Cardiovascular Pharmacology

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Major Cardiovascular Pathologies Requiring Pharmacological Intervention

- Hypertension
- Arrhythmia
- Heart failure
- Reduced vascular blood flow

Antihypertensive drugs

- Diuretics
- Beta-blockers
- Alpha adrenergic blockers
- Calcium channel blockers
- Angiotensin-converting enzyme inhibitors
- Angiotensin II receptor blockers
- Peripheral vasodilators

The "olol"s

- The Beta-Blockers (B1)
- These agents are antagonist of the beta-receptors of the sympathetic nervous system.
- They reduce cardiac output by diminishing the sympathetic nervous system response and sympathetic tone.

The "olol"s

- The Beta-Blockers
- Vascular resistance and heart rate decrease causing reduced blood pressure. Beta-blockers can either block the beta- receptor, beta 2 receptor or BOTH.
- The selective beta-blockers (B1) are specific to one type of receptor only.

The "olol"s

- following are the common beta-blockers- the '-OLOL'
- Non-selective (B1 and B2)
  - Propranolol
  - Carteolol
  - Nadolol
  - Penbutolol
  - Pindolol
  - Timolol

- Selective and Specific (B1)
  - Acebutolol
  - Atenolol
  - Betaxolol
  - Bisoprolol
  - Metoprolol

Pharmacodynamics

- The mechanism of action of the beta-blockers
- These agents block the beta-adrenergic receptors in the body, thereby decreasing the heart rate and in turn, the blood pressure.
- The non-selective agents block both Beta1 and 2 receptors causing bronchial constriction.
- The onset of action is 30 minutes and the duration may range from 6-12 hours.

- **Clinical Indications of the beta-blockers**
- Hypertension
- Angina pectoris
- Myocardial infarction
- **Contraindications and precautions**
- These agents are not given to patients with:
- Heart blocks
- Bradycardia
- Congestive heart failure
- Chronic obstructive pulmonary disease
- Diabetes

Pharmacodynamics

Side effects and adverse effects

- CVS- Bradycardia , Hypotension, rebound hypertension when abruptly stopped
- Respi- Bronchoconstriction, bronchospasms
- Others- insomnia, depression, nightmares, constipation
- Impaired ability of the liver to convert glycogen to glucose causing HYPOGLYCEM

Implementation

- Monitor patient's vital signs. Take the heart rate before giving the drug
- Instruct the patient to take the drug as prescribed. Warn not to abruptly stop the medication
- Suggest to avoid over-the-counter medications
- Give health teaching as to name of drug, dosages and side effects.
- Remind client NOT to change position abruptly as to avoid orthostatic Hypotension

- Alert diabetic clients of the possible hypoglycemic effect
- Inform that this can cause sexual dysfunction
- Implementation
- Advise client to utilize other means to control blood pressure such as diet modification, exercise, lifestyle changes, etc
- Advise to eat high fiber foods to counter-act constipation

The Prils

The Angiotensin Converting Enzyme Inhibitors

- These are commonly called ACE inhibitors because the agents BLOCK the conversion of AI to AII in the LUNGS.
- These agents alter one of the mechanisms of blood pressure control- the RAAS or renin-angiotensin-aldosterone system.
- Angiotensin II is a very powerful vasoconstrictor and stimulus for the release of aldosterone.

The Prils

- Benazepril
- Captopril- prototype
- Enalapril
- Enalaprilat
- Fosinopril
- Lisinopril
- Moexipril
- Quinapril
- Ramipril
- Trandorapril

Pharmacodynamics

- : The mechanism of action of the ACE inhibitors
- These agents prevent the conversion of angiotensin I to angiotensin II by inhibiting the enzyme in the lungs- the angiotensin converting enzyme.
- The action leads to decreased AII and decreased aldosterone level leading to a decrease in blood pressure.
- The effect of lowering the blood pressure is attributed to the decrease in cardiac workload and decrease peripheral resistance and blood volume

Clinical indications of the ACE inhibitors

- Hypertension, either alone or in combination with other agents.
- Congestive heart failure, left ventricular dysfunction

