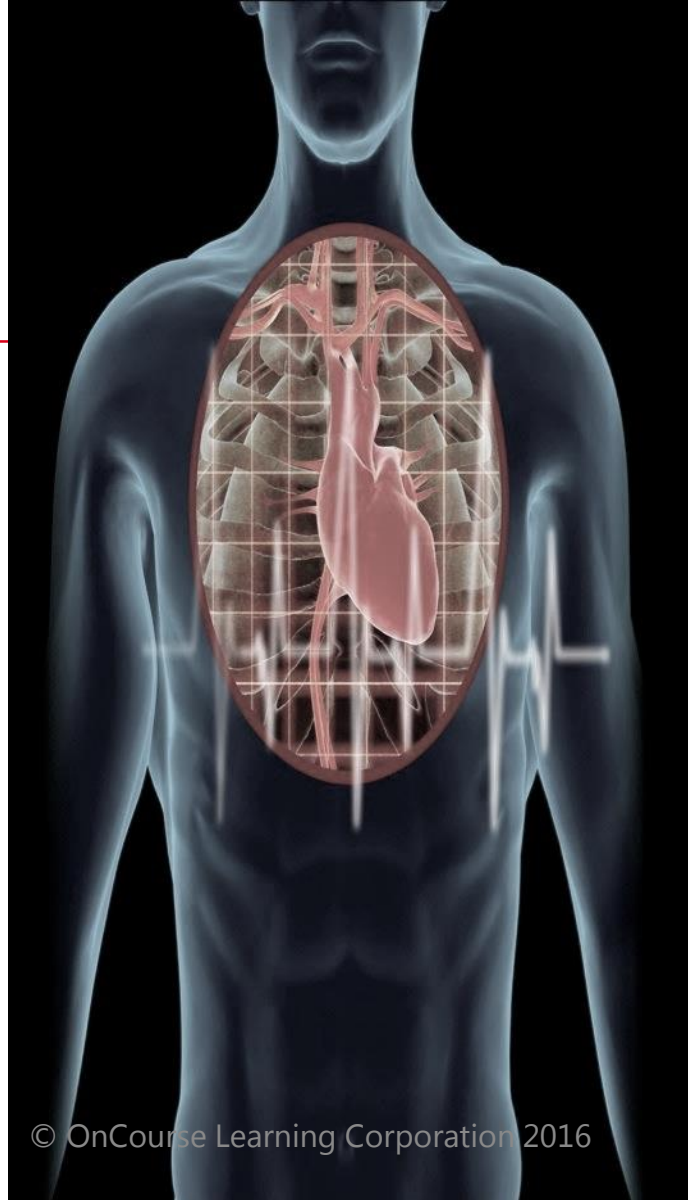
CCRN
CARDIOVASCULAR
PCCN

Essentials of Care: Vital Signs

- Heart rate: What causes tachycardia?
  - Fever, hypovolemia or hypervolemia
  - Almost anything that turns on the sympathetic nervous system will cause tachycardia
- But the physiological reason for tachycardia is tissue hypoxia



Vital Signs

- Respiratory rate: What causes increased respiratory rate?
  - The same triggers that stimulate the sympathetic nervous system may increase respiratory rate
- But the two major physiological reasons for increased respiratory rate are:
  - Tissue hypoxia
  - Metabolic acidosis

Vital Signs

Temperature:–

- Increased temperature
  - Causes increased oxygen demand of all tissues
- Decreased temperature
  - Causes abnormalities in metabolism, vasoconstriction and coagulopathies

Essentials: Blood Pressure

- Systolic: reflects stroke volume
- Diastolic: reflects arterial tone
  - And capillary blood flow
  - With vasoconstriction, diastole increases.
  - With vasodilation, diastole decreases.
- **MAP = [SBP+ 2(DBP)] divided by 3**

Blood Pressure

- Pulse pressure: the difference between systole and diastole,
  - Normal is 35 to 45 mmHg and therefore
  - Reflects LV performance
- For the test, this will help distinguishing different types of shock:
  - Neurogenic (wide pulse pressure) vs.
  - Cardiogenic shock (narrow pulse pressure)

Low Cardiac Output Syndrome

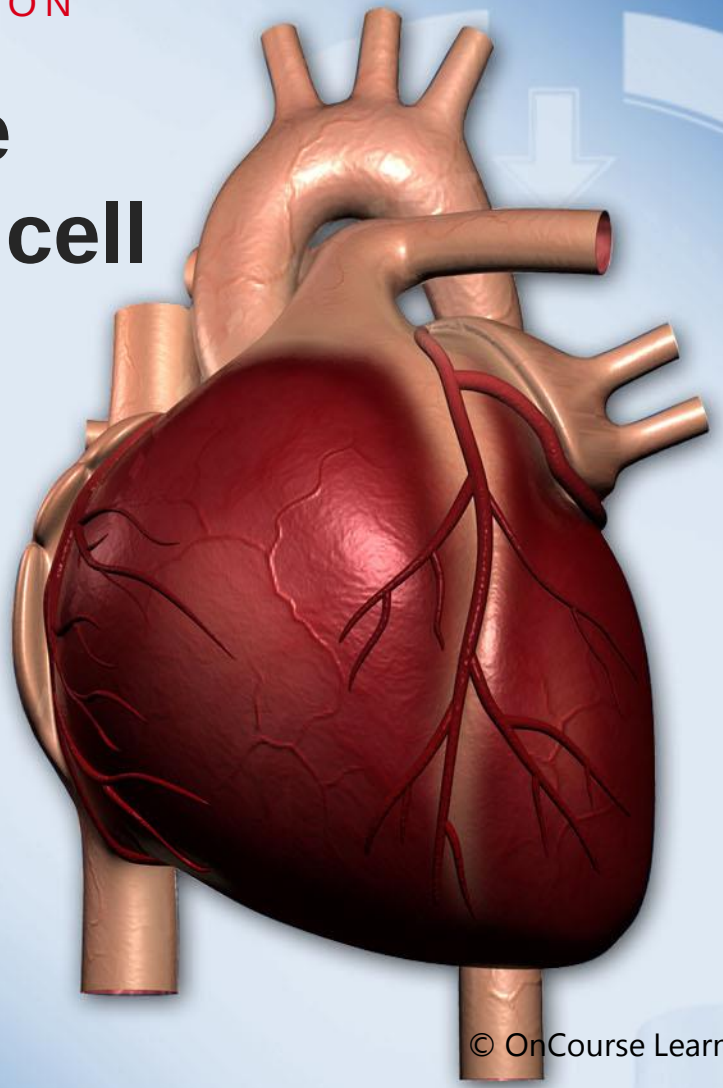
- What does this patient look like?
  - Tachycardia and vasoconstriction
  - Decreased mental status
  - Cool, cold and wet skin (vasoconstriction of the skin)
  - Decreased urine output
  - Narrowed pulse pressure
  - Pale
  - Change in mental status

Essentials

- So what we do for a living in the ICU/PCU is to enhance cardiac output and maintain perfusion
- GOAL for all patients is to:
  - Enhance O2 delivery
  - Decrease O2 demand

Purpose is to drive hemoglobin to the cell

Perfusion Assessment



The Right Ventricle

- Smaller chamber, septum does not improve function of the right ventricle; only the left
- Lower pressure
- Purpose is to pump blood from the right ventricle through the pulmonary vault and fill the left ventricle.

The Right Ventricle

- What happens in acute right ventricular failure?
  - The ventricle dilates and cannot pump blood from right through the pulmonary vault and "backs up" into the right atrium and SVC and IVC:
  - See JVD and later hepatic congestion
- ***NO pulmonary edema in acute right ventricular failure!***

The Right Ventricle

- How to treat RV failure?
  - Volume resuscitation
- Always treat RV failure with fluid: remember no pulmonary edema
- So early signs of RV failure:
  - Tachycardia, S3 and clear lungs
- Give fluid.

