NURSING ASSESSMENT OF THE CARDIOVASCULAR SYSTEM

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TODAYS DISCUSSION

• REVIEW OF
  – Briefly review cardiac anatomy and physiology
  – Discuss concepts of preload and afterload
  – Acute MI
  – CHF
  – Atrial Fibrillation
  – ACLS guidelines

HEART

• Four chambers
  1. Right and Left Atria
  2. Right and Left Ventricles

UN-OXYGENATED BLOOD

• Superior and Inferior Vena Cava drain unoxygenated blood from the body to the RA
• RA thru Tricuspid Valve to RV
• RV thru pulmonic valve to Pul artery to lungs
• Blood circulates thru lungs picks up oxygen
• Exits lungs thru pulmonary veins
• Enters the LA from the lungs
• From the LA thru the Mitral valve to LV
• From LV thru Aortic Valve to aorta to oxygenate the body
OXYGENATED BLOOD
• Leaves lungs via pulmonary veins
• Enters the LA
• Flows thru Mitral valve to LV
• From LV thru Aortic Valve to Aorta and out to oxygenate the body

BLOOD SUPPLY TO HEART
• From Coronary Arteries
• During Diastole when the ventricles are relaxed
• Oxygenated blood enters the coronary arteries through two small openings just above the closed aortic valve.

HEART ACTS AS A PUMP
• Right and Left Ventricles serve as pumping chambers of the heart.
• Right Ventricle – pumps into lungs – lower pressure – walls thinner
• Left Ventricle – pumps against pressure of body’s arterial system – walls

BLOOD FLOW
• Blood flows from higher to lower pressure.
• Valves open and close in response to pressure.
• Diastole – Heart at rest. Blood flows into the Atrium and then into the ventricle. As pressure increases the atria contract, the blood flows into the ventricles.
• Systole – pressure inside ventricle rises and the Ventricles contract.

CARDIAC CONTRACtIONS
• Atrial Kick – Contributes about 30% of the Cardiac Output. Decreased with certain arrhythmias (At. Fib, Tachy)
• Cardiac Output – amount of blood pumped by each ventricle in 1 minute, calculated by multiplying the stroke volume by the heart rate.

Cardiac Output Determined By
• Preload – volume of blood in ventricles at the end of diastole. Amount of blood available to be pumped out with next beat.
• Afterload – amount of pressure the ventricle exerts to eject blood into the circulation. Higher the systemic vascular resistance (SVR), the harder the Left Ventricle has to work. Higher the pulmonary vascular resistance (PVR) the harder the Right Ventricle has to work.
• Stroke volume–amount of blood ejected with one heartbeat. Cardiac Output (CO) equals the stroke volume (SV) times the heart rate (HR). CO=SVxHR
Decrease Preload

- Hypovolemia
- Drugs that decrease volume such as diuretics (Lasix)
- Drugs that interfere with sodium and water retention – ACE inhibitors (ACE Inhibitors end in “pril”, such as captopril).

Increase Preload

- Hypervolemia
- Blood or blood products.
- Albumin, dextran
- Normal Saline, Lactated Ringers solution

Increase Afterload

- Hypertension. Long term untreated hypertension leads to left ventricular failure due to the continuous increase in afterload.
- Aortic or pulmonic stenosis. Heart has to pump harder to push blood through a stenotic valve.
- Drugs – Dopamine and Epinephrine cause vasoconstriction.

Decrease Afterload

- Drugs – which decrease systemic vascular resistance and cause vasodilation.
  - Calcium channel blockers (Such as diltiazem, (cardizem), norvasc, calan)
  - Beta Blockers (drugs that end in “lol” such as labetalol, propanolol, sotalol).
  - ACE Inhibitors (drugs that end in “pril” such as ramipril, enalapril, lisinopril
  - Angiotensin II receptor blockers (ARB’s) (drugs that end in “sartan” such as valsartan.