Contraindications and Precautions in the Use of ACE inhibitors

- Presence of allergy is a clear contraindication.
- The ACE inhibitors are NOT given to patients with renal dysfunction because these drugs may cause further decrease in renal blood flow.
- If given to pregnant women, the drugs cross the placenta and produce renal abnormalities in the fetus

The adverse effects

- CVS- reflex tachycardia , chest pain, angina, cardiac arrhythmias
- CNS- dizziness, drowsiness, and lightheadedness
- GIT- GI irritation, nausea, vomiting, peptic ulcer, constipation and liver damage
- Renal- renal insufficiency, proteinuria
- Others- rash, photosensitivity, dermatitis and alopecia, sodium excretion and potassium retention, fatal pancytopenia.
- COUGH - this cough is really unrelenting and bothersome.

The "sartans"

- The "sartans"
  - The Angiotensin II Receptor Blockers
  - These are SELECTIVE agents that specifically bind to the angiotensin II receptors in the blood vessels and adrenal cortex to prevent the release of aldosterone and to prevent vasoconstriction.
  - Losartan
  - Candesartan
  - Irbesartan
  - Losartan
  - Telmisartan
  - Valsartan

Pharmacodynamics

- The mechanism of action of the A-R-B
- These agents work by attaching to the Angiotensin II receptors in the vascular smooth muscles and in the adrenal gland.
- The action results in VASODILATION because AII action (constriction) is inhibited and BLOCKAGE of aldosterone release

Indications

- Hypertension, either alone or in combination.
- These agents are also used if the patient cannot tolerate the unrelenting cough associated with ACE inhibitors.

Contraindications and precautions

- These agents are contraindicated in the presence of allergy.
- It is NOT GIVEN to pregnant mothers because of the associated FETAL DEATH and severe fetal abnormalities. Lactating women should also avoid these drugs because they can affect the neonate.

The adverse effects

- CNS- headache, dizziness, weakness, syncope and orthostatic Hypotension
- GIT- Diarrhea, abdominal pain, nausea, dry mouth and tooth pain
- Respiratory- mild cough
- Skin- rash, dry skin and alopecia

Calcium channel blockers

- These agents prevent the movement of calcium into the cardiac and smooth muscle cells when the cells are stimulated.
- This blocking of calcium will interfere with the muscle cell's ability to contract, leading to a loss of smooth muscle tone, vasodilation, and a decrease in peripheral resistance .
- These effects will decrease blood pressure, cardiac workload, and myocardial oxygen consumption .

Calcium channel blockers

- The calcium channel blockers that are used in the treatment of hypertension include the following:
- The "-dipine" and others
- Diltiazem (Cardizem, Tiamate)
- Verapamil
- Amlodipine (Norvasc)
- Felodipine (Plendil)
- Isredipine (DynaCirc)
- Nicardipine(Cardene)
- Nifedipine (CALCIBLOC, Procardia XL)-prototype!
- Nisoldipine (Sular)

Pharmacodynamics: Mechanism of action

- Calcium channel blockers inhibit the movement of calcium ions across the membranes of myocardial and arterial muscle cells, altering the action potential and blocking muscle cell contraction.
- This effect will depress myocardial contractility, slow cardiac impulse formation in the conductive tissues, and relax and dilate arteries, causing a fall in blood pressure and a decrease in venous return.

Indications

- Calcium channel blockers are very effective in the treatment of angina because they decrease the cardiac workload.

- The main use of calcium channel blockers is the treatment of angina
- Also in hypertension
- Also in vascular spasm= Raynauds

CONTRAINDICATION and PRECAUTIONS

- These drugs are contraindicated in the presence of allergy to any of these drugs
- With heart block or sick sinus syndrome because these could be exacerbated by the conduction-slowing effects of these drugs
- With renal and hepatic dysfunction, which could alter the metabolism and excretion of these drugs; and with pregnancy and lactation because of the potential for adverse effects on the fetus and neonate.