The Left Ventricle

- Thicker, larger ventricle with more muscle
- Pumping oxygenated blood from the heart to the arterial system
- Higher pressure! Corkscrew performance

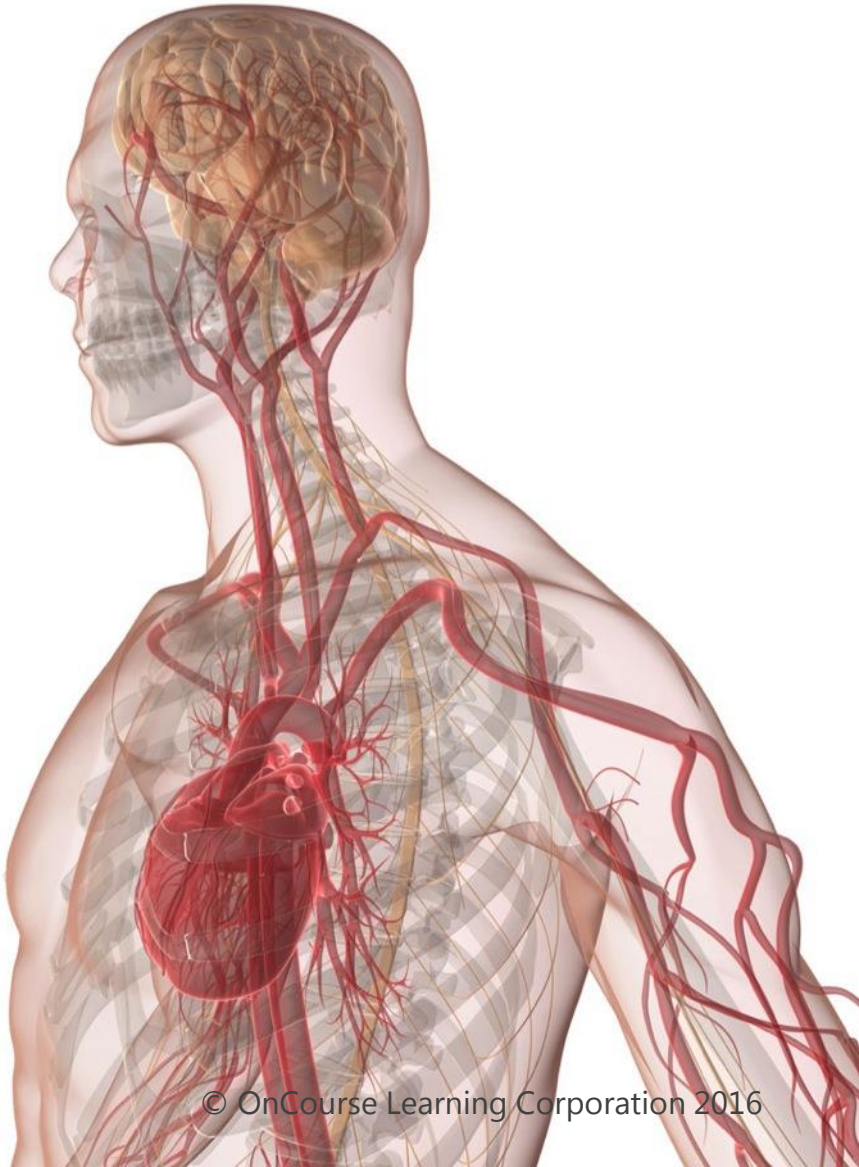
The Left Ventricle

- What happens when the left ventricle fails?
- Pulmonary edema: ventricle dilates,
  - Cannot pump blood through the aorta
  - Blood “backs up” into the left atrium and to the lungs. Pulmonary edema
- Early signs of LV failure:
  - Tachycardia, S3 and pulmonary congestion
  - Low cardiac output syndrome

The Left Ventricle

- Treatment:
  - Decrease intake of fluids
  - Decrease preload: diuresis
  - Decrease afterload: vasodilation
  - Possibly inotropic support for contractility

Coronary Circulation

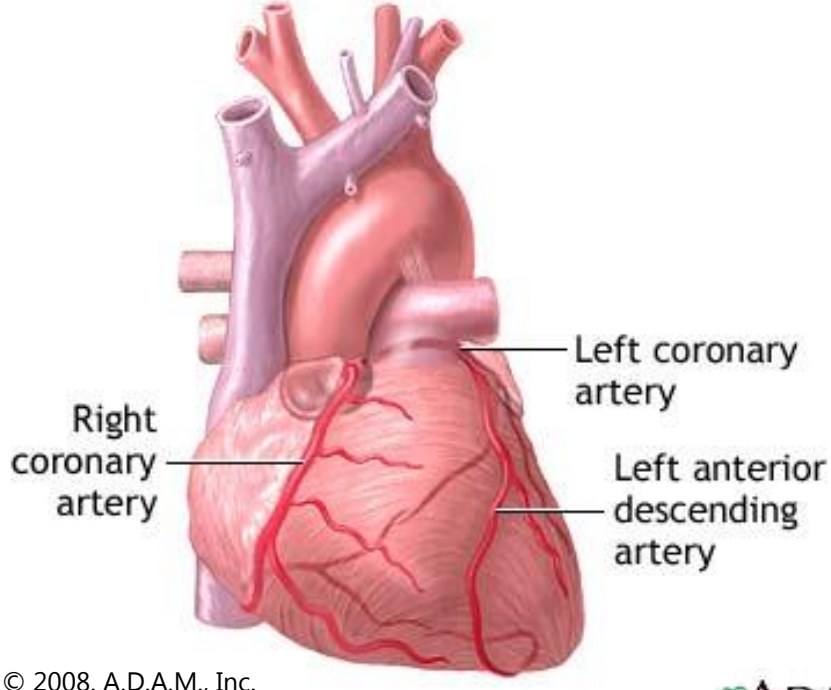


Coronary Circulation

- Right coronary artery: RCA
  - Feeds right atrium and right ventricle
  - In 90% of all people the RCA is dominate: It crosses the ventricular groove inferiorly to supply blood to the inferior left ventricle.
- In an inferior wall STEMI (ST elevation myocardial infarction), it is the RCA that has an occlusion

Coronary Artery Circulation

- Left main
  - Left circumflex: feeds left atrium and high anterior and lateral left ventricle
  - Left anterior descending: feeds the entire left anterior wall, 2/3 ventricular septum and the apex of the left ventricle



Coronary Perfusion

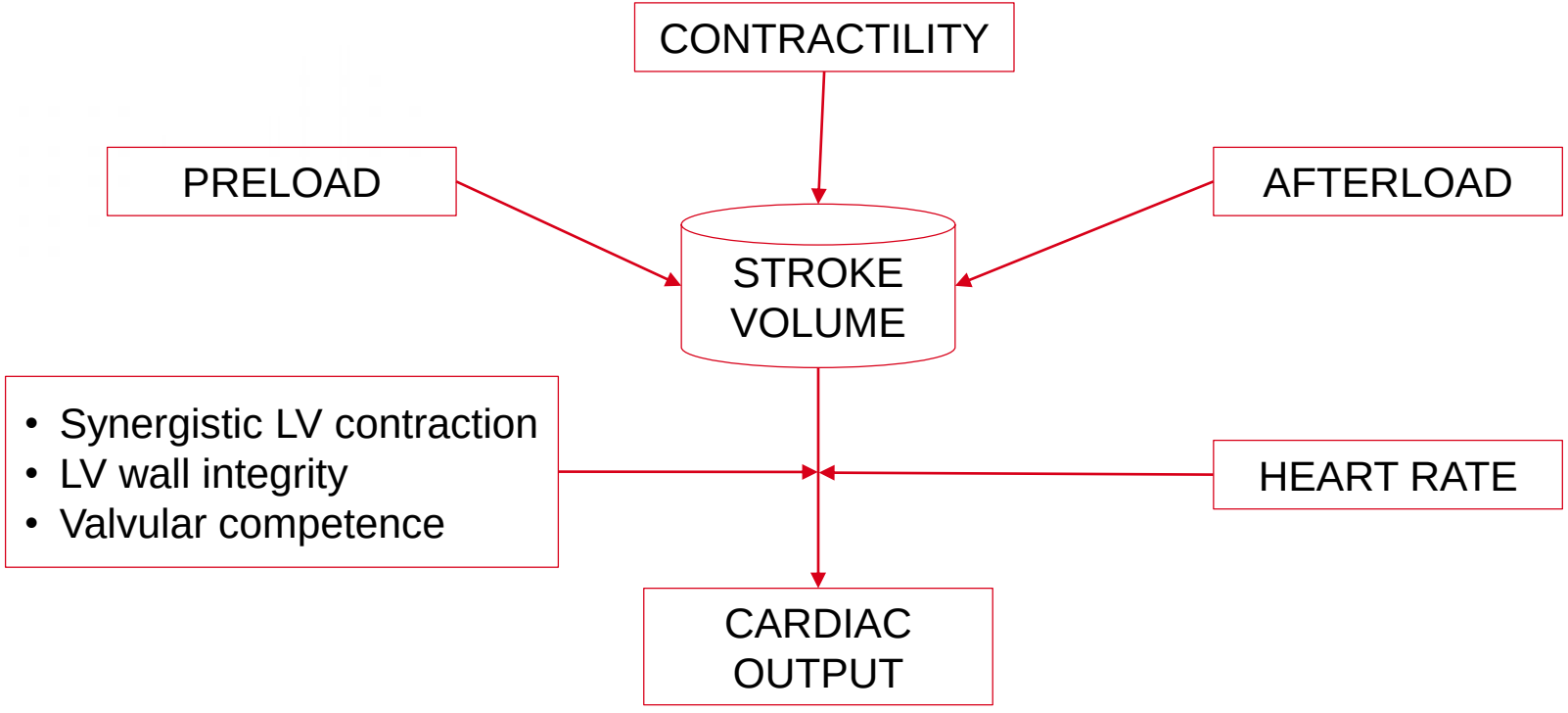
- Cardiac cycle: coronary artery perfusion depends on diastolic time
- Aortic pressure: coronary artery perfusion depends on aortic diastolic pressure
- Coronary artery perfusion pressure
  - CAPP = diastolic BP – PAWP (pulmonary artery wedge pressure)
  - Normal is 60-80 mmHg

Coronary Perfusion

- Coronary perfusion occurs only during ventricular diastole
- Coronary perfusion depends on
  - Diastolic time
  - Diastolic pressure

Determinates of Ventricular Function

This is a very important slide!!



