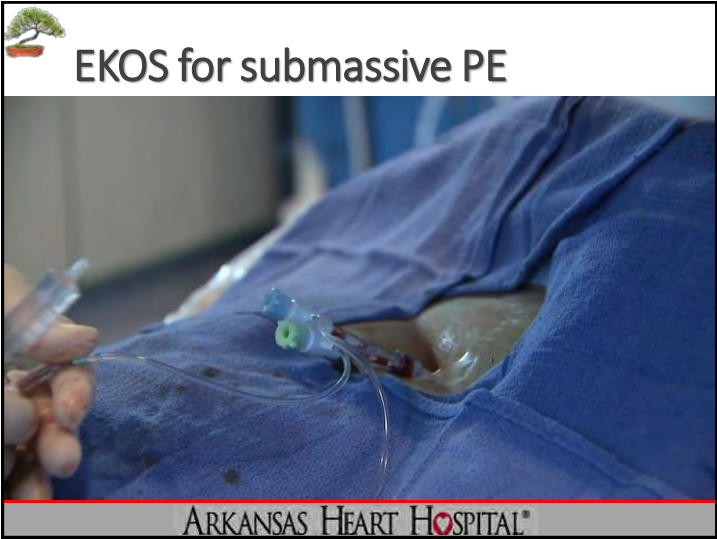
85



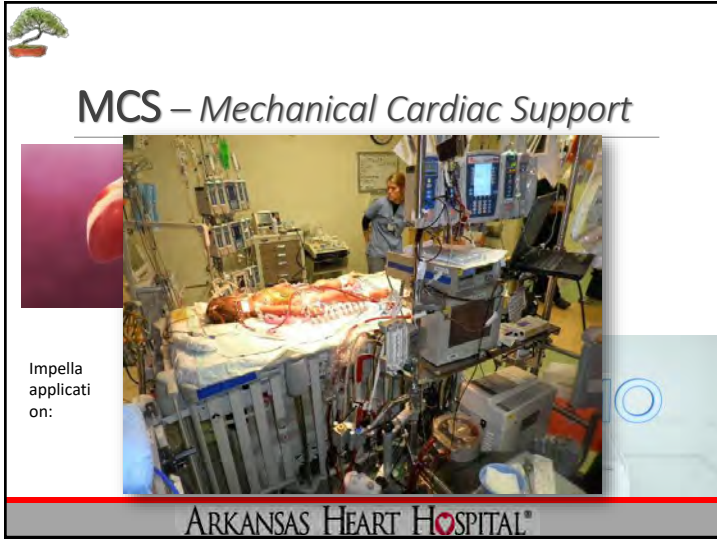
86



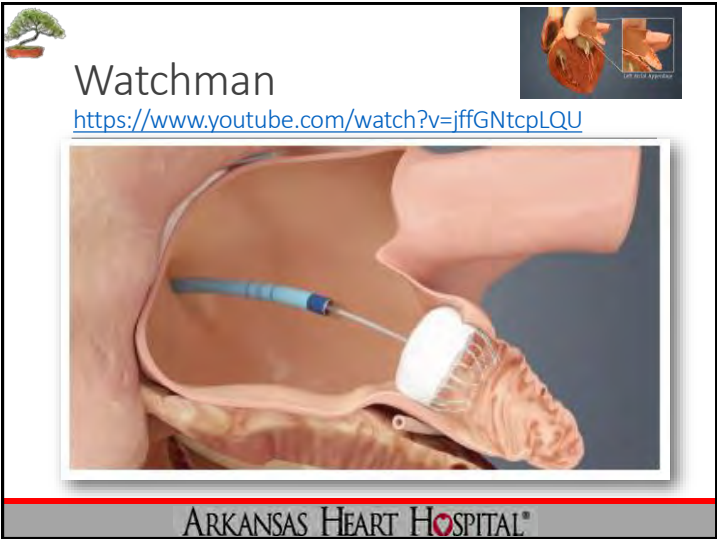
88



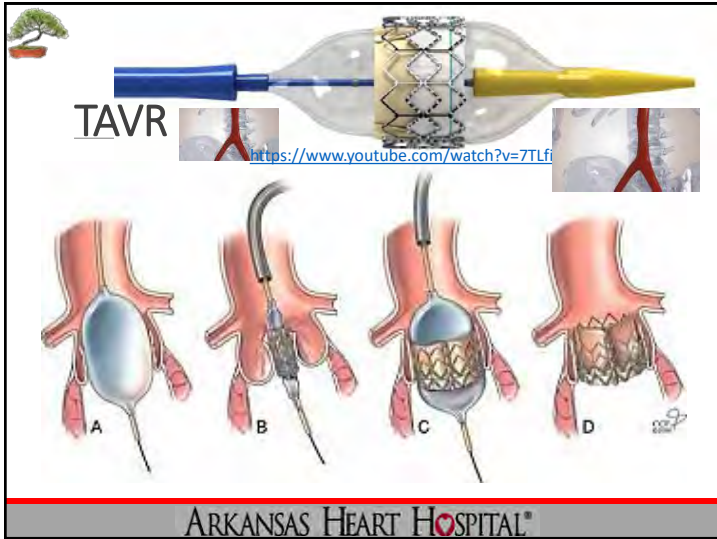
89



92



93



94



Complications

- ★ Heart Perforation
- ★ [Embolism](#)
- ★ Arrhythmias
- ★ [Allergic reaction](#)
- ★ MI
- ★ [Hematoma](#)
- ★ [Hemorrhage](#)
- ★ Infection
- ★ [Artery dissection](#)
- ★ DEATH



95



Post Procedure care

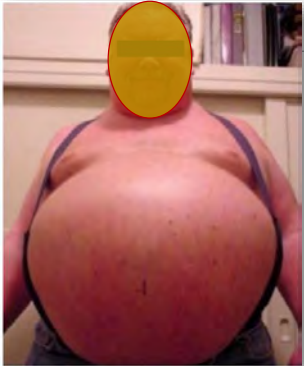
- ★ Anticoagulation
- ★ Care of femoral insertion site: distal circulation, use of femstop, bed rest.
- ★ Continued Cardiac Monitoring
- ★ Blood check: Coags
- ★ Cardiac Rehab



96



Every case begins and ends with Access!



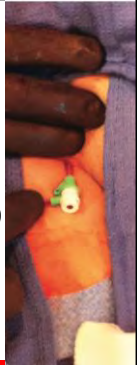
ARKANSAS HEART HOSPITAL®

97



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



ARKANSAS HEART HOSPITAL®

98



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

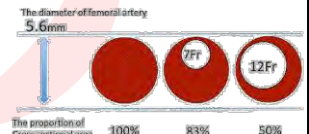
Impella, mitraclip 13-14fr
AVR, 10-12 fr

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

The diameter of femoral artery
5.6mm

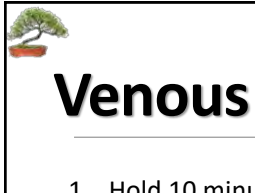
The proportional cross sectional area

100%	83%	50%
------	-----	-----



ARKANSAS HEART HOSPITAL®

99



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.

ARKANSAS HEART HOSPITAL®

100

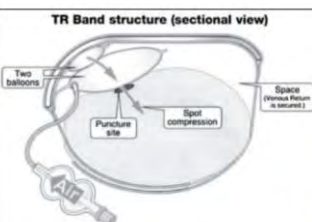


Radial Access

Access Site Care- Radial Artery

- TR band placed at termination of case

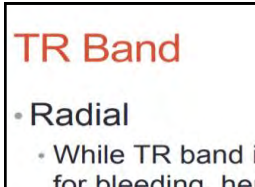
TR Band structure (sectional view)





ARKANSAS HEART HOSPITAL®

101



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

ARKANSAS HEART HOSPITAL®

102

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



ARKANSAS HEART HOSPITAL®

103



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%

ARKANSAS HEART HOSPITAL®

104



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)

ARKANSAS HEART HOSPITAL®

105



107



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.

ARKANSAS HEART HOSPITAL®

108



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

116

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.