STROKE VOLUME

- Volume of blood ejected by ventricle each beat
- Determined by:
  1. Intrinsic contractility of cardiac muscle
  2. Degree of stretch of the cardiac muscle before its contraction (preload)
  3. The pressure against which the heart muscle must pump during contraction (afterload).
- Stroke Volume = Preload + Afterload + Contractility

INCREASING STROKE VOLUME

- Exercise – increases preload thru increased venous return, increases contractility through sympathetic nervous system discharge and decreases afterload thorough peripheral vasodilation with decreased aortic pressure.
- Medications
Contractility – STARLING’S LAW
- Contractility is ability of muscle cells to contract after depolarization. This depends on how much the muscle fibers are stretched at the end of diastole.
- Over stretching or under stretching alters contractility and amount of blood pumped out of the ventricles.
- Larger the preload, the greater the stroke volume, but only up to a point.
- Like stretching a rubber band

EJECTION FRACTION
- Percentage of the end-diastolic volume that is ejected with each heart beat.
- EF of 55% to 75% is normal
- Used to measure myocardial contractility.
- Decreased if contractility is decreased.

Nerve Supply
- Heart supplied by the two branches of the autonomic nervous system.
  1. Sympathetic (adrenergic) – heart’s accelerator.
  2. Parasympathetic (cholinergic) – heart’s breaks.

SYMPATHETIC
- Norepinephrine (nonadrenaline) and epinephrine (adrenaline) influenced by this system.
- These chemicals – increase heart rate, automaticity, atrioventricular conduction, and contractility.

PARASYMPATHETIC
- Vagus nerve – carries impulses that slow heart rate and the conduction of impulses through the SA node, AV node, AV junction and myocardium of the atria.
- Baroreceptors – specialized nerve cells in the aorta and the internal carotid arteries.
- Stimulation of baroreceptors – stimulate vagus nerve to apply cardiac brakes.
- Hypertension – stretches baroreceptors and stimulates vagus nerve
- Carotid sinus massage – activates the baroreceptors to stimulate vagus nerve to slow heart rate

Atropine and the parasympathetic nervous system
- Atropine interferes with the effects of the parasympathetic nervous system. Atropine blocks the vagal response and increases the heart rate.
- Atropine will only have an effect on low heart rates that are the result of parasympathetic stimulation, such as a vagal response – not if is due to heart block
Blood Pressure

- Measure of pressure exerted by blood against the walls of the arterial system
- Systolic – peak pressure when heart contracts
- Diastolic – cardiac relaxation. Heart fills, Coronary Arteries receive blood supply.

Cuff - Size

- BP cuff too large = falsely low reading
- BP cuff too small = falsely high reading

HYPERTENSION

- Headache and Nosebleeds not symptoms of Hypertension
- Common Symptoms –
  - Dizziness
  - Palpitations
  - Angina
  - Dyspnea

CAUTIONS IN TREATMENT FOR HYPERTENSION

- Many different meds may be used to treat HTN (same meds may be used for other reasons – patient education important)
- Classifications of meds used for HTN
  - Beta Blockers
  - ACE inhibitors
  - Calcium Channel Blockers
  - ARB’s
  - Combinations – often with diuretics

- Patient Teaching points
  - Never stop medications without physician order – abrupt withdrawal may cause severe hypertensive reaction
  - Know why taking medication
  - Prone to orthostatic hypotension – stand and change position carefully
  - Beta Blockers often used for HTN may cause impotence
Orthostatic Hypotension

- Significant drop in BP with upright posture
- Symptomatic – dizziness, lightheadedness or syncope.
- Most common causes:
  - 1. reduced fluid or blood in the circulatory system
  - 2. inadequate vasoconstrictor mechanisms
  - 3. insufficient autonomic effect on vascular constriction.

Measuring Orthostatic BP

- Position patient flat for 10 min and check BP
- Record both heart rate and BP
- Have patient sit on edge of bed with feet dangling wait 1 to 3 minutes check BP and HR
- Stand patient wait 1 to 3 minutes check BP and HR

Normal Postural BP Changes

- When a person stands up or goes from lying to sitting position
  - 1. Increase in HR of 15 to 20 beats
  - 2. Drop in SBP of up to 15mm Hg
  - 3. Slight drop to an increase of 5 to 10mm Hg in diastolic pressure

Examination of Pulse

- Pulse Rate – Normal 60 to 100. Dependent on conditioning, age and activity
- Pulse Rhythm – Is it regular or irregular. Normal, especially in young people, to increase during inspiration and slow during expiration.
- Irregular radial pulse – auscultate the apical pulse for a full minute at the apex of the heart
- Pulse deficit – difference between the apical rate and the peripheral rate. Common with various dysrhythmias