ADVERSE EFFECTS

- The adverse effects associated with these drugs are related to their effects on cardiac output and on smooth muscle.
  - CNS effects include dizziness , lightheadedness, headache, and fatigue.
  - GI problems can include nausea and hepatic injury related to direct toxic effects hepatic cells.
  - Cardiovascular effects include hypotension, bradycardia, peripheral edema, and heart block . Skin flushing and rash may also occur

The Vasodilators

- Vasodilators produce relaxation of the vascular smooth muscle, decreasing peripheral resistance and reducing blood pressure.
- They cause the reflex tachycardia that occurs when blood pressure drops

- Indications
  - The vasodilators are used to treat severe hypertension

- Diazoxide (Hyperstat)
- Hydralazine (Apresoline)
- Minoxidil (Loniten)
- Sodium Nitroprusside (Nitropress)
- Tolazoline (Priscoline)

Pharmacodynamics

- : Mechanism of action of the vasodilators
- The vasodilators act directly on vascular smooth muscle to cause muscle relaxation, leading to vasodilation and drop in blood pressure .
- They are indicated for the treatment of severe hypertension that has not responded to other therapy.

Contraindications and Precautions

- The vasodilators are contraindicated in the presence of known allergy to the drug; with pregnancy and lactation because of the potential for adverse effects on the fetus and neonate; and with any condition that could be exacerbated by a sudden fall in blood pressure, such as cerebral insufficiency.

the adverse effects of the vasodilators

- CNS- dizziness , anxiety, headache
- CVS- reflex tachycardia , CHF, chest pain, edema; skin rash, lesions (abnormal hair growth with minoxidil), hypotension
- GI upset, nausea, and vomiting
- Cyanide toxicity (dyspnea, headache, vomiting, dizziness, ataxia, loss of consciousness, imperceptible pulse, absent reflexes, dilated pupils, pink color, distant heart sounds, shallow breathing) may occur with nitroprusside , which is metabolized to cyanide and which also suppresses iodine uptake and can cause hypothyroidism

Drugs for the Treatment of Congestive Heart Failure

- Vasodilators- Nitrates that act to directly relax vascular muscle tone and cause decrease in blood pressure with pooling of blood in the veins. The preload and afterload will be decreased
- ACE inhibitors- are agents that block the conversion of angiotensin I to angiotensin II. The result is blockage of the vasoconstriction and decreased blood volume. The afterload will be decreased.

- Diuretics are employed to decrease the blood volume, which decreases the venous return and the blood pressure. The results are decreased preload and decreased afterload
- Beta stimulators will stimulate the beta receptors in the sympathetic nervous system, increasing the myocardial contraction- called positive inotropic effect.

- Cardiotonic drugs- these agents affect the INTRACELLULAR calcium levels in the heart muscles leading to increased contractility . The result is increased cardiac output, increased renal blood flow, increased perfusion and increased urine formation. The cardiotonic drugs are: the cardiac glycosides and the phosphodiesterase inhibitors.

- The cardiac glycosides are agents extracted from the foxglove plant. They are available in oral and parenteral preparations. The following are the cardiac glycosides:
- Digoxin (Lanoxin)
- Digitoxin (Crystodigin)
- Ouabain

The cardiac glycosides

Pharmacodynamics: the Mechanism of action

- They ***increase the level of CALCIUM*** inside the cell by inhibiting the Sodium-Potassium pump.
- More calcium will accumulate inside the cell during cellular depolarization.

The cardiac glycosides

- ***Positive inotropic*** Effect- the myocardium will contract forcefully
  - Increased cardiac output
  - Increased blood flow to the body organs like the kidney and liver
- ***Negative chronotropic*** effect- the heart rate is slowed due to decreased rate of cellular repolarization
  - Bradycardia
- ***Decreased conduction velocity*** through the AV node

The cardiac glycosides

Clinical Use of the cardiac glycosides

- Treatment of congestive heart failure
- Treatment of dysrhythmias like atrial flutter, atrial fibrillation and paroxysmal atrial tachycardia

The cardiac glycosides

Contraindications and Precautions

- Contraindicated in the presence of allergy to any cardiac glycoside.
- They are ***NOT*** given to patients with ventricular dysrhythmias, heart block or sick sinus syndrome, aortic stenosis, acute MI, electrolyte imbalances (***HYPOKALEMIA, HYPOMAGNESEMIA and HYPERCALCEMIA***) and renal failure (may cause accumulation of drug)