118



Tilt Test

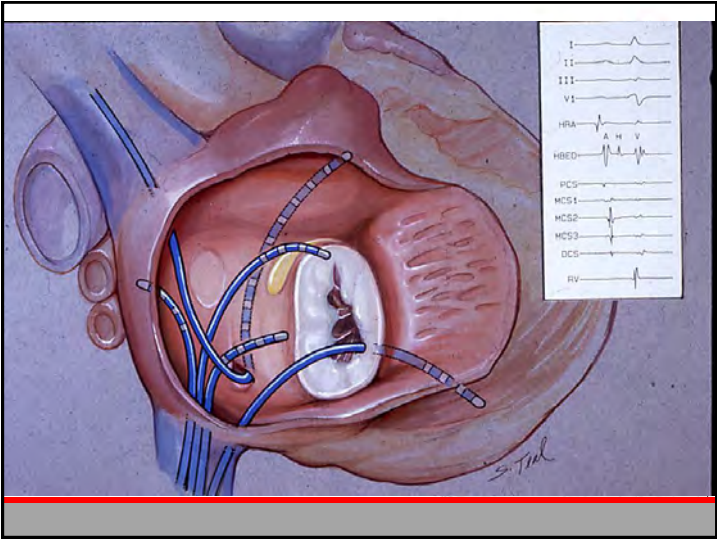
Response/diagnosis

Characteristics

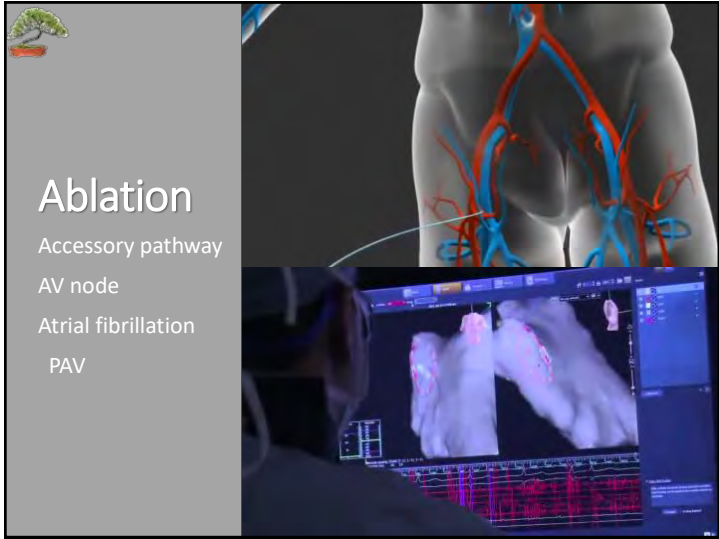


HEAD UP TILT PHASE 60° (20 mins)
PASSIVE PHASE

119



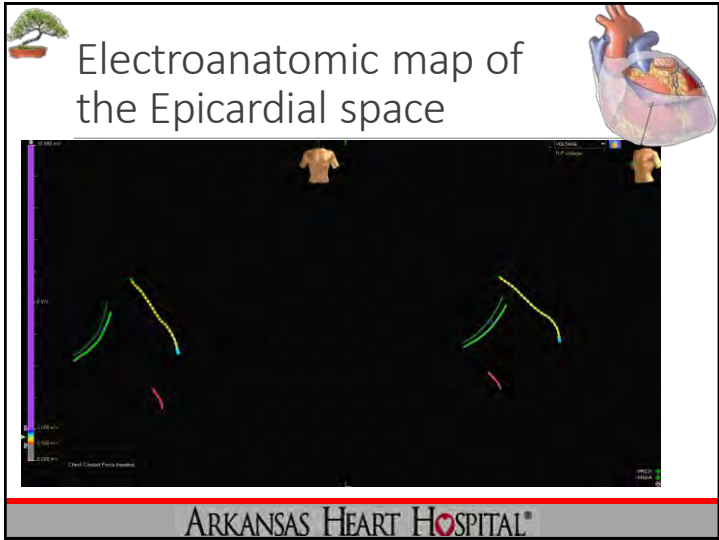
120



121



122



135

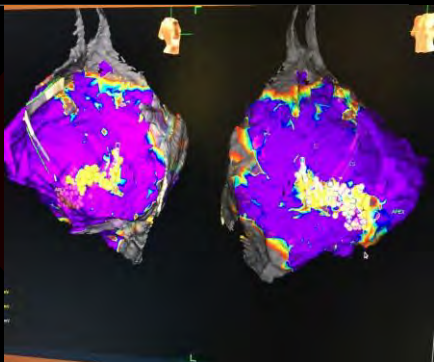


Final.

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.

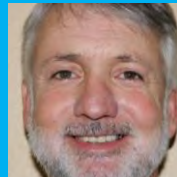


ARKANSAS HEART HOSPITAL®

139



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**

ARKANSAS HEART HOSPITAL®

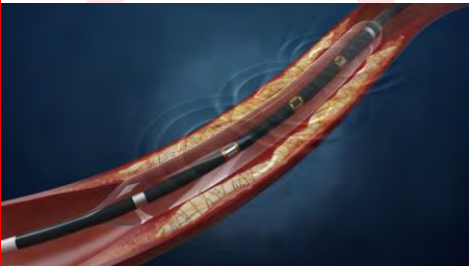
142



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



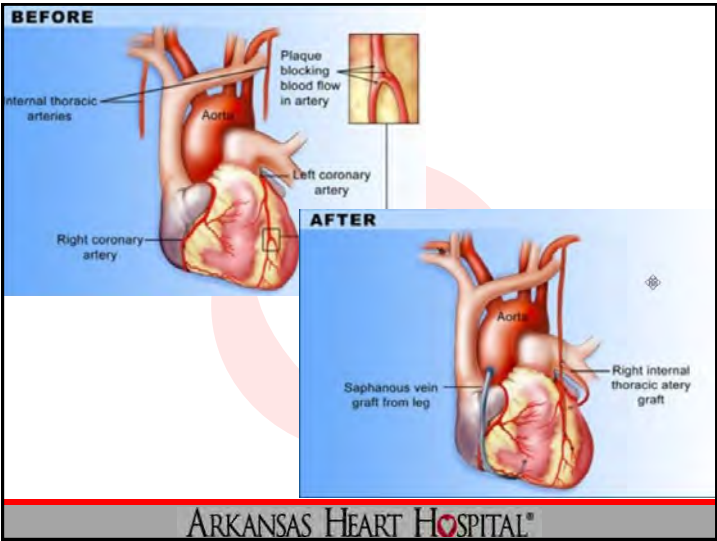
143



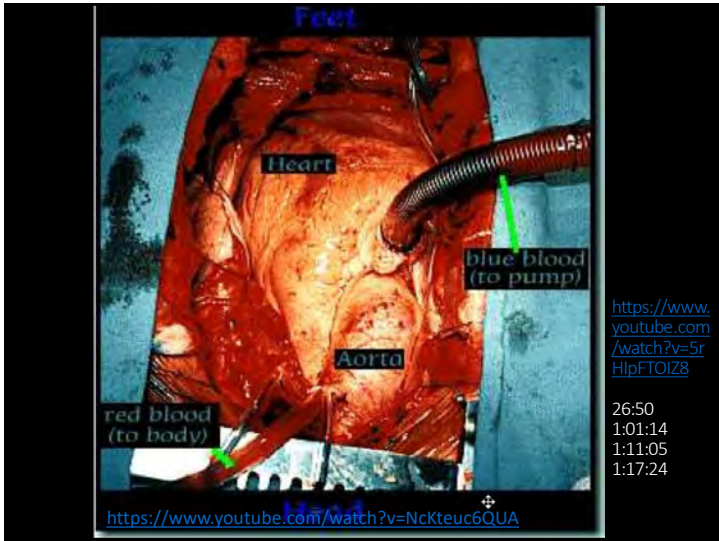
CABGs

- ★ 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

144



145



146

Post Op Care

Pulses – assess ALL pulses

- $\nearrow$ HR may indicate dysrhythmia, shock, fear, fever, hypoxia, CHF, or hemorrhage.
- Decreased pulses in extremities may indicate peripheral embolus

Body Temperature – slight elevation normal

- Temperature rise 1-1.5°C on 1st & 2nd POD is normal x 3-4D

Respirations – even, unlabored, equal

Arterial blood gases & chest x-rays as ordered.