NURSING ASSESSMENT

- Patient history
- Present - fatigue, fluid retention, hypertension, palpitations, SOB, chest pain, calf tenderness, dizziness, alert, orientation, medications.
- Past – Surgery, anemia, bleeding, pneumonia, rheumatic fever, rubella, scarlet fever, strep throat, syncope, CAD,MI, CHF, CVA, arrhythmias.
- Family History– CAD,PVD,HTN,CVA, etc.
- Social History– ETOH, recreational drugs, smoking, work, activity/exercise, stress, diet, sleep patterns

AUSCULTATION OF HEART SOUNDS

- S1 – closure of the tricuspid and mitral valves. Left fifth ICS along midclavicular line. Soft Lub sound.
- S2 – Closure of aortic and pulmonic valves. Right second ICS with patient sitting and leaning forward slightly. Sharp dub sound.
- Gallops – may be heard when extra heart sounds (S3 and S4) are present. May hear Gallop in CHF
- Murmurs – described as swishing sounds caused by incompetence of valves and or abnormal blood flow patterns.
HEART SOUNDS
• Different levels of expertise in identifying heart sounds
• Med-Surg nurse should be able to recognize that what they are hearing is not normal.
• Report abnormal findings to physician.

FRICION RUB
• Pericarditis – abrasion of the pericardial surfaces during the cardiac cycle.
• A harsh grating sound may be heard in both systole and diastole.
• Heard best using the diaphragm of the stethoscope with patient sitting up and leaning forward.

COMMON LABS FOR THE CARDIAC PATIENT
• Cardiac Enzymes (Isoenzymes)
  1. Breaks in myocardial cell membranes lead to release of certain isoenzymes into blood
  2. Should be evaluated in all patients with chest pain indicative of acute coronary syndrome
  3. Should be repeated if initial results are normal and pain duration of less than 6 hrs.
  4. Should be repeated a minimum of 12 hrs following symptom onset

TROTONIN I or T
• Specific to damaged cardiac cells
• Initial elevation – within 3 to 12 hours
• Peak increase – 2 to 4 days
• Remains elevated - 6 to 12 days
• Not affected by recent cocaine use

CPK-MB ISOENZYMES
• CPK and CPK-MB levels are elevated when myocardial cell injury occurs
• CPK may be elevated with other muscle injury
• CPK-MB more specific to cardiac injury
• CPK-MB may be elevated pericarditis or intracardiac injections
• May have false positive in patients with cocaine – associated chest pain

CPK-MB
• Initial Increase – 3 to 12 hrs
• Peak Increase – 24 hrs
• Remains elevated – 48 to 72 hrs
## POTASSIUM
1. Too high or too low – cardiac irritability
2. Affected by renal function and medications (may be increased by some ACE inhibitors ex: Vasotec)
3. Decreased by some diuretics - Lasix

## MAGNESIUM
- Low – increases arrhythmia risk
- May be decreased in
  - Chronic TPN, Alcoholism, Nutritional deficiency

## CALCIUM
- Essential to coordination of electrical and mechanical cardiac events
- Important for blood coagulability
- Too High or too Low can cause arrhythmias

## POSSIBLE CAUSES OF CHEST PAIN
- Acute Bronchitis, Pneumonia, Pneumothorax, TB
- Anxiety
- Aortic Aneurysm
- Cholecystitis
- Esophagitis
- Musculoskeletal strain, Rib fractures
- Pancreatitis
- Peptic ulcer
- Pulmonary embolism
- Withdrawal from beta blockers
- Angina or MI
- Pericarditis

## IS THE CHEST PAIN CARDIAC?
- Angina
  1. Isn’t affected by body position/ changes
  2. Isn’t aggravated by respirations
  3. Tends to be diffuse, not sharply localized
  4. Tends to feel like it comes from deep within
  5. Usually described as a dull ache, seldom sharp or stabbing

## CORONARY ARTERY DISEASE (CAD)
- Accumulation of plaque, cholesterol, and connective tissues on the intimal wall of the coronary vessels. Also known as atherosclerosis.
- Etiology not completely understood
- May be stable plaque or plaque ruptures causing occlusion
- May have inflammatory component
### CAD Risk Factors
- Family history
- Age
- Gender
- Hypertension
- Diabetes
- Obesity
- Diet
- Smoking
- Inactivity
- Stress

### Treatment of CAD
- Surgical
- PCI
- Medical

### Angina
- Usually first sign of CAD
- Inadequate blood flow to meet the demands of the heart muscle
- Decreased oxygen carrying capacity of blood as seen in severe anemia
- May be stable, unstable or Prinzmetal’s