The cardiac glycosides

Pharmacodynamics: the Adverse Effects of the Cardiac glycosides

- CNS- Headache, weakness , seizures and drowsiness
- CVS- arrhythmias
- If digitalis toxicity is developing- the nurse must assess the following adverse effects: ***Anorexia, nausea and vomiting, visual changes- YELLOW halo around an object, and palpitations or very slow heart rate***

The cardiac glycosides

Drug-Drug Interactions

- If taken with Verapamil, Amniodarone, quinidine, quinine, erythromycin and tetracyclines- can increase the risk of INCREASED effects of digitalis.
- ***If taken with potassium-losing diuretics like furosemide- can INCREASE the risk of toxicity and arrhythmias. Potassium replacement must be given.***
- If given with cholestyramine, charcoal and colestipol- can cause impaired absorption of digitalis

The cardiac glycosides

Implementation

- Administer the initial rapid digitalization and loading dose as ordered intravenously
- Monitor the ***APICAL*** pulse rate for ONE full minute before administering the drug. Withhold the drug if
  - Less than 60 in adults
  - Less than 90 in infants
  - More than 110 in adults
- Retake pulse in one hour, if pulses remain abnormal, refer!

The cardiac glycosides

Implementation

- Check the spelling of the drug- DIGOXIN is different from DIGITOXIN!
- Check the dosage preparation and the level of digitalis in the blood. (*Therapeutic level is 0.5 to 2.0 nanograms/mL*)
- Administer intravenous drug VERY slow IV over 5 minutes to avoid arrhythmias. Do NOT administer intramuscularly because it can cause severe pain

The cardiac glycosides

Implementation

- Administer the drug ***without food if possible to avoid delayed absorption***. Weight patient daily to determine fluid retention
- Maintain emergency equipment and drugs= Potassium salts, Lidocaine for arrhythmias, phenytoin for seizures, atropine for bradycardia.
- Provide comfort measures- small, frequent meals, adequate lighting, comfortable position, rest periods and safety precautions

The cardiac glycosides

Evaluation

Evaluate effectiveness of the drug:

Increased urine output

Normal heart rate in arrhythmia

The Antianginal drugs

- In the treatment of angina, three agents are commonly employed-
  - ***Organic nitrates***
  - ***Beta-blockers and***
  - ***Calcium-channel blockers.***
- The benefits of the drugs lie in their different mode of action.

The Antianginal drugs

- The nitrates can cause vasodilatation of the veins and to some extent, coronary artery

The Antianginal drugs

- Beta-blockers will decrease the heart rate

The Antianginal drugs

- Calcium-channel blockers will decrease force of contraction leading to a decreased myocardial workload and demand.
- They can also produce vasodilation

The Organic nitrates

- These agents are simple nitric and nitrous acid esters of alcohols. Being alcohol, they differ in their volatility. The following are the nitrates commonly used:
- ***Nitroglycerin-*** A moderately volatile nitrate
- Isosorbide Dinitrate (Isordil) or mononitrate
- Amyl nitrate- an extremely volatile nitrate

The Organic nitrates

Nitroglycerin

- This agent is supplied in oral, spray, transdermal and ointment preparations.

The Organic nitrates

Pharmacodynamics: the mechanism of action

- ***Nitroglycerin relaxes the smooth muscles in the vascular system by its conversion to nitric oxide, a chemical mediator in the body that relaxes smooth muscles.***

The Organic nitrates

Administered nitrates

↓

Increased nitrates in the blood

↓

increased formation of nitric oxide

↓

increased cGMP formation

↓

increased dephosphorylation of myosin

↓

Vascular smooth muscle relaxation

↓

vasodilatation

The Organic nitrates

Pharmacokinetics- absorption to excretion

- It can be given orally, parenterally and topically.
- The onset of action of nitroglycerin is more than 1 hour.
- Because significant first-pass hepatic effect, Nitroglycerin is given SUBLINGUALY.

The Organic nitrates

Pharmacodynamics: Side effects and adverse effects

- ***HEADACHE is the most common effect of nitroglycerin.***
- CVS- postural Hypotension, facial flushing, tachycardia
- TOLERANCE- the tolerance to the actions of nitrates develop rapidly. This can be managed by providing a day of abstinence.