Cardiac Function

- Cardiac function is measured by cardiac output/cardiac index.
- To enhance cardiac performance, the treatment is to increase or improve cardiac output/cardiac index.

Definitions

- Cardiac output = heart rate (and rhythm) times stroke volume (SV)
- Stroke volume equals
  - Preload
  - Afterload
  - Contractility

Definitions

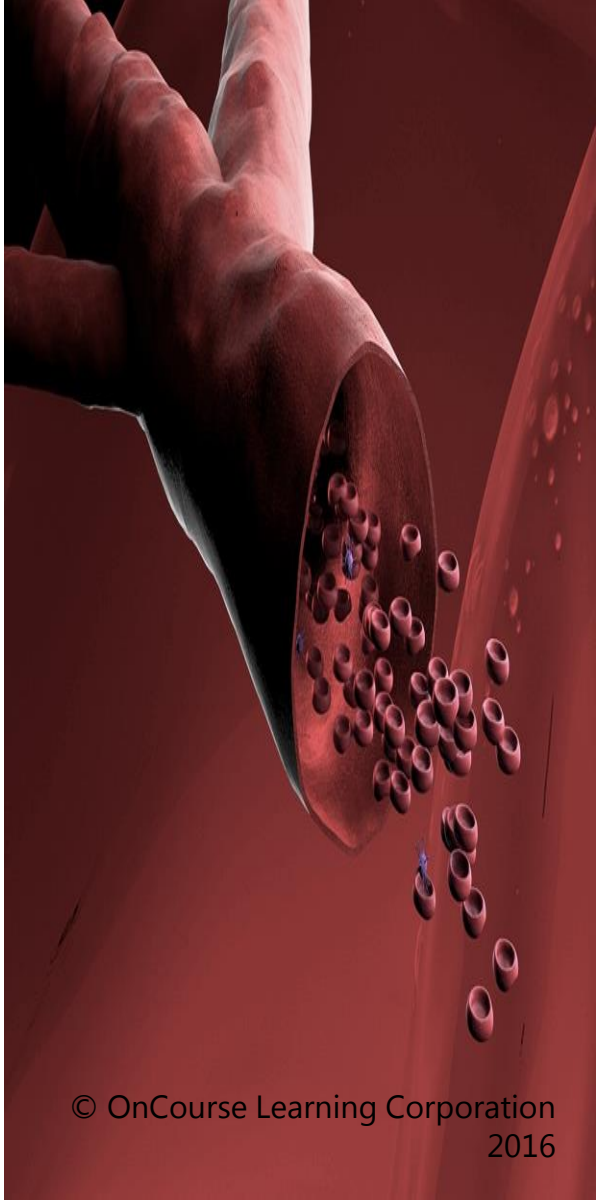
- SV = preload: amount of blood returning to the heart at the end of diastole
- SV = afterload: impedance to ventricular emptying: how much work the ventricle has to do to contract and eject blood
- Contractility: the amount of contraction the muscle of the ventricle can do

Drugs and Hemodynamics

- Heart rate and rhythm
  - All anti-dysrhythmics apply here
  - Know ACLS for dysrhythmias
  - Very few questions on pacemakers
  - Too fast or too slow: fix rate and rhythm

Drugs and Hemodynamics

- Preload
- Low: hypovolemia
  - Give fluid that patient needs
  - Fluid resuscitation
- High: Patient is volume overloaded
  - The patient in heart failure
  - Diuretics (if the kidneys work) or vasodilators (nitroglycerin)



Drugs and Hemodynamics

- Afterload, Low: low SVR (systemic vascular resistance)
  - Vasoconstrictors: vasopressin, levophed, neo-synephrine, dopamine

Drugs and Hemodynamics

- Afterload, high: high SVR
  - Vasodilating drugs: sodium nitroprusside, NTG
  - ABCs:
    - ACE-I (angiotensin converting enzyme inhibitors)
      Ex: enalapril, captopril, lisinopril, ramipril
      ARBs (angiotensin receptor blockers)
      Ex: candesartan, losartan, valsartan
      Alpha antagonists,
      Ex: doxazosin, prazosin
    - Beta blockers
      Ex: atenolol, labetalol, metoprolol
    - Calcium chanel blockers
      Ex: diltiazem, verapamil, nicardipine

Drugs and Hemodynamics

- Contractility
- Drugs that improve contractility: inotropes
  - Digoxin
  - Dobutamine: Dobutrex
  - Milrinone: Primacor
  - Dopamine
- IABP (intra-aortic balloon pump): not a drug, but remember this option

Supply and Demand

Remember the goals:

- Improve delivery of oxygen
- Decrease demand

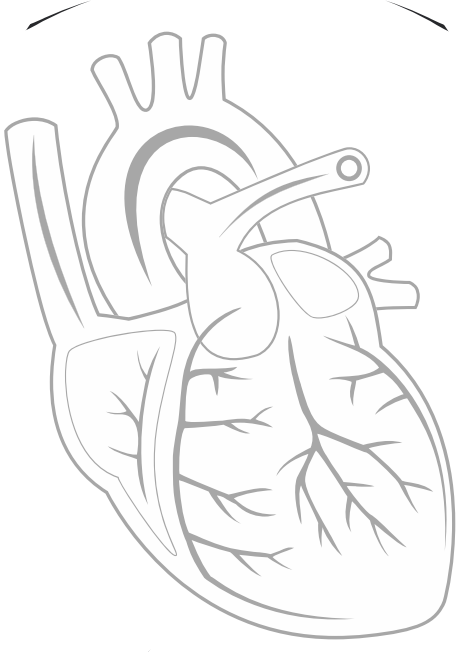
Supply and Demand

- The next slide shows the determinants of myocardial oxygen supply
  - Open coronary arteries, diastolic time and pressure, normal Hgb and SaO2
- Determinants of oxygen demand
  - Preload, afterload, HR and contractility

Oxygen Supply And Demand

Supply

- Coronary artery potency
- Diastolic pressure
- Diastolic time
- O_2 extraction
  - Hgb
  - SaO_2



Demand

- Heart rate
- Preload
- Afterload
- Contractility

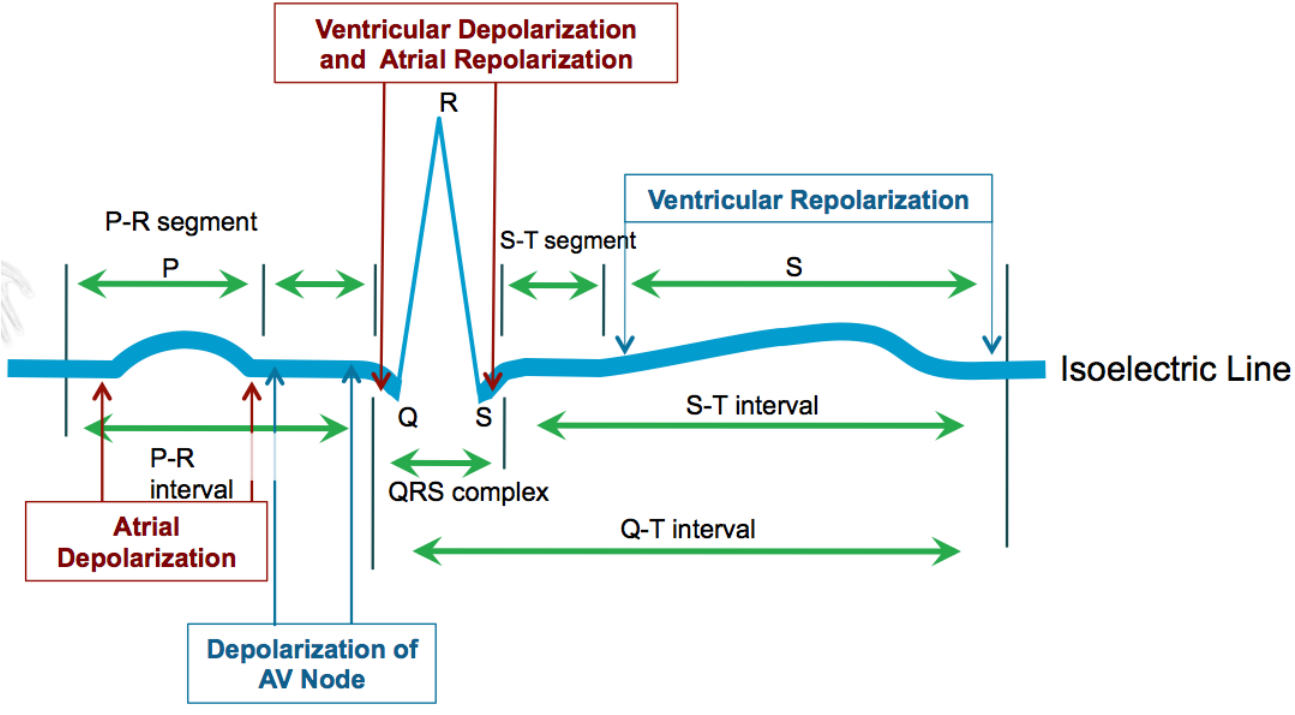
Heart Sounds: S3

- S3: always pathologic in the adult,
  - Reveals fluid overload in the patient
- An early sign of heart failure
  - Early diastolic heart sound
  - Low pitched