### Stable Angina
- Lasts a few seconds to a few minutes
- Tends to be predictable
- Triggered by activity, cold weather, stress
- Typically relieved by rest or NTG
- Diagnosed by exercise stress tests

### Unstable Angina
- May be more severe than stable angina
- Commonly precedes an MI
- Duration of up to 30 minutes
- Unpredictable course
- Diagnosis from history and may have transient ST segment changes on EKG
- May have only partial relief with NTG, may require narcotics

### PRINZMENTAL’S ANGINA
- Also called variant angina
- Occurs at rest
- Results from coronary artery spasm
- Possible without coronary atherosclerosis
- Affects some heavy smokers
- Frequent occurrences between midnight and 8 a.m.
- Prompt relief from NTG
- Marked ST – Segment elevation during chest pain that quickly disappears with NTG or other pain control measures
LOCATION OF ANGINA PAIN
- Upper chest
- Neck and jaw
- Beneath sternum with or without radiating to jaw, neck, down left arm
- Epigastric area with or without radiation
- Left shoulder with or without radiation to inner aspect of both arms
- Intrascapular region – especially in females

CHARACTERISTICS OF ANGINA PAIN
- Pain may be described as heaviness to a crushing discomfort
- Pain may be aching, burning, squeezing
- Pain may be accompanied by SOB, diaphoresis, nausea or vomiting

MYOCARDIAL INFARCTION (MI)
- Narrowing of coronary arteries progresses to point of interfering with blood flow and deprives heart of oxygen and nutrition causing tissue damage
- Risk of actual MI with coronary artery disease increases with the number of risk factors.
- Actual event of having a MI is currently referred to as Acute Coronary Syndrome.

ACUTE CORONARY SYNDROME
- Clinical manifestations –
  1. Substernal chest pain – (Diabetics may have no pain. Women may have atypical pain) unrelieved by rest or NTG.
  2. SOB
  3. Nausea
  4. Weakness/ Fatigue
  5. Dizziness
  6. Feeling of impending doom

PHYSICAL EXAM
- Elevated BP (occasionally may be hypotensive). Important to check in both arms.
- Increased heart rate and respiratory rate
- Skin color changes
- Jugular vein distention (JVD)
- Cardiac auscultation – Gallops, murmurs, rubs may be present
LEVINE’S SIGN

• Universal sign for Myocardial Infarction
• Tightly gripped fist placed over the sternum

DIAGNOSIS OF MYOCARDIAL INFARCTION

• PHYSICAL SYMPTOMS
• EKG
• ENZYMES
• HISTORY
• CORONARY ANGIOGRAPHY

DIAGNOSTIC PROCEDURES

• Chest x-ray – note size of heart and examine lungs
• EKG
• Echocardiogram
• Nuclear cardiology
• Thallium imaging
• Cardiac catheterization

ACLS PROTOCOL FOR ACS

• Oxygen (Titrate to keep O2 Sat at 94%)
• EKG
• IV
• Pain Control – NTG if no relief Morphine
  — Pain control important decreases myocardial O2 consumption, decreases anxiety, NTG and Morphine both vasodilate – watch
• Soluble Aspirin, (Chew 4 baby Asa)
• Labs -Cardiac Enzymes, Lytes, CBC, BUN, Cr, PT/PTT, Glucose

ANTICOAGULATION

• Aspirin
• Unfractionated heparin (PTT at 2-2.5 x normal)
• Platelet glycoprotein inhibitors IIB/IIIa platelet inhibitors, (Aggrastat, Integritil, ReoPro)
• Low molecular weight heparin (LMWH), Lovenox SQ or IV.
• Clopidogrel (Plavix) -300mg loading dose
• Prasugrel (Effient) – Newer medication may be used in place of plavix 60mg loading dose – faster onset than plavix

FIBRINOLYTIC THERAPY

• Dissolves clots in Coronary Arteries
• Consider if cath lab not available
• Optimal door-to-drug time less than 30 min.
• Screen for contraindications (Partial List)
  1. Recent CVA
  2. Recent GI Bleed
  3. Recent Surgery
  4. Traumatic CPR
  5. SBP >180; DBP>110
**FIBRINOLYTIC AGENTS**
- Alteplase, Recombinant (rtPA)
- Streptokinase
- Reteplase, Recombinant
- Tenecteplase

**PERCUTANEOUS CORONARY INTERVENTION (PCI)**
- Angioplasty with or without coronary stenting
- Should be less than 90 minutes from door to inflation of catheter
- Earlier reperfusion better outcome. Ideal within 3 hrs of onset of pain
- Some benefit up to 12 hours from onset

**CP - EKG NON DX - CAD ?**
- Check enzymes
- Nuclear stress test Exercise or Chemical—Hold the caffeine or any med that slows the heart rate especially if using adenisone.
- Exercise Stress Echo— if can’t get heart rate up may use dobutamine and do a chemical stress test. Hold meds that slow Heart Rate.