The Nitrates

Implementation

- Monitor vital signs, especially watchful for hypotensive episodes
- Advise patient to remain supine or sit on a chair when taking the nitroglycerin for the first time. Emphasize that he should change his position slowly or rise from bed slowly to avoid orthostatic Hypotension
- Offer sips of water before giving sublingual nitroglycerin because dryness may inhibit drug absorption

The Nitrates

Implementation

- Apply nitroglycerin ointment to the designated mark on paper.
- The nurse should remove any excess ointment on the skin from the previous dose.
- She should ***NEVER USE*** her bare fingers because the drug can be absorbed, utilize gloves or tongue blades instead.

The Nitrates

Implementation

- Apply nitroglycerin patch to an area with few hairs. Never touch the medication portion.
- The patch and the ointment should NOT be applied near the area for defibrillation because explosion and skin burns may result

The Nitrates

IMPLEMENTATION

- Provide client health teaching- the sublingual nitroglycerin tablet is USED if chest pain occurs
- **The dose may be repeated if pain is unrelieved within 5 minutes.**
- **Repeat the medication administration if the pain has not yet subsided.**
- ***DO NOT give more than 3 tablets!*!! If chest pain persists for more than 15 minutes, hospital consult should be done immediately.**

The Nitrates

IMPLEMENTATION

- Instruct the client to avoid alcohol while taking nitroglycerin to avoid potentiating the hypotensive effect of the medication
- If beta blockers and calcium-channel blockers are given, instruct the patients to consult the physician before discontinuing the medication

The Nitrates

IMPLEMENTATION

- Other components of health teaching for home self-administration:
  - If taking Sublingual Nitroglycerin, the patient should be instructed to ***place the tablet under the tongue*** for quick absorption.
  - A burning sensation/biting/stinging sensation may indicate that the tablet is ***FRESH!***
  - Store the tablet in a ***dark container***, keep it away from heat and direct sunlight to avoid lessening the potency

The Nitrates

IMPLEMENTATION

- Other components of health teaching for home self-administration:
  - HEADACHES are common in the initial period of nitroglycerin therapy. Advise patient to take PARACETAMOL for relief
  - The nitroglycerin patch is applied once a day, usually in the morning. The sites should be rotated, in the chest, arms and thighs avoiding hairy areas.

Drugs for Shock

Dopamine

- This is a sympathomimetic drug often used to treat Hypotension in shock states that are not caused by Hypovolemia.
- This drug is an immediate precursor of nor-epinephrine, occurs naturally in the CNS basal ganglia where it functions as a neurotransmitter.

Drugs for Shock

Dopamine

- Pharmacodynamics: It can activate the alpha and beta adrenergic receptor depending upon the concentration. It stimulates receptors to cause ***cardiac stimulation and renal vasodilation***.
- The dose range is 1-20 micrograms/kg/min

Drugs for Shock

Dopamine

- Pharmacokinetics: Dopamine is administered IV, excreted in the urine.
- At ***low dose*** (1-2 micrograms), dopamine DILATES the renal and mesenteric blood vessels producing an increase output (dopaminergic effect)

Drugs for Shock

Dopamine

- At ***moderate dose*** of 2-10 micrograms, dopamine enhance cardiac output by increasing heart rate (beta 1-adrenergic effect) and elevates blood pressure through peripheral vasoconstriction (alpha adrenergic effect)

Drugs for Shock

Dopamine

- At higher doses of more than 10 micrograms- ***vasoconstriction of all vessels*** will predominate that can lead to diminished tissue perfusion

Drugs for Shock

Dopamine

- Dopamine is indicated to treat Hypotension, to increase heart rate and to increase urine output (given less than 5 mg/kg/min)
- The nurse typically prepares the dopamine drip- dopamine (at a concentration of 400-800 mg) is mixed in 250 mL D5W and administered as drip via an infusion pump for precise dosage administration.
- Sodium bicarbonate will inactivate the dopamine

Drugs for Shock

Dopamine

- Pharmacodynamics: side effects- Tachycardia
  hypertension
  ectopic beats, angina
  dysrhythmias, myocardial ischemia, nausea and vomiting.