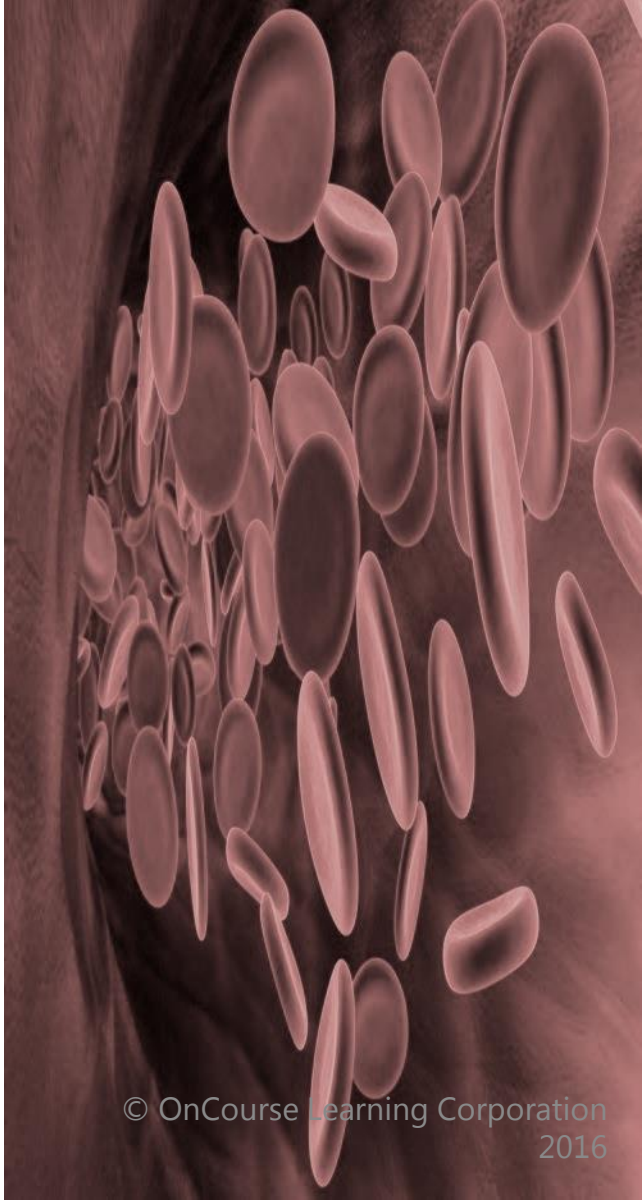
Heart Sounds: S4

- S4: always pathologic in the adult
  - Reveals a stiff (noncompliant) ventricle
- What can cause this?
  - Chronic hypertension, aortic stenosis, and
  - STEMI (noncompliance of the infarcted muscle)
  - Late diastolic sound
  - Low pitched

Cardiac Cycle



Hemodynamics



Hemodynamics: Pulmonary Artery Catheter

- RA pressure: 3-5 mmHg: CVP pressure
- RV pressure: 25/3-5 mmHg: not a monitored pressure
- PA pressure: 25/8-12 : pressure in the pulmonary parenchyma
  - Can be affected by any disease of the lung, pulmonary embolism, pneumonia or hypoxia

Pulmonary Artery Catheter

- PAWP (pulmonary artery wedge pressure or PAOP pulmonary artery occlusive pressure): 8-12mmHg
- Pressure reflects left atrial and left- sided filling pressures
  - The wedge can not be higher than the pulmonary artery diastolic

Hemodynamics

CO 4-8 liters/min

CI 2.5-4 liters/min/m2

CVP 2-6

PAP 20-25/8-12

PAWP 4-12

PVR 37-250dynes/sec/cm2

SVR 800-1200dynes/sec/cm2

Hemodynamics

- True mixed venous saturations: SVO2
- SvO2 is reflective of
  - Cardiac output/cardiac index
  - H&H
  - Oxygenation
  - Metabolic demand: important info of tissue utilization of oxygen: consumption
  - Important in determining shock states

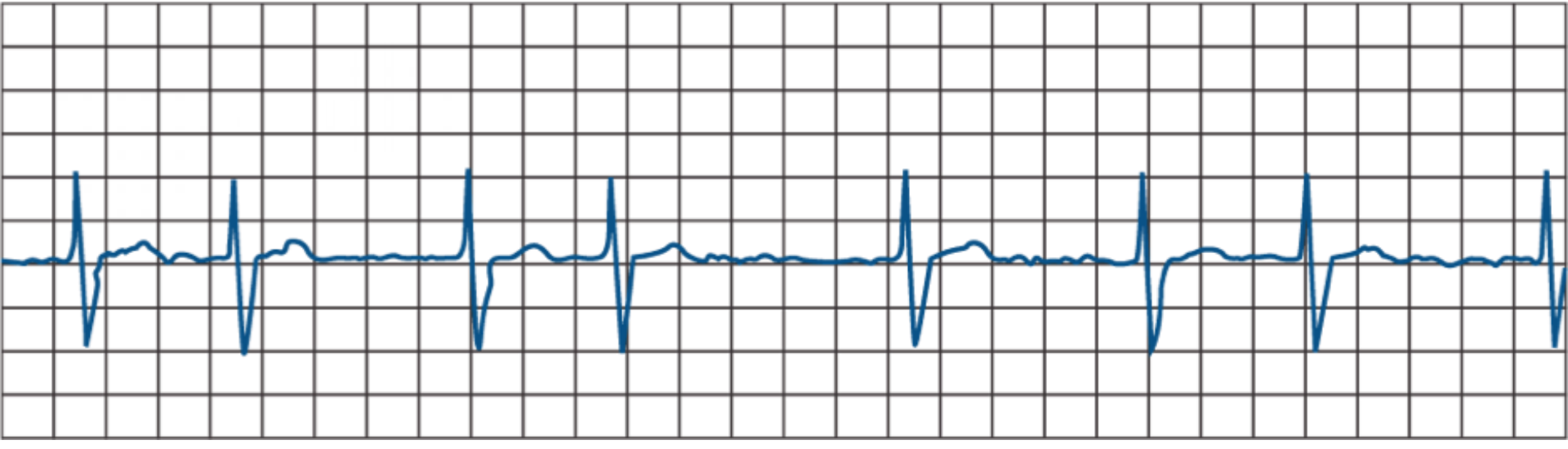
Cardiac Assessment

- First: Assess cardiac rate and rhythm
- Second: vital signs
- Then: physical assessment
  - Mental status
  - Skin: warm or wet
  - Urine output (the poor man's cardiac output machine)
    - 0.5 ml/kg/hour for urine output

Electrical Conduction

- It is all about ACLS,
  not what you do at **YOUR** hospital
- ***Know all rhythms!***

Interpreting Rhythm Strips



- You may have one strip to read; usually atrial fibrillation

Electrolytes

The following slides review electrolytes as they pertain to the heart

- At the end, there is an electrolyte review

Hypokalemia

Hypokalemia: ventricular irritability

- Flat T with prominent U wave
- T-wave + U-wave same amplitude
- ST seg flattening
- Prolongation of QT interval (K < 2.0)
- ST seg depression

Hypokalemia

Treatment:

- Give potassium
- Careful: How fast can you give potassium and where?