Peripheral Pulses

The diagram shows a human body with pulse points labeled A through H. A is at the carotid artery, B is at the radial artery, C is at the brachial artery, D is at the femoral artery, E is at the popliteal artery, F is at the posterior tibial artery, G is at the anterior tibial artery, and H is at the pedal artery.

ARKANSAS HEART HOSPITAL®

147

Post-Op Care

Heart Sounds

- Pericardial Rubs - irritation and inflammation
- A S3 gallop - probably indicates hypervolemia
- S4 gallop - decreased compliance, HTN
- Absent or diminished heart sounds - Cardiac Tamponade.

Blood Pressure – Systolic >90 and <140

- Beware of Hypertension & Hypotension.
- If the MAP is <60 tissue perfusion to organs is compromised.
- If the MAP is >110 then it can exacerbate bleeding from the mediastinal tubes. It can also increase the left ventricular workload.

Pulse Oximetry - > 90%

The diagram shows a human heart with internal structures labeled. The right ventricle is on the left, and the left ventricle is on the right. The aorta is at the top, and the pulmonary artery is at the bottom.

ARKANSAS HEART HOSPITAL®

148



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](#)

ARKANSAS HEART HOSPITAL®

149



References

Jowett, N. I., & Thompson, D. R. (2007). *Comprehensive Coronary Care*. (4th ed.). London, UK: Bailliere Tindall Elsevier.

Murphy M.J., Berding C.B., "Use of Measurements of Myoglobin and Cardiac Troponins in the Diagnosis of Acute Myocardial Infarction". *Critical Care Nurse* 19:1, February 1999, pp 58-65.

National Heart Foundation. (2006). *Guidelines for the Management of acute coronary syndromes 2006*. *The Medical Journal of Australia*, 184(8), p. S12-S13.

Woods, S. L., Froelicher, E. S. S., Underhill Motzer, S., & Bridges, E. J. (2005). *Cardiac Nursing*. (5th ed.). Maryland, USA: Lippincott, Williams & Wilkins.

Theodoropoulos, K., Mehran, R. "Dual-Therapy Stenting: The Next Step in the Evolution of Stent Design" *Cardiology Today's Intervention*, May/June 2012

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](#)

Simova, I., [..tilt-table testing in syncope diagnosis and management](#), *European Society of Card V12*, No 35, Nov 2015

Makhija et. Al, Three dimensional epicardial mapping and ablation of recurrent noon-ishaemic 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/PMC3860797/](#)

Phillip Mumford, RCIS, Director Cath Lab 2012The 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](#)

ARKANSAS HEART HOSPITAL®

150

Pacemaker History

Pacemaker History

Pacemaker History

EKG before pacemaker

The First Pacemaker Implanted into a Patient

The First Pacemaker Implanted into a Patient

The First Pacemaker Implanted into a Patient

Deemakers

Pacemakers

Pacemakers

Pacemakers

Pacemakers

Pacemakers

Pacemakers

Pacemakers

Pacemakers

extension cord	(Lilabe; Belden)	had not evolved significantly	cardiac chambers	position to power
(Furman)				pacing circuits

extension cord	(Lilabe; Belden)	had not evolved significantly	cardiac chambers	position to power
(Furman)				pacing circuits

Pacemakers

Pacemakers

Pacemaker History

Pacemaker History

The First Pacemaker Implanted into a Patient

The First Pacemaker Implanted into a Patient

Pacemakers

Pacemakers

Location of Pacemaker Leads

- Temporary Pacing**
 - Transcatheter: 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



Pacemaker Terminology

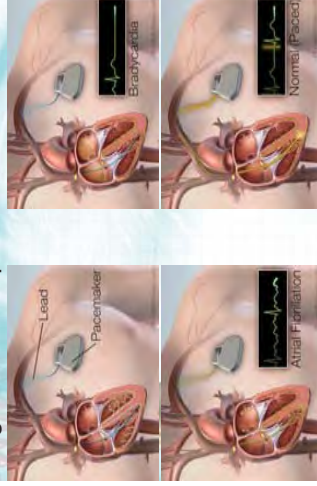
- Single chamber pacing: Only one lead in the heart; only one chamber being paced such as the right atrium or right ventricle.
 - Pacing the right atrium is called
 - Atrial pacing
 - Pacing the right ventricle is called
 - Ventricular pacing



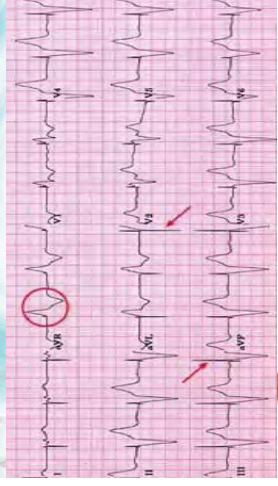
Permanent Generator



Single Chamber pacemaker

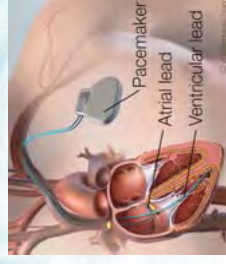


SINGLE PACEMAKER SPIKES

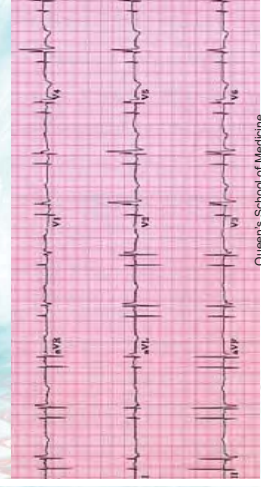


Queen's School of Medicine

Dual Chamber pacemaker



DOUBLE PACEMAKER SPIKES



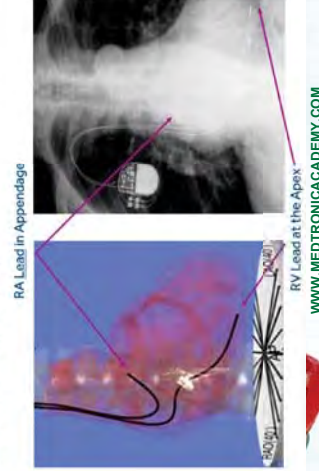
Queen's School of Medicine

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



DUAL CHAMBER PACEMAKER



WWW.MEDTRONICACADEMY.COM

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

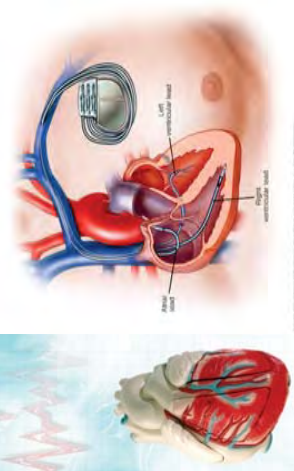


Equipment needed for temporary pacing:

- Generator
- Leads
- Patches



Biventricular Pacemaker



What type of pacemaker is this?



www.medtronicacademy.com

Transcutaneous Pacing

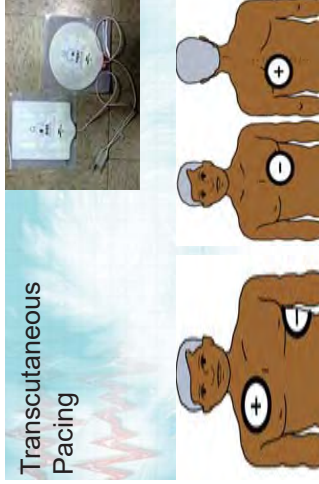


Temporary Pacemakers

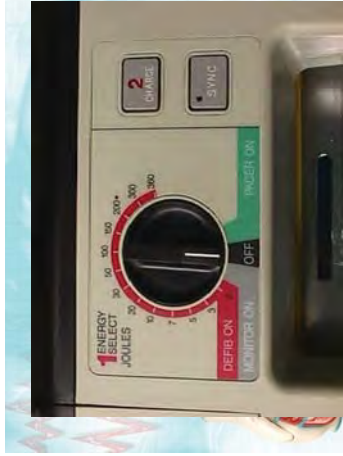
- Transcutaneous
- Epicardial
- Transvenous



Transcutaneous Pacing



Stimulating the heart with transcutaneous pacing. In female patients, position the negative pacing electrode under the breast. In male patients, position the negative pacing electrode under the breast. (Modified from illustrations supplied by Physio-Control, Inc., Redmond, WA.)