**After an MI**
- Most common complication post MI is an arrhythmia usually monitored in ICU
- Common to have increase in temperature due to inflammatory process due to myocardial cell death
- Activity level once moved to telemetry unit patient encouraged to be up in room and hall with cardiac rehab

**BETA BLOCKERS**
- Increase myocardial salvage in area of infarct
- Prevent extension of infarction by reducing oxygen consumption
- Reduce short and long term mortality rates in survivors of acute MI
- Reduce the incidence of PVC’s & VF/VT
- Initiated po in first 24 hrs of onset of MI, (IV if hypertensive or tachyarrhythmias at admission)

**CONTRAINDICATIONS TO BETA BLOCKERS**
- Bradycardia (HR <60)
- Hypotension (SBP<100)
- Severe Heart Failure
- Shock
- Second or Third degree heart block
- Severe COPD (relative)
- History of Asthma (relative)
Considerations with Beta Blockers

- May decrease reaction to hypoglycemia in insulin requiring diabetics
- May contribute to weight gain especially in type 2 diabetics
- Impotence is significant side effect
- Abrupt discontinuation may cause hypertensive reaction
- Reduce total work possible by 15% so contribute to fatigue if can’t increase HR

ACE INHIBITORS

- Given orally in first 12 to 24 hours
- Prevent adverse LV remodeling
- Delay progression of heart failure
- Decrease sudden death and recurrent MI
- Continue long term if tolerated (an angiotension receptor blocker, ARB, should be administered to patients intolerant of ACE’s)

ANTIARRHYTHMICS

- Not given routinely
- Can actually cause arrhythmias
- Most common
  1. Amiodarone – for atrial and vent arrhythmias
  2. Lidocaine – for vent arrhythmias
  3. Procainamide – for atrial and vent arrhythmias

CALCIUM CHANNEL BLOCKERS

- Prevent passage of calcium ions across the myocardial cell membrane
- Causes vasodilation of coronary and peripheral arteries – Increases coronary blood flow
- Decreased contractile force of the myocardium – decreased myocardial oxygen demand
- Decreased cardiac workload

CHF

- To Diagnose Heart Failure
  - History – SOA – Edema – Meds – Smoking
  - Labs – BNP – Lytes – Thyroid – H & H
  - Chest X Ray
  - Echo – Important to determine if have HF and to determine type and to determine treatment.
  - Rare to need cardiac cath to determine diagnosis of HF or treatment of HF

TERMS ASSOCIATED WITH HF

- RIGHT SIDED
- LEFT SIDED
- SYSTOLIC
- DIASTOLIC
CONGESTIVE HEART FAILURE (CHF)

- Inability of heart to effectively pump blood forward
- Left heart failure –
  1. Left ventricle produces less than normal cardiac output
  2. Backup of blood into the pulmonary vasculature
  3. Pulmonary congestion

PATIENT HISTORY

- Fatigue – low cardiac output
- DOE – decreased exercise tolerance
- Orthopnea- SOB in recumbent position
- PND- paroxysmal nocturnal dyspnea
- Nocturia
- Cough – venous congestion

Congestive Heart Failure (Continued)

- Right Heart Failure
  1. Tends to follow Left Heart Failure as the right ventricle has to pump against greater pulmonary vascular resistance
  2. Right ventricle produces less than normal cardiac output
  3. Backup of blood systemically
  4. Congestion in Liver, spleen, extremities

Patient History (Continued)

- Weight Gain – NA and water retention
- Ankle or abdominal edema
- Chest Pain – may occur with LV dilation and increased LV oxygen demands
PHYSICAL EXAM

- Sinus tachycardia
- S3 gallop
- Crackles in lungs
- JVD (Jugular Vein Distention) – congestion of portal vein and hepatic enlargement
- HJR (Hepatojuglar Reflux)