Hyperkalemia

- Hyperkalemia; asystole
  - Greater than 5.5 = tall, narrow, peaked T waves (tall peaked T waves in all 12 Leads)
  - QRS widens
  - P-wave widens
  - > 6.5 QRS widens
  - > 8.0 Wide QRS
  - P-wave barely visible

Hyperkalemia

- Hyperkalemia
  - Treatment: Remove K
  - Remove K: kayexalate or dialysis
  - Shift K: insulin and dextrose, NaHCO
  - Calcium: to protect the heart
- Remember: Repeat potassium levels every fours hours if treating

Calcium

- Hypocalcemia: torsades de pointes
  - Prolonged QT
  - Prolonged ST seg
- Hypercalcemia: agonal or asystole
  - Shortened QT
  - Shortened ST seg

Magnesium

- Hypomagnesemia: torsades de pointes
  - Prolonged QT
  - Broad, flattened T-wave
  - Dysrhythmias
- Hypermagnesemia: agonal to asystole
  - PR, QT prolonged
  - Prolonged QRS

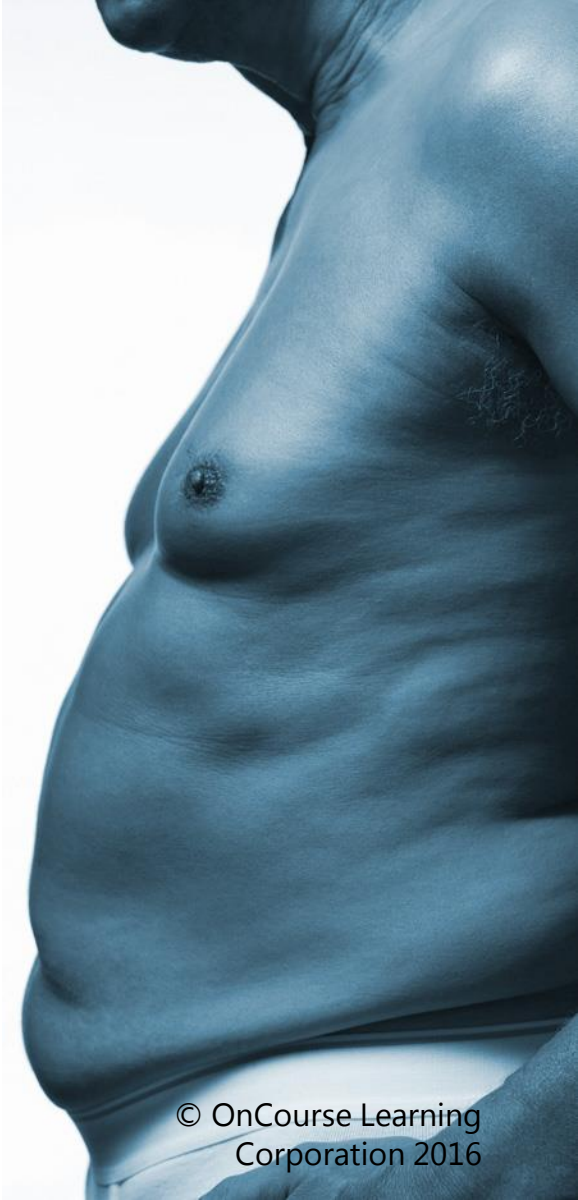
Coronary Artery Disease: CAD

- Definition: a blood flow limiting lesion
- Pathophysiology and etiology: inflammation
- Risk Factors: number one is diabetes
- Clinical manifestations
  - Heart failure, sudden death
  - Acute coronary syndrome: stable angina, unstable angina
  - NSTEMI (non-ST elevation MI)
  - STEMI (ST elevation MI)

Risk Factors

This man has all the risk factors:

- Hypertension
- Obesity
- Noninsulin dependent diabetes
- Atherosclerosis
- Obstructive sleep apnea
- Maybe hypothyroidism, a smoker and a drinker



Stable Angina

Clinical presentation: pain with exertion

- ECG presentation: normal
- Negative cardiac enzymes
- Treatment modalities
- Rest, NTG (nitroglycerine), ASA (aspirin)

Angina Management

- Antiplatelet therapy
  - ASA
  - ADP inhibitors (Plavix/Effient)
- Anticoagulant
  - Heparin/low molecular weight (fragmin, lovenox, arixtra)
  - Coumadin

Angina Management

- Vasodilator
- NTG: patch, SL, longer acting: Imdur
- Beta blocker
  - Decreases MVO2 (myocardial oxygen demand)
  - Regulates BP, HR, rhythm
- Ace I (angiotensin converting enzyme inhibitors)
  - BP control, reduces remodeling

Unstable Angina

- Clinical presentation: unpredictable pain, change in character of pain, rest pain
- ECG: ST changes, depression
- Enzymes: troponin elevated
- Pathophysiology: blood clot in coronary
- In the ACC/AHA guidelines: unstable angina (UA) and NSTEMI are together.

UA/NSTEMI

- Biochemical markers
  - Troponin +, CPK -
  - DX: UA minimal myocardial damage
  - DX: NSTEMI There is myocardial damage.
    - Prognosis: high-risk patient

UA/NSTEMI: Treatment

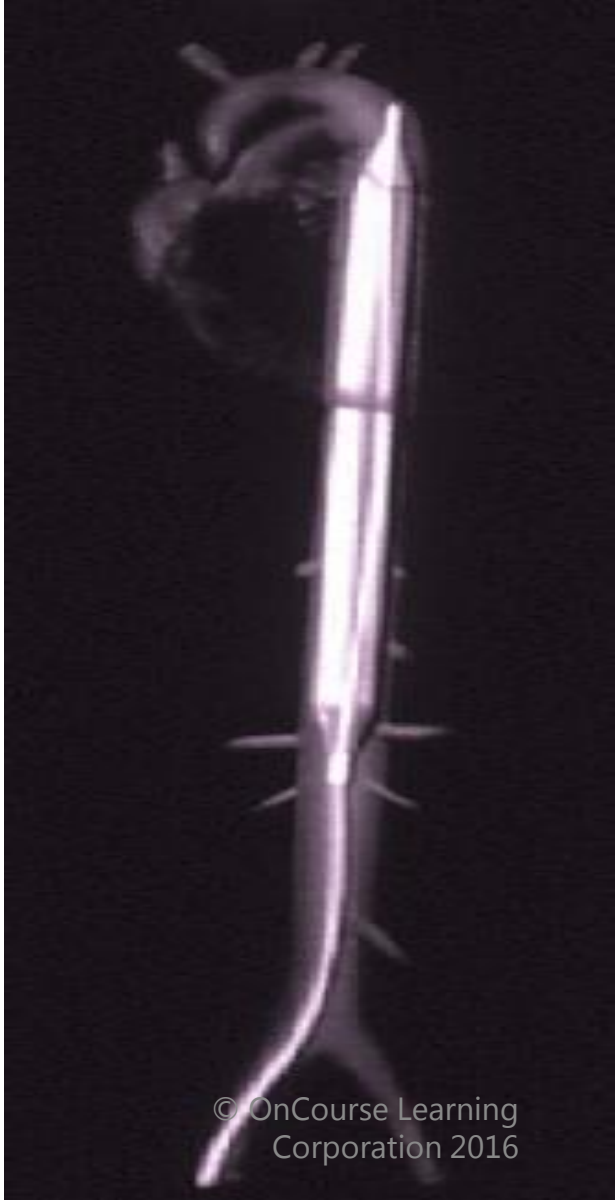
- Increase MVO_2 supply: Decrease MVO_2 demand: Put patient at rest
  - ASA and oxygen
  - Beta blockers
  - Heparin
  - NTG
  - Morphine
  - GP IIb-IIIa Inhibitor drugs

UA/NSTEMI

- Management: patient with refractory pain
- Assistance for the ventricle
- Medications: antiplatelet, Ilb IIIa inhibitors (ReoPro, Integrilin, Aggrastat), nitroglycerin, pain relief
- Mechanical assist
- IABP (Intra-aortic balloon -pump)
- Additional diagnostics: cardiac cath

IABP

- Two functions
  - Decrease afterload
  - Increase coronary perfusion
- Absolute contraindication: aortic insuff.
- Monitor for:
  - Vascular exam
  - Timing



UA/NSTEMI

- Medical interventional (cath lab)
  - PTCA (percutaneous transluminal coronary angioplasty)
  - Stent placement: drug eluting or bare metal stents
  - DCA (directional coronary atherectomy)
  - Cath lab intervention (PCI: percutaneous cardiac intervention)

Nursing Care of Cardiology Interventional Patient

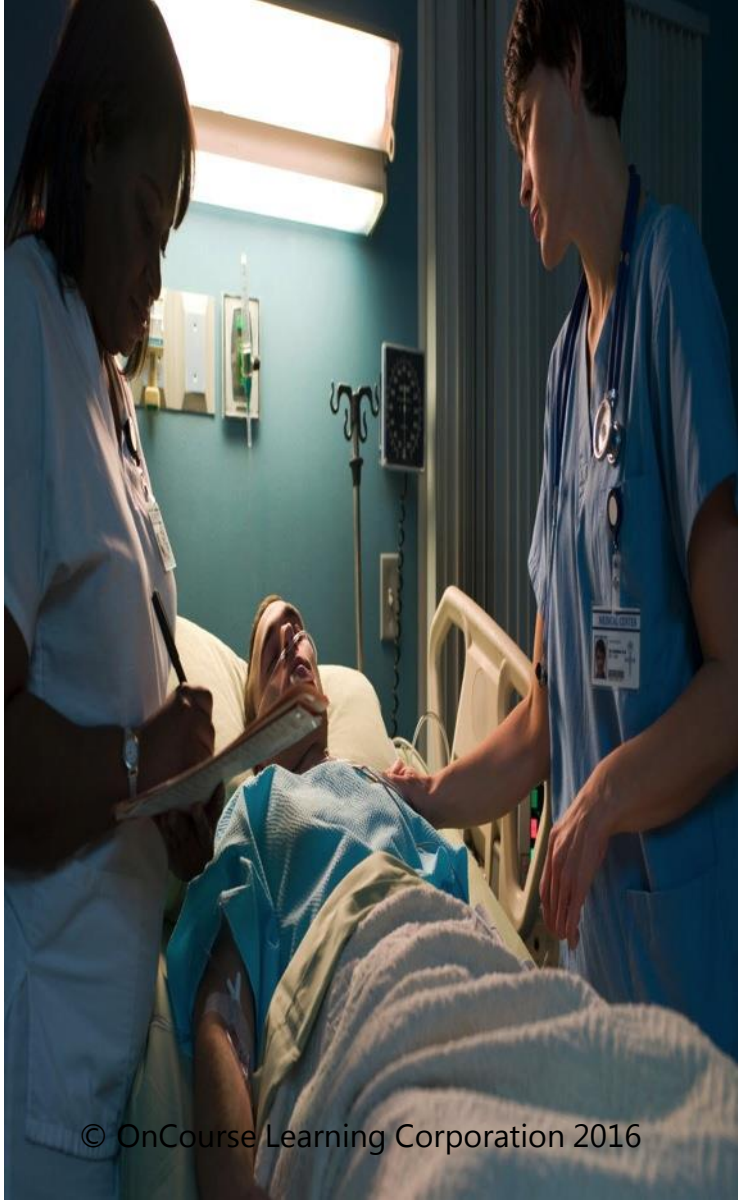
- Preprocedure
  - NPO, consent
  - Labs, ECG, insulin orders, prehydrate
  - Renal protection if needed
  - Vascular exam, allergies
  - On-call meds: ASA, Plavix, etc.