34

Temporary Generator (Transvenous or Epicardial)

Single Chamber or Dual Chambers



www.medtronicacademy.com

Epicardial Wires



37

Pacer cables

Disposable

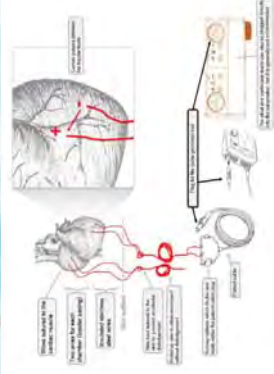


Non-disposable



37

Epicardial Wires



Anatomy of the temporary pacemaker circuit. Alan Yankov

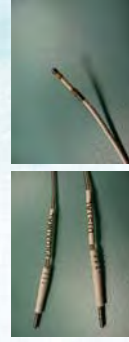
Transvenous Pacing



Insertion Sites

www.medtronicacademy.com

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.

42

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

44

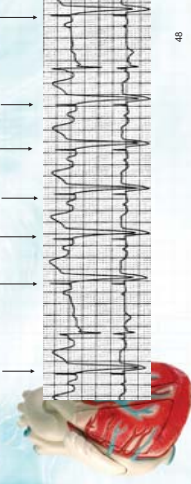
Universal Pacemaker Code

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

46

Pacemaker Identification

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



48

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

45

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.

47

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

Examples of pacing modes which are typically programmed:

DDD VVI DDIR VVIR



www.medtronicacademy.com

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**



49

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

A Pacemaker Rhythm

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



50

Interpreting a Pacemaker Rhythm

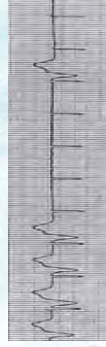
- Look for the "spike"
- Does spike precede a P wave or a QRS complex?
 - Measure rate of pacemaker
 - Measure from spike to spike
 - Is there appropriate function?
 - Firing, Capture, Sensing
- Interpretation: Ventricular Pacing, rate with complete capture; Comment on sensing if sensing can be evaluated.



52

Pacemaker 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



53

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



54

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



Benjamin J. Gold, MD, Dallas, TX. Emergency Medicine Residents' Association. 2017.

Pacemaker Rhythms

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



55

Pacemaker Malfunction

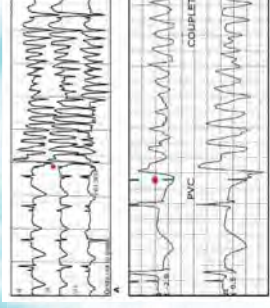
- Failure to Capture**
 - Capture is the successful depolarization of the heart by the pacemaker
 - Capture is recognized by a pacer spike which is followed by the appropriate wave form
 - P wave if atrial pacing
 - QRS if ventricular pacing
- Failure to capture (loss of capture) recognized as **pacer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



56

Pacemaker Malfunction

- Failure to Sense (Undersensing)**
 - Pacemaker spikes appear on the ECG
 - However, the generator has not sensed the intrinsic cardiac activity
 - Not generator has failed to recognize intrinsic cardiac activity



57

Benjamin J. Gold, MD, Dallas, TX. Emergency Medicine Residents' Association. 2017.

Pacemaker Malfunction

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



Benjamin J. Gold, MD, Dallas, TX. Emergency Medicine Residents' Association. 2017.

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.



58

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)



59

Implantation

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



60

Pacemakers

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



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



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



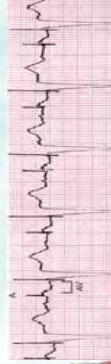
Post-Implant

- 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



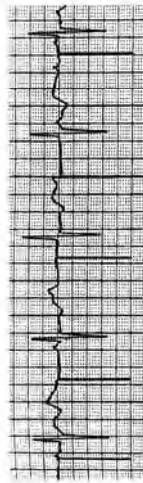
Pacemaker Rhythms

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



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?



WWW.MEDTRONICACADEMY.COM

Pacemaker Rhythms



Atrial Pacing



Ventricular Pacing



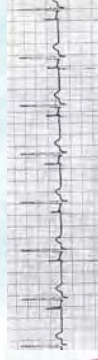
Pacemaker Rhythms

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



Pacemaker Rhythms

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

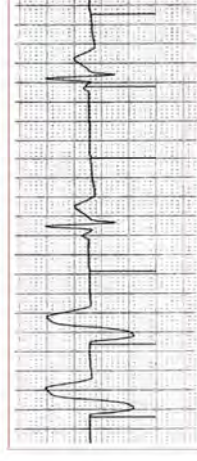


Pacemaker Rhythms

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



Gather Information and Identify the Problem



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

WWW.MEDTRONICACADEMY.COM

Gather Information and Identify the Problem



Pacemaker fails to emit stimuli at the programmed intervals

WWW.MEDTRONICACADEMY.COM

ATRIAL ELECTROGRAM

Indications:

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



http://booksite.elsevier.com/978032370654

WWW.MEDTRONICACADEMY.COM

ORDER A CHEST X-RAY

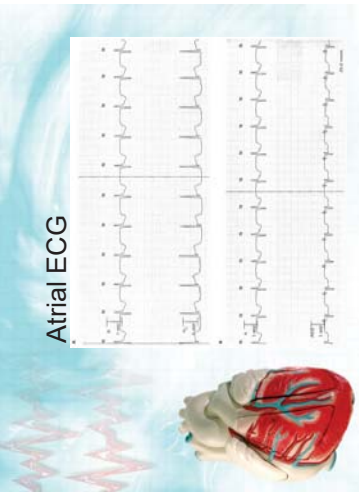
The chest x-ray revealed a dislodged lead



Atrial Electrocardiogram

- ## Atrial Electrocardiogram

Atrial ECG

[illegible]

- [illegible]

Hemodynamic Monitoring

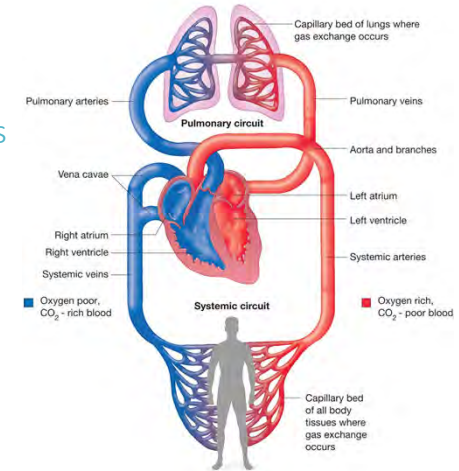
Kelly Urban, PhD, MEd, BSN, RN, CCRN-K, TCRN
University of Arkansas for Medical Sciences



1

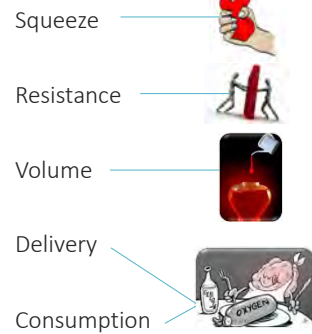
4 Factors that Affect the Hemodynamic Conditions

Myocardial Contraction
Heart Rate
Intravascular Volume
Vasoactivity



2

The main goal of Hemodynamic Optimization



3

Noninvasive Hemodynamic Monitoring

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



4

Invasive Hemodynamic Monitoring

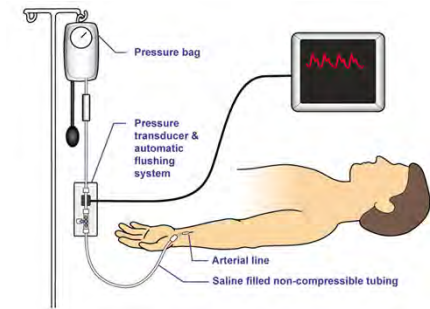
Arterial Blood Pressure
Central Venous Pressure
Pulmonary Artery Pressure