Physical Exam (Continued)

- Liver engorgement
- Acites
- Bilateral ankle edema
- Hoarseness – pressure from increased myocardial size presses on the laryngeal nerve

TESTING FOR HEART FAILURE

- Chest X-Ray – check vascular congestion and heart size
- EKG - look for old or new MI or arrhythmias like At Fib as cause for CHF
- ABG’s or O2 Sats
- BNP

BNP – Brain Natriuretic peptide

- Found in cardiac ventricles
- Released in response to stretch and higher volume in ventricles
- Level increases as CHF worsens
- Can be used to differentiate dyspnea due to CHF from other causes such as COPD
- May be extremely elevated and not have HF

EJECTION FRACTION

- Single most valuable measurement with CHF
- Determined by Echocardiogram, Cardiac Cath
- Is a measure of the percent of blood pumped out with each heart beat
- Normal EF is between 55% to 75%
Important to know If Diastolic or Systolic Heart Failure

• HFNEF – Heart Failure with Normal EF
  – Over last 20 years have seen increase – may be due to increase in hypertension
  – Diastolic Heart Failure
  – Problem ventricle can’t fill but normal EF
  – Why can’t diagnose or judge severity of HF solely on EF

CHF TREATMENT

• Low Sodium Diet
• Daily Wts
• Medications
  – Diuretics
  – Ace Inhibitors
  – Vasodilators – Calcium Channel Blockers
  – Beta blockers
  – Digitalis – Used to be front line now rarely used unless other medications not working

DIGITALIS

• Only available oral inotropic agent
• Reduces symptoms and readmission
• Does not prolong life
• Hold for HR below 60
• Can be given IV or PO
• Use with caution if on amiodarone or diltiazem as can increase serum dig concentrations

DIURETICS

• Severe LV dysfunction causes aldosterone secretion which causes Na and H2O retention
• Types available –
  1. Mild congestion – thiazides (HCTZ) or K – sparing (Aldactone)
  2. Moderate Congestion – Loop diuretics – Lasix or Bumex
  3. More severe – Zaroxolyn may be added

VASODILATORS

• Standard for Chronic Heart Failure
• Reduce afterload
• Lower pressure and volume in LV
• Increase Cardiac Output
• Increase exercise tolerance

TYPES OF VASODILATORS

• Nitrates – dilate venous system and decrease venous return and decrease preload
• SL NTG, Nitro patch, Isosorbide
• Tend to develop tolerance
• Side effects – headaches hypotension
**ACE INHIBITORS**

- Angiotensio-converting enzyme
- Standard Tx for CHF
- Decrease mortality and increase exercise tolerance
- Decreases LV remodeling and enlargement when used after MI
- Start at low dose and gradually increase based on BP

**SIDE EFFECTS OF ACE INHIBITORS**

- Hypotension
- Decreased renal function – check BUN & Cr
- Hyperkalemia – avoid use with K+sparing diuretics or K+
- Persistent nonproductive cough

**ANGIOTENSION RECEPTOR BLOCKERS (ARB’S)**

- Usually used only when there are side effects with ACE inhibitors
- Diovan (valsartan) - has been approved by FDA for heart failure.

**ANTIARRHYTHMIC THERAPY**

- Frequent PAC’s, PVC’s, At. Fib and asymptomatic runs of VT are common in heart failure
- Do not treat if asymptomatic
  1. Proarrhythmic effect of antiarrhythmics
  2. Not proven to prevent sudden death
  3. Negative inotropic effects occur with some antiarrhythmic drugs
  4. AICD improves survival

**BETA BLOCKERS**

- May improve long term results by decreasing ischemia and suppressing renin-angiotensin system
- Coreg (Carvedilol) helps decrease progression of HF
  1. Worsening of HF symptoms may occur initially
  2. Hypotension – eat before to slow absorption and lower chance of orthostatic effects
  3. If also on ACE inhibitors take 2 hrs apart
  4. Bradycardia may limit dosage
- Metoprolol (Lopressor) and bisoprolol (Zebata) also utilized. Labetalol for IV use.