Postprocedure

- Monitor ECG
- Vascular assessment
- Labs, heparin protocol, IIbIIIa infusion
- Activity restrictions, progression
- Sheath removal
- Medications
- Monitor for bleeding

Arterial Insufficiency or Occlusion

Nursing Care



Vascular Assessment

- Six Ps of assessment
  - Pulse: presence of palpable or doppler able
  - Pain: good indicator of ischemia
  - Pallor: pale extremity
  - Polar: cold extremity
  - Paresthesia: tingling, pins and needles in extremity
  - Paralysis: unable to feel or move extremity

Peripheral Vascular Insufficiency

Arterial vs. Venous

Carotid Artery Stenosis: Neuro checks

Femoral-Popliteal Bypass: Blood Pressure monitoring

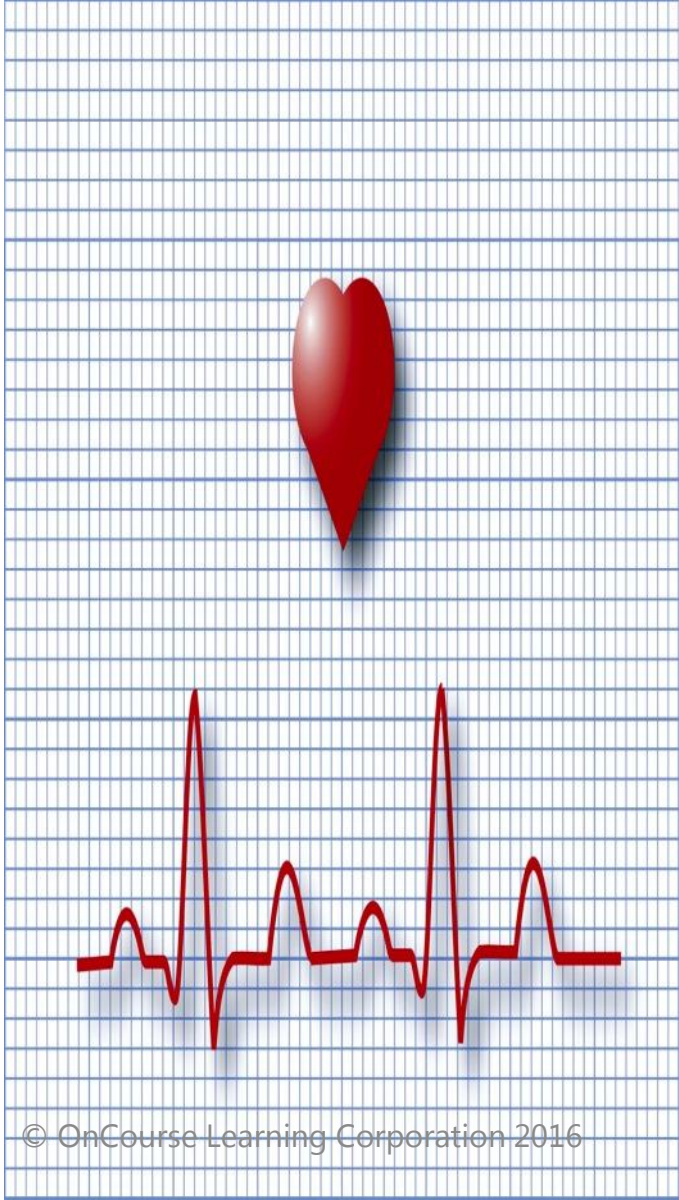
Peripheral Stents

Improving Blood Flow: Perfusion assessments

AMI/STEMI

Time = Muscle

Muscle = Life



AMI/STEMI

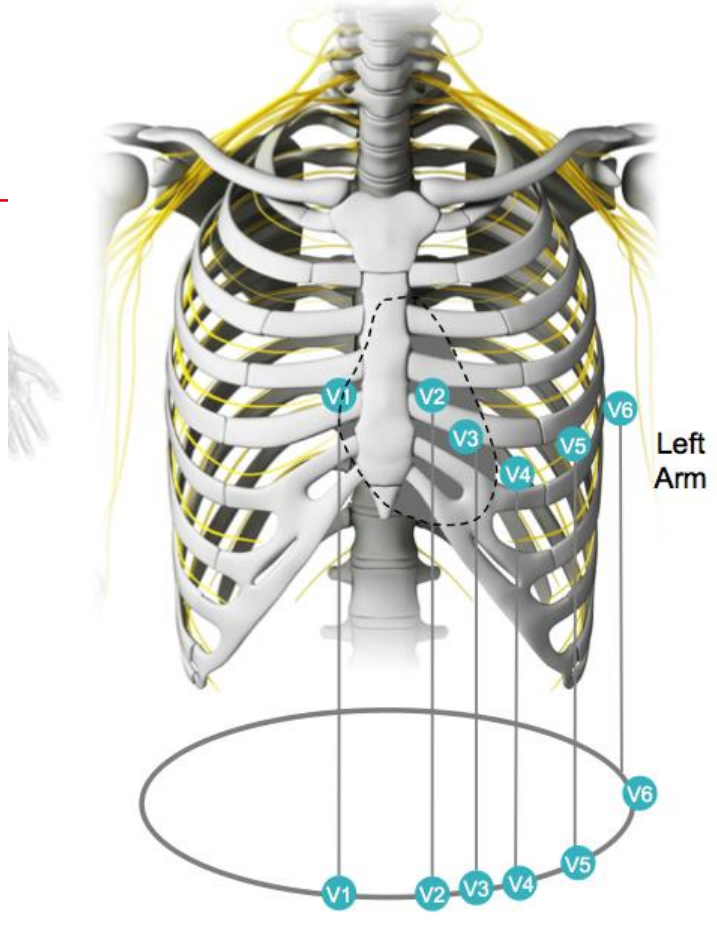
- Etiology: atheroma rupture with clot formation
- Pathophysiology: inflammation
- Clinical presentation: men vs. women
- Labs: troponin, LDH, CPK, MB band
- ECG, ECHO
- Cath lab

ECG Changes During STEMI

- First: The first ECG change in early infarction is T wave elevation and peaking, only in the leads associated with the injury.
- Second: T wave inversion
- Third: ST segment elevation
- Fourth: Q wave formation and ST elevation

12 Lead ECG in STEMI

- Leads: II, III and AVF are inferior leads
- Leads: I, AVL are high anterior leads
- Leads: V1 and V2 are septal leads
- Leads: V3-V6 are anterior lateral leads



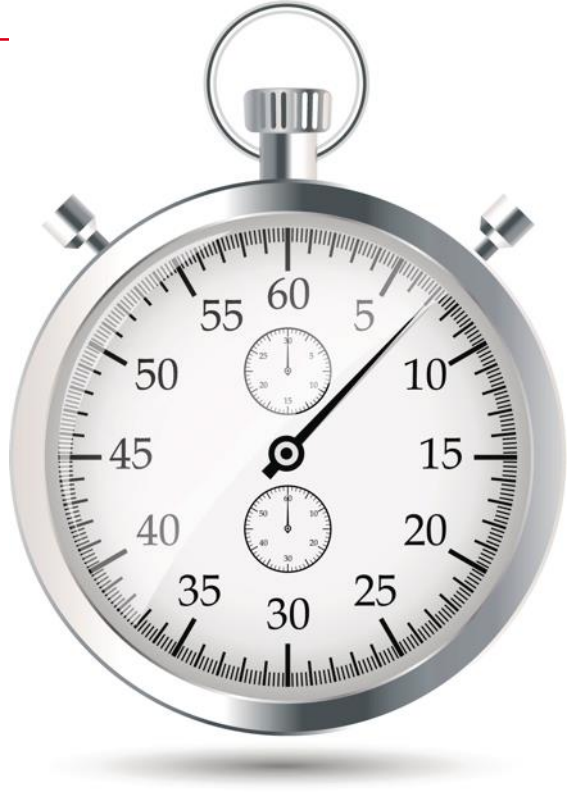
STEMI: Right Ventricular Infarction

Assess for clinical indications of right ventricular myocardial infarction

- ECG changes
  - V_{4R}, V_{5R}, V_{6R}
  - Decreased RAP, decreased PAWP
  - Decreased CO, CI, MAP; Increased SVR
  - Clinical indications of right ventricular failure
  - Minimal to absent pulmonary congestion