5

Pressure System Set-Up

Pressure Bag
Fluids
Pressure Transducer



6

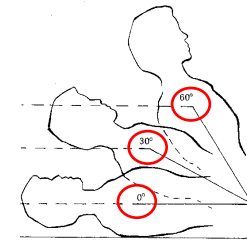
Phlebostatic Axis

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

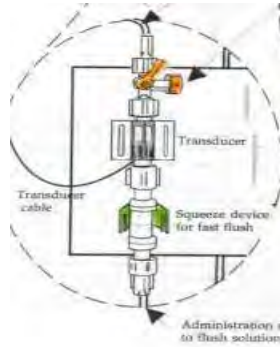
Accuracy

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

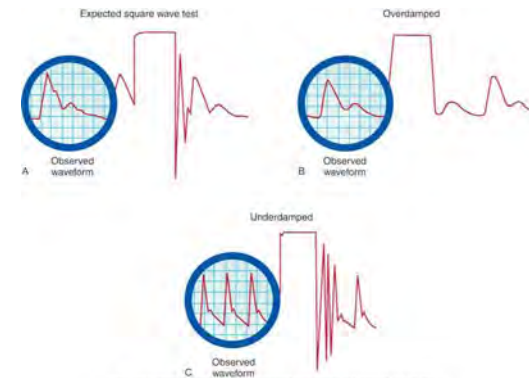
Maintaining continuous flush

- 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



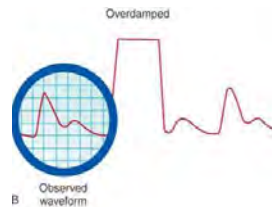
10

Overdamped: < 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



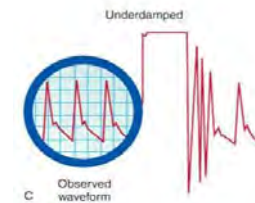
11

Under Damped: > 2 oscillations

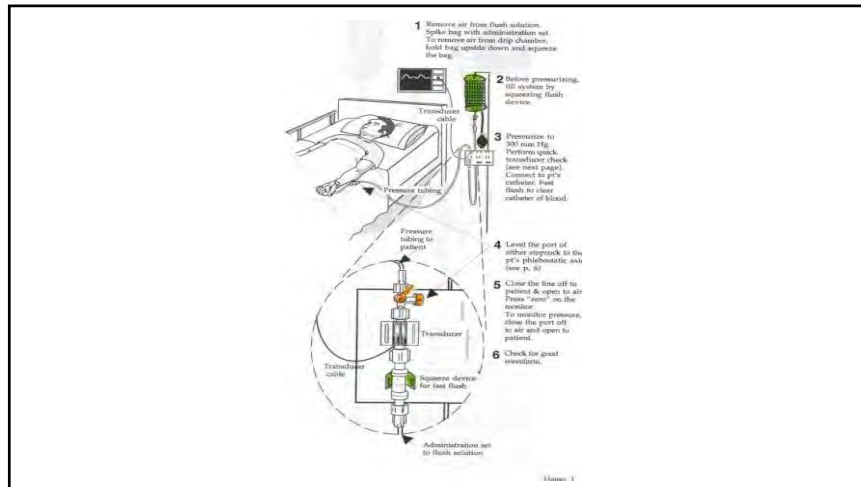
Results in erroneously high SBP and low DBP

Causes:

- Small air bubbles
- Tubing too long
- Defective transducer



12



13

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

14

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

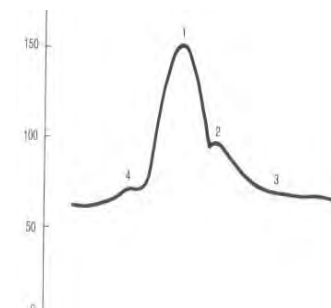
Peak Systolic Pressure

Dicrotic Notch

Diastolic Pressure

Anacrotic Notch

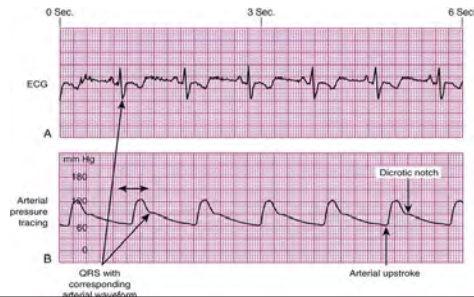
Pulse Pressure



16

A-line Waveforms

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



17

Normal Pressures

Systolic: 90-140 mmHg

Diastolic: 60-90 mm Hg

Mean: 80-100 mm Hg

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

$$\text{Example: } 120/80 = 120 + 160 \div 3 = 93$$

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

19

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

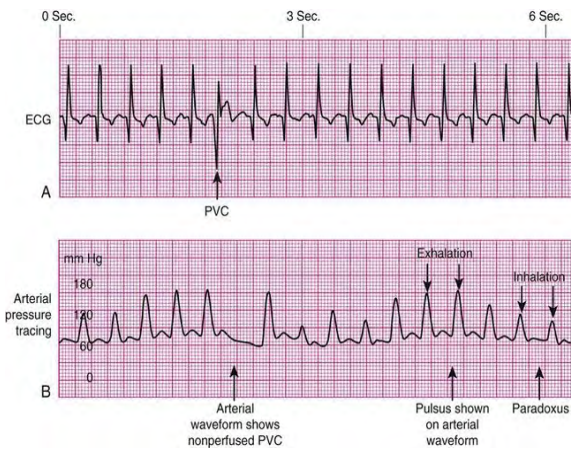


20

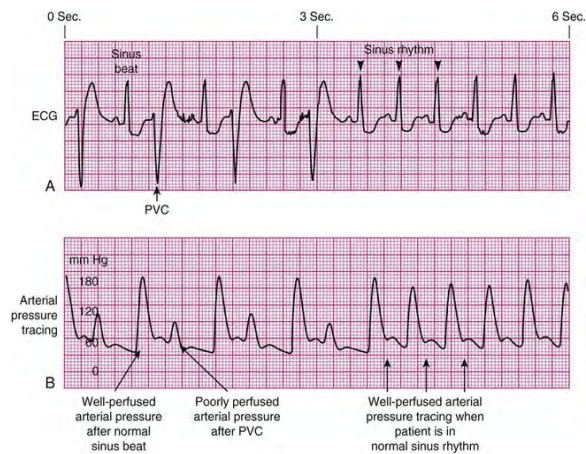
Complications

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

21



22



23



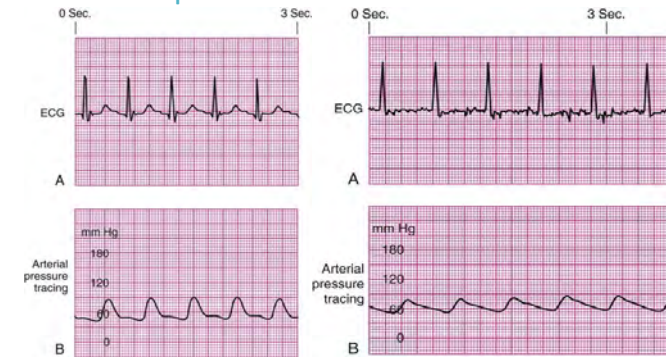
24

Pulsus Paradoxus



25

Low Pulse Amplitude or Dampened Wave Form

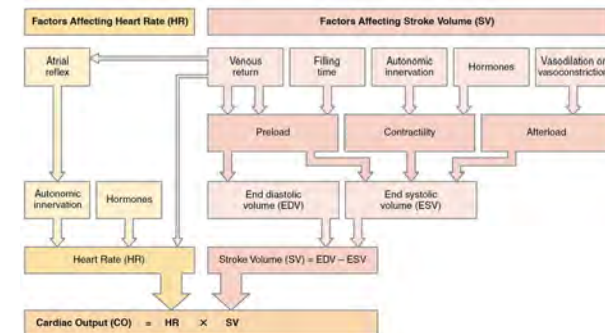


26



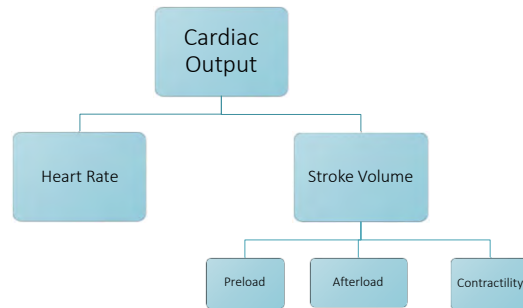
27

Cardiac Output



28

Cardiac Output



29

Cardiac Output Terminology

Cardiac Output (CO):