**MAGNESIUM**

- Increased incidence of low Mg++ in CHF
- Loop diuretics increase urinary excretion of Mg++
- Digoxin decreases renal tubular reabsorption of Mg++
BIVENTRICULAR PACEMAKERS

• Cardiac resynchronization therapy
  1. Three lead system — RA, RV, LV
  2. Improved EF
  3. 2/3 of patients improvement

CALCIUM CHANNEL BLOCKERS

• Different Calcium Channel Blockers differ greatly in their effects, indications and side effects. Cannot be considered as equivalents.
  • Positive effect on myocardial oxygen supply and demand
  • Relax coronary vascular smooth muscle and peripheral vessels
  • Slow conduction thru the AV node

INDICATIONS FOR CALCIUM CHANNEL BLOCKERS

• Angina
• Hypertension
• Supraventricular Tachycardias
• Control Ventricular rate in At. Fib (Cardizem – (diltazem) commonly used)

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ATRIAL FIBRILLATION

– Often asymptomatic
– Pulse irregular
– Deficit between apical and radial pulse
– Increases risk for pulmonary emboli and CVA – often first sign of At Fib is TIA or Stroke.
– Decreases atrial kick
– Left untreated can lead to cardiovascular collapse, HF, thrombus formation and systemic arterial or pulmonary embolism
CAUSES OF AT. FIB
• Valve disease
• CAD
• Acute MI
• Post open heart surgery
• Pericarditis
• Atrial septal defects
• Hyperthyroidism

Causes of Atrial Fib (Continued)
• Hypoxia
• Infection
• Excessive coffee, alcohol or cigarette use
• Fatigue and stress

TREATMENT OF AT. FIB
• Control the rate/convert/maintain sinus rhy
  1. Calcium Channel Blocker (cardizem)
  2. Dofetilide – (Tikosyn) (risk of long QT)
  3. Flecainide – (Tambocor)
  4. Propafenone – (Rythmol)
  5. Sotalol –(Betapace)
  6. Digoxin – not as common
  7. Beta Blockers
  8. Amiodarone

Anticoagulation
• Type of anticoagulation based on
  – Cost
  – Risk of stroke
  – Risk of falls
  – Compliance
  – Speed of desired onset of anticoagulation
  – Valvular or non-valvular atrial fib?
  – Type of valve (mechanical vs. bioprosthetic)

Types of Anticoagulants
• Aspirin – somewhat effective – safest if fall risk
• Heparin –IV if need to bridge to surgery or to longer acting coagulant
• Warfarin –
  – Measure INR requires frequent lab work
  – follow consistent diet

Newer Anticoagulants
Do not require monitoring labs
• Rivaroxaban (Xarelto)
  – Oral given once a day
  --Factor Xa inhibitor
• Apixaban (Eliquis)
  – Oral twice a day
  – Factor Xa inhibitor
• Dabigatran (Pradaxa)
  – Twice a day
  – Direct thrombin inhibitor
CARDIOVERSION

- Utilized if onset of At. Fib less than 48 hours (over 48 hrs, anticoagulate first)
- May need TEE before safe to convert
- May need medical treatment to maintain SR or control rate if can’t convert
  1. Digoxin, procainamide, propranolol, amiodarone, calcium channel blocker

LIFE THREATENING ARRHYTHMIAS

1. Bradycardia — AV Block, CHB
2. Ventricular Tachycardia
3. Ventricular Fibrillation
4. Asystole and PEA
   - May require medication and or pacemaker
   - Investigate the cause, due to medications or MI?

BRADYCARDIA

- Severe Bradycardia – rate below 50
- If Symptomatic Consider Medications — Atropine 0.5 mg IV every 3 to 5min (Max 3 mg), Dopamine IV infusion 2-10 mcg/kg per minute, Epinephrine IV 2-10 mcg per minute

VENTRICULAR TACHYCARDIA (VT)

- VT with a pulse
  1. Stable — treat with medication (amiodarone, lidocaine, procanamide)
  2. Unstable — cardiovert
- VT without a pulse
  1. Same as V Fib
  2. Defibrillate

VENTRICULAR FIBRILLATION

- No cardiac output
- Immediate defibrillation
- Effective CPR

PULSELESS ELECTRICAL ACTIVITY (PEA)

- Electrical pattern on monitor/EKG
- No palpable pulse
- Successful treatment is to treat cause
- Need effective CPR and Epinephrine
- Possible causes — Hypovolemia, hypoxia, cardiac tamponade, tension pneumothorax, pulmonary emboli, hyperkalemia, acidosis, some drug toxicities.
ASYSTOLE

• Effective CPR
• Epinephrine
• Very poor prognosis
• Treat the cause
• Investigate code status