STEMI: Acute Management

- Manage and monitor
  - 12 lead ECG, enzymes
  - ECG, VS, BLS, ACLS
  - Hemodynamic parameters
- Reduce size of infarct
  - It's all about timing
  - Door to diagnosis and treatment (90 minutes)
  - Time is muscle



STEMI: Management

- Diagnose
- Clinical presentation: different between men and women, ECG, enzymes
- Remember that in women, an early symptom of STEMI may be nausea and vomiting or epigastric distress
- Take home message: get a 12-lead ECG

STEMI: Management

- Treatment paradigm: open artery:
  - ABCs, oxygen, pain management, ASA , NTG
  - Reperfusion therapies
    - Cath lab (PCI)
    - Fibrinolytic therapy (if delay in treatment and patient is a candidate)
    - CABG

STEMI: Dysrhythmias

- Inferior wall STEMI: most common dysrhythmia: bradycardia and heart block
- Anterior wall STEMI: most common dysrhythmias are tachycardias, including VT and VF

STEMI: Management

Increase Oxygen Supply

Oxygen therapy if O2 Saturations less than 94%

Nitroglycerin: increase supply

Open the artery: Catheterization Lab

STEMI: Management

- Decrease myocardial oxygen consumpti
  - Oxygen (Sats less than 94%)
  - Beta blockers
  - ACE inhibitors
  - Pain control
  - Rhythm control

STEMI: RV Infarction

- Assess for clinical indications of RVMI
- Right sided ECG: 15-18 Lead ECG
  - ECG changes V4R, V5R, V6R
  - High CVP, low or normal PAWP
  - Low CO/CI, MAP low
  - Clinical indications of RV failure
  - Minimal to absent pulmonary congestion

RV Infarction: Management

- Maintain adequate filling pressures
- Administer volume
- Avoid diuretics and/or venodilators (NTG)
- Maintain contractility
- Remember: fluid is the answer here

Hemodynamics
RV vs. LV infarction

RV	
CVP	High
PAP	Normal or Low
PAWP	Normal or Low
CI/CO	Low
SVR	High

LV	
CVP	Normal or High
PAP	High
PAWP	High
CI/CO	Low
SVR	High

Complications: STEMI

- Dysrhythmias
- Heart failure
- Cardiogenic shock
- Papillary muscle dysfunction or rupture
- VSD (ventricular septal defect)
- Cardiac rupture
- Ventricular aneurysm
- Pericarditis
- Dressler's syndrome
- Sudden death

Pericarditis

- Inflammation of the pericardial sac
- Due to infection, connective tissue disorder, scar formation following MI, and/or renal failure due to azotemia
- Dressler's syndrome: inflammation of pericardium following infection, connective tissue disorder or surgery on the heart

Pericarditis

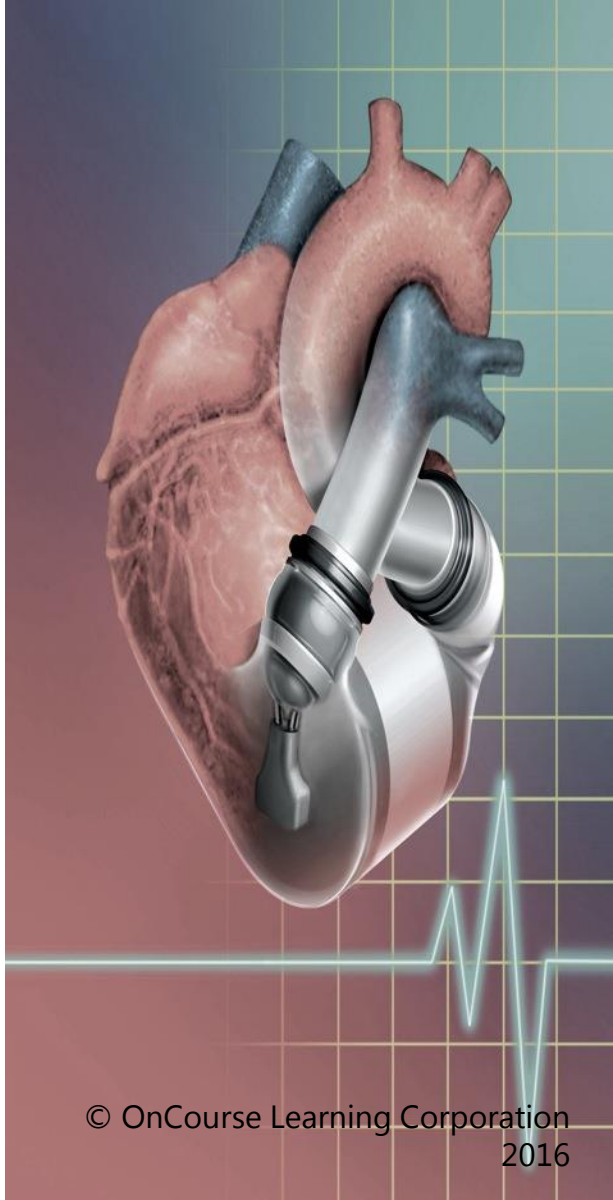
- Patient with chest pain
  - Increases with deep inspiration
  - Improves with upright position
- 12-Lead ECG with ST-T wave elevation in all 12 leads
  - Pericarditis

CAD

- Treatment: Re-vascularization
- Medical: DX, open the artery, antiplatelet therapy, vasodilator, beta blocker (BB), ACE inhibitor (ACE-I), statin
- Surgical: CABG: antiplatelet, vasodilator, BB, ACE-I, and statin (same meds)
  - Statins: atorvastatin (Lipitor), fluvastatin (Lescol), lovastatin (Mevacor), pravastatin (Pravachol), rosuvastatin (Crestor), simvastatin (Zocor, Lipex)

Heart Failure (HF)

- An evolving definition
- Heart failure should be viewed as a neurohormonal model, in which heart failure progresses as a result of the overexpression neurohormones that are capable of exerting toxic effects on the heart and circulation … contributing to disease progression independently of the hemodynamic status of the patient
- A progressive syndrome resulting in malperfusion: an inability of the heart to meet the demands of the body

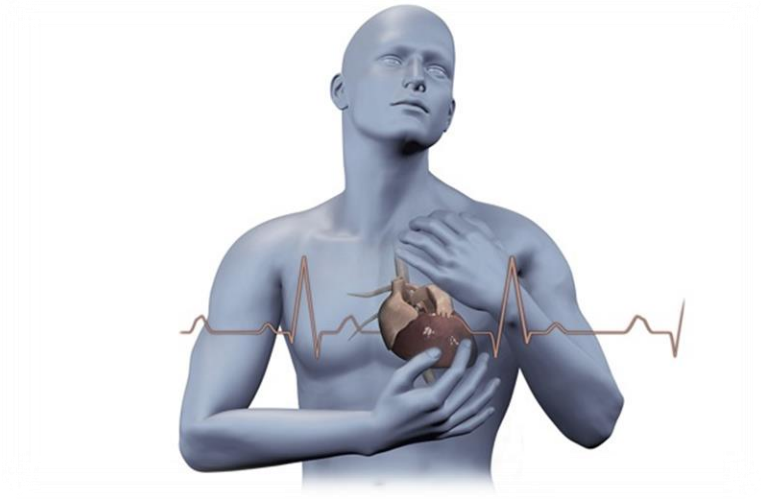


Etiology of Heart Failure

- LVF
  - CAD/LV infarct
  - Dysrhythmias
  - Volume overload
  - Valvular disease
  - VSD
  - Cardiomyopathy
  - Coarctation of aorta
  - Tamponade
- RVF
  - CAD/RV infarct
  - Dysrhythmias
  - Volume overload
  - Valvular disease
  - VSD
  - Cardiomyopathy
  - Myocardial contusion
  - Pulmonary HTN

Etiology of Heart Failure

The most common cause of heart failure in the US today is **ischemic cardiomyopathy**