Amount of blood ejected from the ventricle in 1 minute

Measured in Liters/minute

$CO = \text{Heart Rate (HR)} \times \text{Stroke Volume (SV)}$

Normal CO: 4-8 L/min

Cardiac Index (CI):

CO / BSA (body surface area)

Normal CI: 2.5 – 4 L/min/M²

Hemodynamic Definition of Shock: $CI \leq 2$

30

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%

31

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

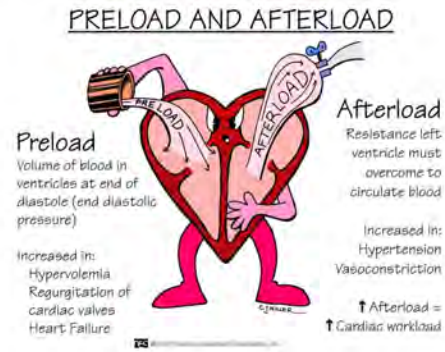
Preload

CVP: Right Ventricular Filling Pressure reflects

Volume
Ventricular Compliance
Valve Integrity (Tricuspid)

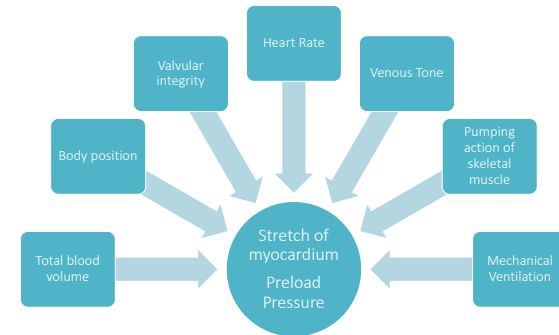
PAOP: Left Ventricular Filling Pressure reflects

Volume
Ventricular Compliance
Valve Integrity (Mitral)



33

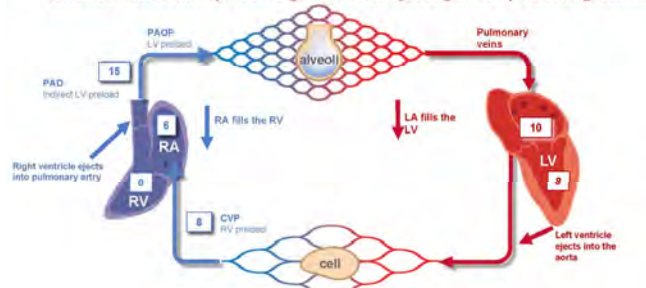
Preload – Filling the Ventricles Depends on



34

Preload

Ventricles fill down a pressure gradient and eject against a pressure gradient



35

Preload

VOLUME

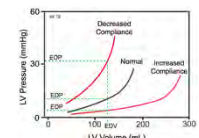
↑ filling volume = ↑ preload = ↑ stroke volume

↓ filling volume = ↓ preload = ↓ stroke volume

VENTRICULAR COMPLIANCE

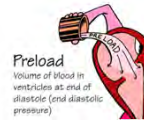
Normal Compliance: easy to fill
Large ↑ volume = small ↑ pressure

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



36

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)

37

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

38

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



39

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

40

Hepato-jugular Reflex



41

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

LOW

Hypovolemia
Tachycardia

HIGH

Right Ventricular Dysfunction
Pulmonary Hypertension
Tricuspid Valve Dysfunction
Volume Overload

42

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

LOW

Hypovolemia
Tachycardia

HIGH

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

PA Diastolic reflects the PAOP

43

↑ 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

44

Treatment of ↑ Left Preload

Diuretic to ↓ circulating volume

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

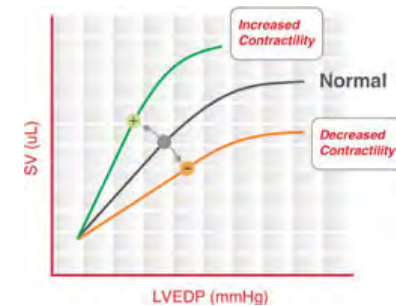
Positive Inotrope: increase strength of contraction (dobutamine)

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

45

Preload Responsiveness

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



46

Stroke Volume and Preload Responsiveness

300 -500 ml of saline=

Stroke Volume Variation > 15% = Preload Responsive

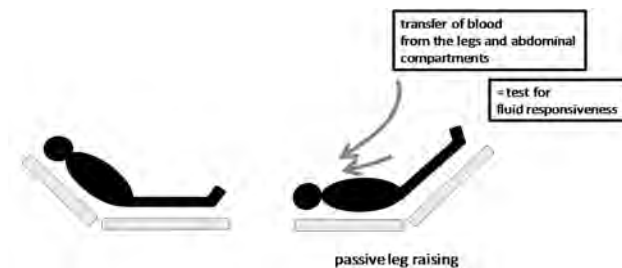
SV = 60 ml; after Fluid bolus SV = 90 ml

SVV % < 10% ≠ Preload Responsive

SV = 60 ml; after Fluid bolus, SV = 64 ml

47

Approx. 300 ml challenge = ↑ SV in 90 sec



48

Not measuring Stroke Volume?

Look at pulse pressure
 Systolic – diastolic = Pulse Pressure
 Normal is 30-40 mm Hg
 ↑ in Pulse Pressure = ↑ in stroke volume

49

SVV Normal < 13%

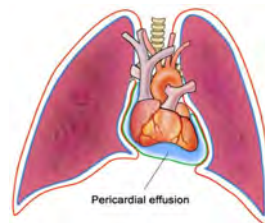
Limitations

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



51

Afterload

Tension developed by the myocardial muscle fibers during ventricular systolic ejection

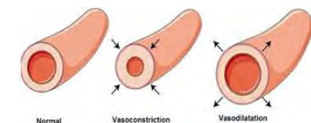
Described as resistance, impedance, or pressure that the ventricle must overcome to eject its blood volume

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



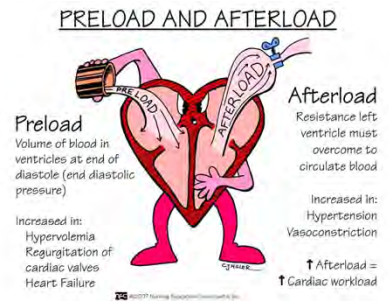
52

Afterload

Afterload has an inverse relationship to ventricular function

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

As resistance ↑, myocardial oxygen consumption ↑



53

SVR & PVR

$$\text{SVR} = \frac{\text{MAP} - \text{CVP}}{\text{CO}} \times 80$$

$$\text{PVR} = \frac{\text{MPAP} - \text{PAWP}}{\text{CO}} \times 80$$

MAP: Mean Arterial Pressure

MPAP: Mean Pulmonary Artery Pressure

54

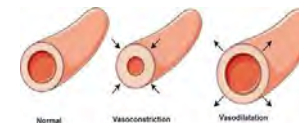
Afterload Affected By

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

55

Afterload



HIGH – VASOCONSTRICTION

Hypertension
Vasopressors
Aortic Stenosis
Hypothermia
Pulmonary Hypertension
Hypoxia
PE
Pulmonary Stenosis

LOW - VASODILATION

Distributive Shock states
Vasodilators
IABP

56

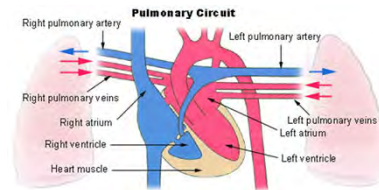
Right Ventricular Afterload (PVR)

HIGH

Pulmonary Embolism
Pulmonary hypertension
Left ventricular failure

LOW

Lysis of Pulmonary Embolism
Treatment of $\uparrow$ PVR



57

Left Ventricular Afterload (SVR)

HIGH

Vasoconstriction
Vasopressors
Hypertension
Compensatory mechanism for $\downarrow$ CO states
(hypovolemia/cardiogenic shock)

Aortic Valve Stenosis/ aortic aneurysm

Miss-timed IABP

LOW

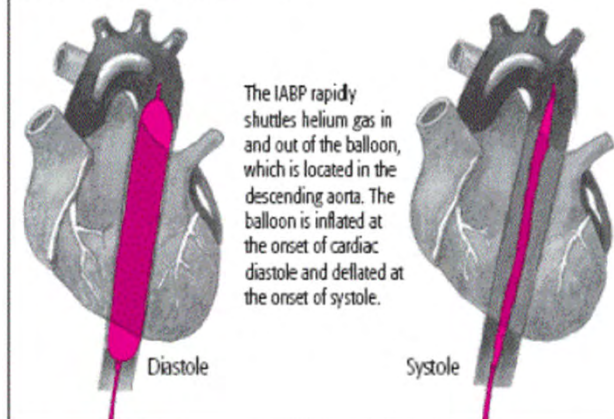
Vasodilation
Nitride /NTG drips

Distributive Shock States
Anaphylaxis
Sepsis
Neurogenic

Intra-Aortic Balloon Pump

58

The ins and outs of the IABP



59

Treatment of High Afterload

Medication to vasodilate

Surgery to correct aortic stenosis/ aneurysm

$\uparrow$ is due to $\downarrow$ CO, treatment is aimed at
 $\uparrow$ CO (positive inotrope, etc)

60

Treatment of Low Afterload

Medications for vasoconstriction

61

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



62

Contractility

Pumping ability of the heart

Estimated by Stroke Volume

$SV = 60-130 \text{ ml}$

$SV = CO \div HR$



63

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



64

Signs & Symptoms

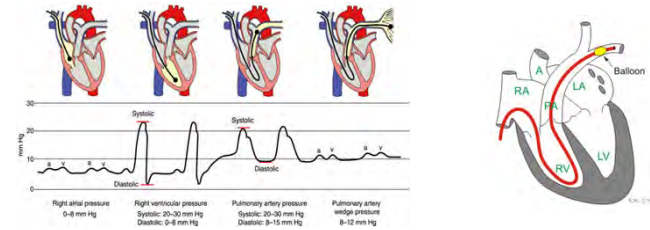
Too much contractility: chest pain, tachycardia

Too little: Heart Failure, Pulmonary Edema, decreased perfusion

65

Pulmonary Artery Pressure Monitoring

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



66

Pulmonary Artery Pressure Monitoring

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

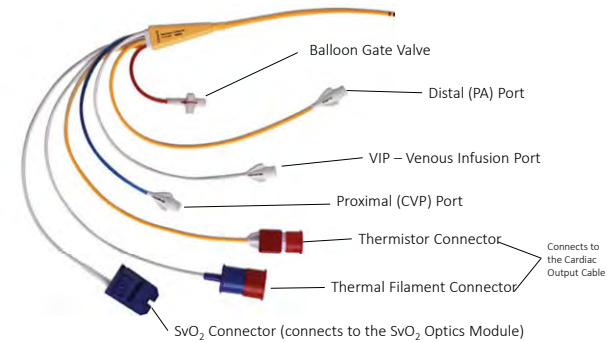
Indirectly reflects left ventricular end-diastolic pressure (LVEDP), which indicates left ventricular function

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



67

Pulmonary Artery Catheter Components



68

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

69

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

70

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

71

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)

72

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

73

Preparation for Insertion of Pulmonary Artery Catheter

- Zero & Calibrate bedside monitor
- Check balloon integrity
- Assist MD with introducer insertion
- Calibrate fiberoptics (SvO₂) – 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.

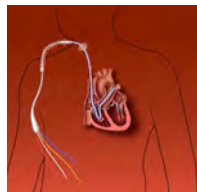
74

Preparation for Insertion of Pulmonary Artery Catheter

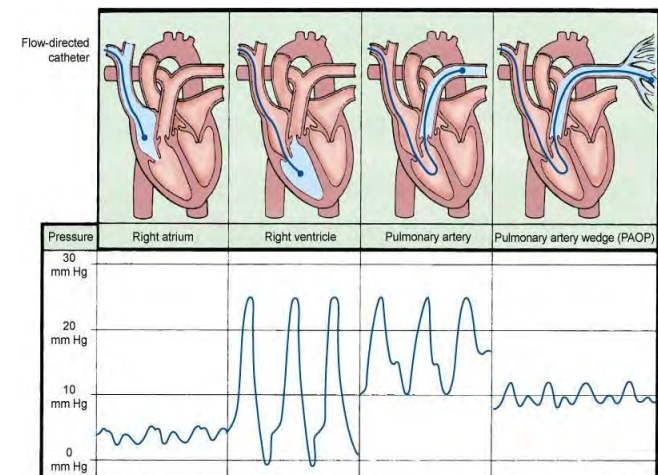
Catheter advancement to the pulmonary artery should be rapid, since prolonged manipulation can result in loss of catheter stiffness

Common sites for percutaneous approach include:

- Internal jugular
- Subclavian vein
- Femoral vein



75

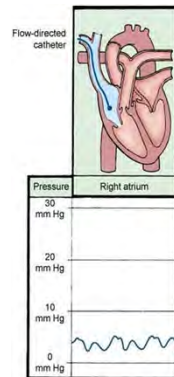
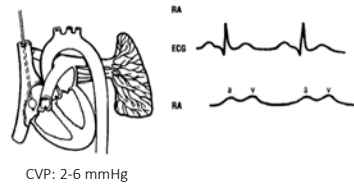


76

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.



77

Insertion of Pulmonary Artery Catheter: Right Ventricle

The next chamber is the right ventricle

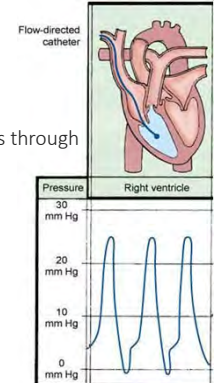
Waveforms show taller, sharp uprisers and low diastolic dips

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



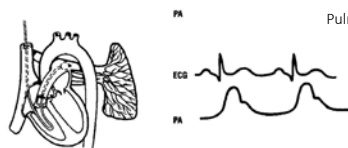
78

Insertion of Pulmonary Artery Catheter: Pulmonary Artery

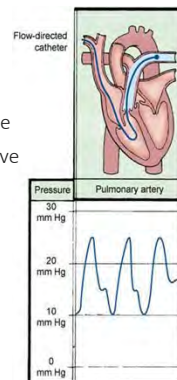
As the catheter floats into the pulmonary artery, characteristic waveforms can again be noted

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



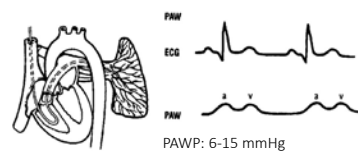
79

Insertion of Pulmonary Artery Catheter: Wedge

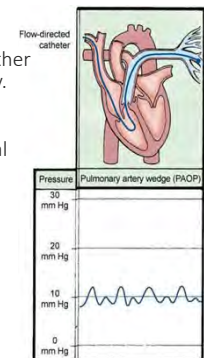
The catheter (with the balloon still inflated) is now advanced further until it finally wedges in a central branch of the pulmonary artery.

Waveform seen is a reflection of the left atrium

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



PAWP: 6-15 mmHg



80

Insertion of Pulmonary Artery Catheter



81

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)

86

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

87

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

89

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.)