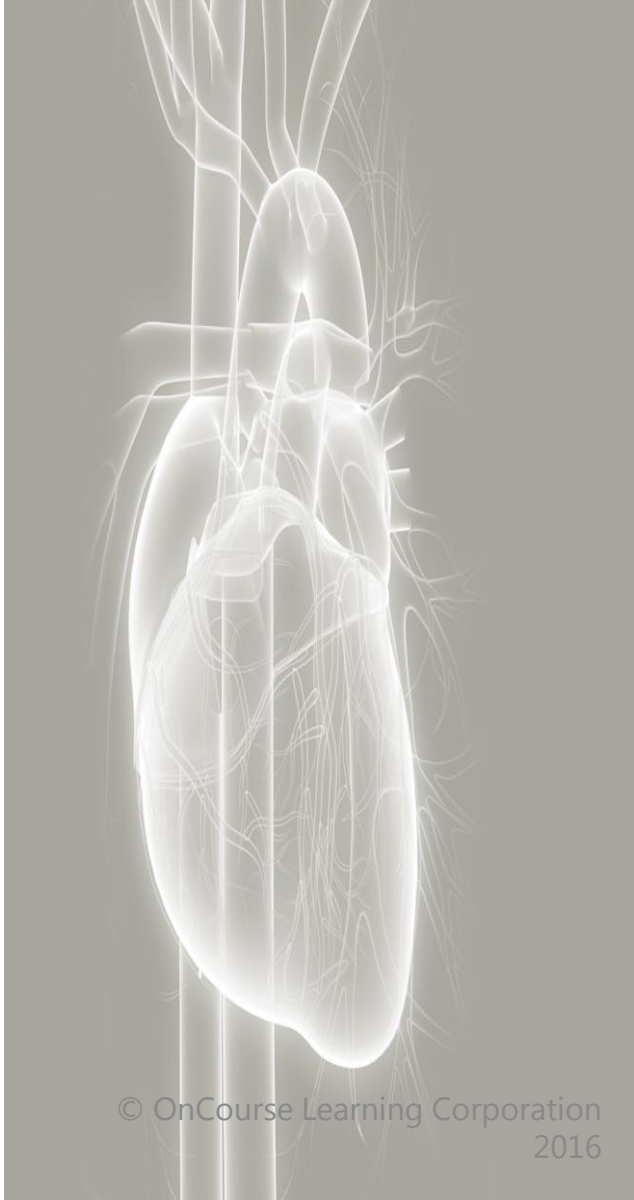


Cardiomyopathy

- Cardiomyopathy: dilation of the ventricles causing loss of contractile function, resulting in reduced ejection fraction and signs and symptoms of heart failure
- Most common in the U.S. is ischemic cardiomyopathy resulting form coronary artery disease

Cardiomyopathy

- Types of cardiomyopathies
  - Ischemic
  - Viral
  - Medication induced: ETOH, drugs, Adriamycin
  - Postpartum
  - Idiopathic



Clinical Presentation: LVF

- Tachycardia
- Tachypnea, dyspnea, orthopnea
- Paroxysmal nocturnal dyspnea (PND)
- Left sided S3
- Dry cough at night
- Pulsus alternans
- Weakness, fatigue
- Mental confusion
- Murmur mitral regurgitation
- ECG: atrial arrhythmia, LAE, LVH
- Oliguria

Clinical Presentation: RVF

- JVD
- HJR (hepatojugular reflux)
- Dependent edema
- Hepatomegaly
- Anorexia, nausea, vomiting, abd pain
- Ascites
- Nocturia
- Weakness, fatigue
- Wt gain
- Murmur tricuspid regurg
- Right-sided S3
- Abn liver functions
- ECG: RAE, RVH, atrial dysrhythmia

HF: Management

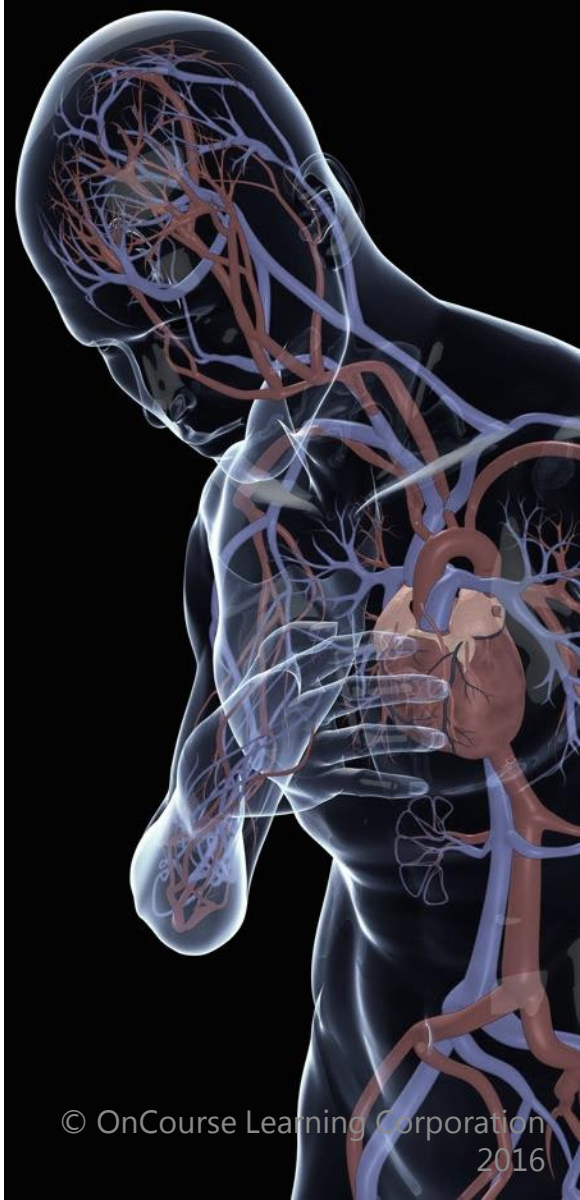
- Decrease preload:
  - Monitor volume status
  - Diuretics, natrecor
  - Nitroglycerin, ACE-I, pulmonary vasodilators (oxygen), IABP
- Decrease MVO2: decrease afterload
- Beta blockers, carvedilol (Coreg)
- Control dysrhythmias (at fib)

HF: Management

- Increase contractility: IABP
  - SNS stimulants: dobutamine
  - PDE inhibitors: milrinone
  - Dopamine (at inotropic dosage)
  - Digoxin (not used acutely)

Heart Failure

- Chronic HF Treatment
- Preload
  - Low-salt diet
  - Diuretics: Lasix, potassium
- Afterload
  - Beta Blockers and ACE-I
- Contractility
  - Digoxin



Chronic Heart Failure

- If patient continues to gain weight secondary to fluid retention (the patient already on Lasix)
- Add: Aldactone (aldosterone inhibitor)
- Careful of K+ levels

Acute Decompensated Heart Failure (Pulmonary Edema)

- Acutely decrease preload
  - Stop all intravenous fluids
  - Lasix
  - If not responding to diuretics, add vasodilators like nitroglycerin
  - Ultrafiltration if not responding to diuretic or vasodilation therapy

Acute Decompensated Heart Failure (Pulmonary Edema)

- Acutely decrease afterload
  - Vasodilator
  - Beta Blocker: as long as patient not in cardiogenic shock
  - IABP

Acute Decompensated Heart Failure (Pulmonary Edema)

- Increase contractility
  - Add inotrope if in cardiogenic shock: dobutamine, milrinone, dopamine
  - IABP

Acute Decompensated Heart Failure: Pulmonary Edema

- Questions on the test:
  - What drugs would you anticipate the patient being on in pulmonary edema?
  - Each answer has three drugs: What do you pick?
    - Preload reducer, afterload reducer and contractility drug

Cardiogenic Shock

- The heart now fails to meet the metabolic demands of the tissues; at such a rate, that th body can no longer compensate
- What do you see?
  - SHOCK

Shock: Cold and Wet

- Vital sign changes:
  - Tachycardia, dysrhythmias (at fib), lowered systolic BP with elevated diastolic BP = narrowed pulse pressure, increased RR
- Patient with altered mental status
- Cold and wet skin
- Decreased or NO urine output

Aneurysm/Dissection

Control Blood Pressure

- Repair: stent or surgical replacement
- Post-operative Care: Pain control, Blood pressure control (surgeon determines), renal and pulmonary care
- Post-operative Complications: Spinal Cord Ischemia/Infarction, Pulmonary Insufficiency, Renal Injury
- Ambulate, Incentive Spirometer, Ambulate

Cardiac Pearls

- ABCs: always remember the ABCs
- Maintain CI/CO: preservation of PERFUSION
- Maintaining HR × SV
  - Preload
  - Afterload
  - Contractility

Cardiac Pearls

- Remember to look at pulse pressure
- In questions about shock, look closely at the clinical presentation
- Look at pulse pressure!

Cardiac Pearls

- ST segment depression = ischemia
- ST segment elevation = current of injury
- IABP = increase coronary perfusion, decrease afterload:
  - Increases myocardial oxygen supply
  - Decreases demand

Cardiac Pearls

- ST elevation II, III, AVF = inferior infarction
- ST elevation I, AVL, V leads = anterior infarction
- Remember: The pulmonary artery catheter: the wedge can not be higher than the pulmonary artery diastolic.

Cardiac Pearls

- Acute arterial occlusion is a limb-threatening complication: the 6 Ps of arterial circulation
  - **P**ulse
  - **P**ain
  - **P**allor
  - **P**olar
  - **P**aresthesia
  - **P**aralysis

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