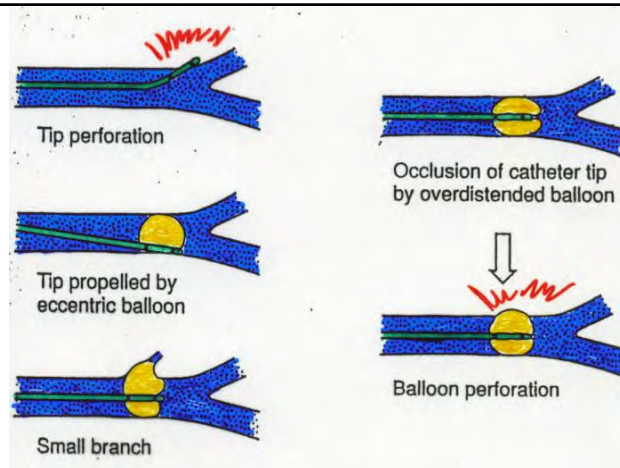
Bleedbacks: caused by leaks in the PA catheter apparatus or a pressure bag that is inflated below 300mm Hg

- 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



91

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

92

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

93

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*

94

Online Resources

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

95

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

96

Hemodynamic Applications

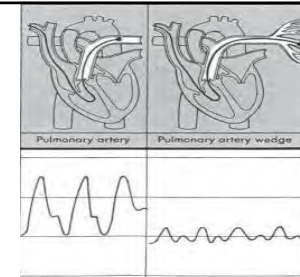
You would expect a Dobutrex (dobutamine) drip to increase _____

You would expect a Nipride drip to decrease _____

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

97

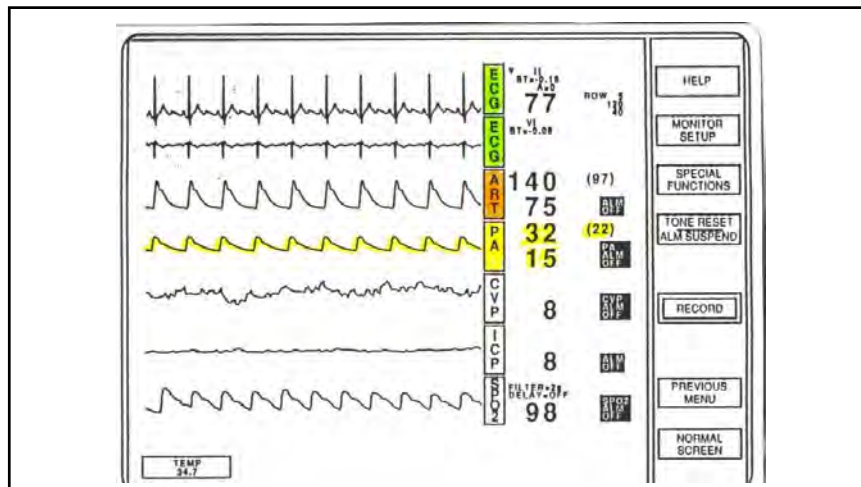
What pressure usually estimates the PAWP?



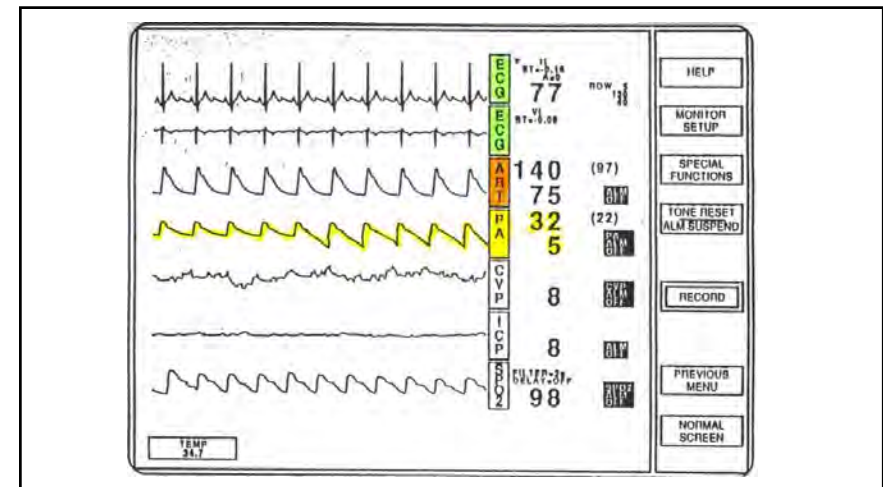
What landmark estimates the level of the right atrium?



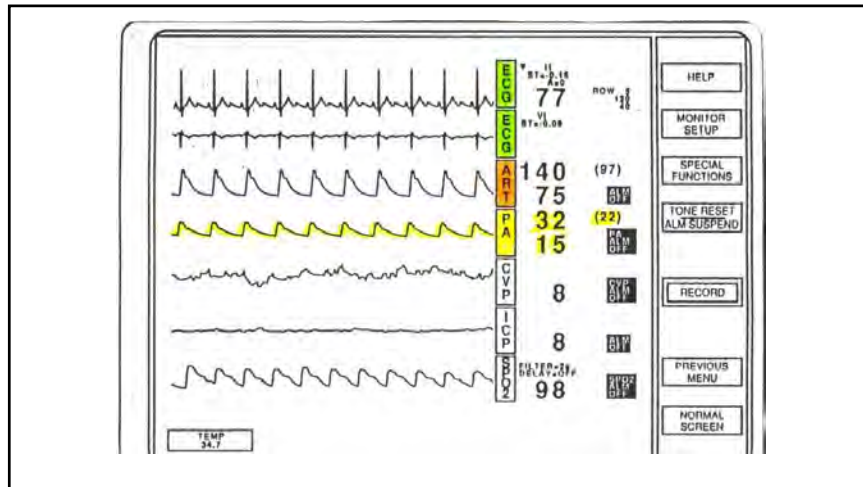
98



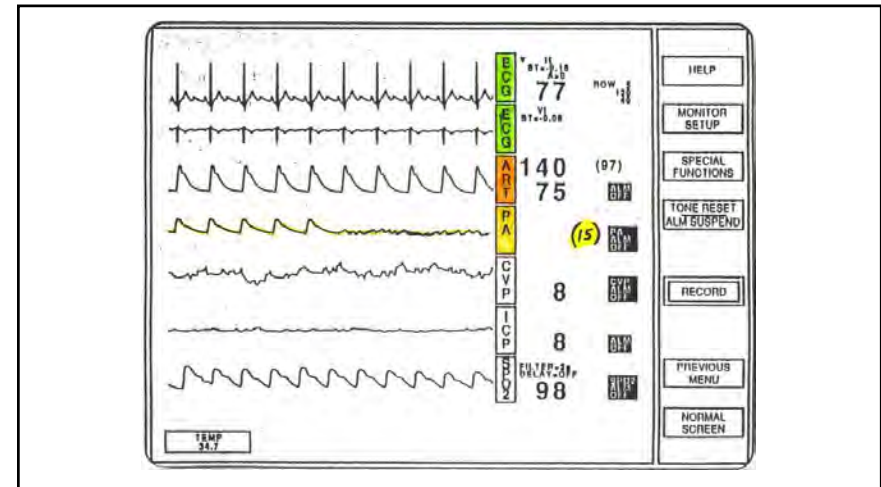
99



100



101



102

Review

Hemodynamic definition of Shock?

103

Review

Hemodynamic definition of Shock?
Cardiac Index (CI) ≤ 2

104

Review

Increase Preload with:

105

Review

Increase Preload with:

↓ circulating volume: fluids

Vasodilation: fluids, vasoconstrictor drip

106

Review

Decrease Preload with:

107

Review

Decrease Preload with:

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

108

Review

Hemodynamic definition of Heart Failure?

109

Review

Hemodynamic definition of Heart Failure?

PAWP 25

CVP 18

110

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?

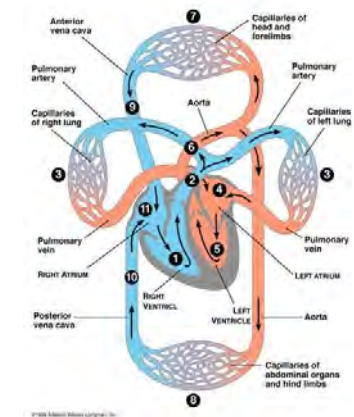
Work of Heart?

Oxygen consumption of heart?

Anxiety (sympathetic nervous system)?

111

Questions?



112

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.