Drugs for Shock

Dopamine: Nursing consideration

- **Check the IV site hourly for signs of drug infiltration of dopamine, which can cause severe tissue necrosis.**
- **Phentolamine should be infiltrated in multiple areas to reduce tissue damage.**
- **Drug is effective if Urine output is increased and BP is increased**

Anti-Arrhythmias

- Arrhythmias (sometimes called Dysrhythmias) are conduction dysfunctions caused by abnormalities in impulse generation or impaired transmission of the impulses.
- They are simply deviations from the normal rate or pattern of the heartbeat.

Anti-Arrhythmias

Four things may happen during arrhythmias:

- The heart will beat too FAST (tachycardia, either the atrium or the ventricle)
- The heart will beat too slow (bradycardia)
- The heart will respond to other impulses generated by the cardiac cells (other than the SA node)
- The heat will respond to impulses traveling along extra pathways

Anti-Arrhythmias

- Anti-arrhythmic agents are given to modify impulse generation and conduction.
- The desired action of these antidysrhythmics is to restore the cardiac rhythm to normal.
- They affect the action potential of the cardiac cells, altering their automaticity, conductivity, or BOTH

Anti-Arrhythmias

- **Because the anti-arrhythmic drugs affect the conduction system, they also can produce ARRHYTHMIAS!**
- **They are also Pro-Arrhythmics!**

Anti-Arrhythmias

The classes of anti-arrhythmic agents:

- Class 1 agents - are fast SODIUM channel blockers that affects the depolarization phase.
- Class 2 agents- are BETA blockers that affect the depolarization.
- Class 3 agents- are POTASSIUM channel blockers that diminish the outward potassium current during repolarization of cardiac cells.
- Class 4 agents- are calcium channel blockers that decrease the calcium influx into the specialized cardiac muscle cells causing slowed conduction

Anti-Arrhythmias

The classes of anti-arrhythmic agents:

- **Class 1 agents -**
  - **Class 1 A- Quinidine, Procainamide, Disopyramide, Moricizine**
  - **Class 1 B- Lidocaine, Mexiletine, Tocainide**
  - **Class 1 C- Encainide, Flecainide, propafenone**

Anti-Arrhythmias

- **The classes of anti-arrhythmic agents:**
- **Class 2 agents- are BETA blockers that affect the depolarization. They decrease the conduction velocity, automaticity and recovery time. Examples are: Propranolol, Metoprolol, pindolol, acebutol and esmolol**

Anti-Arrhythmias

- **The classes of anti-arrhythmic agents:**
- **Class 3 agents- are POTASSIUM channel blockers that diminish the outward potassium current during repolarization of cardiac cells.**
- **They increase the refractory period and prolong the action potential. Examples are: sotalol, bretylium, amniodarone**

Anti-Arrhythmias

- **Class 4 agents- are calcium channel blockers that decrease the calcium influx into the specialized cardiac muscle cells causing slowed conduction. They increase the refractory period of the AV node, which decreases the ventricular response.**
- **The examples of calcium channel blockers are diltiazem and verapamil**

Anti-Arrhythmias

- **Miscellaneous- digoxin and adenosine**

Anti-Arrhythmias

General Nursing Process for Anti-arrhythmics

Assessment

- Patient History- the nurse obtains health and drug histories. She should elicit symptoms of shortness of breath, heart palpitations, coughing, chest pain, previous angina or dysrhythmias, and the current medications.
- Physical Examination- the nurse performs assessment and baseline monitoring
- Laboratory exams- the nurse obtains ECG results and cardiac markers- CK-MB, AST and LDH)

Anti-Arrhythmias

Nursing Diagnoses

- Alteration on perfusion: decreased cardiac output
- Alteration in thought processes and sensory-perceptual alteration
- Anxiety related to irregular heartbeat
- Risk for activity intolerance
- Risk for injury related to CNS effects
- Knowledge deficit regarding drug therapy

Anti-Arrhythmias

Planning

- The client will no longer experience abnormal cardiac rhythm
- The client will comply with the drug regimen

Anti-Arrhythmias

Implementation

- Monitor Vital signs especially BP and HR. Patient can develop Hypotension
- Administer the parenteral drugs slow IV for a period of 2-3 minutes
- Monitor ECG for abnormal patterns and report findings.
- Instruct to report palpitations and abnormal cardiac rate
- Maintain life support equipment on stand by to treat severe adverse reactions that might occur

Anti-Arrhythmias

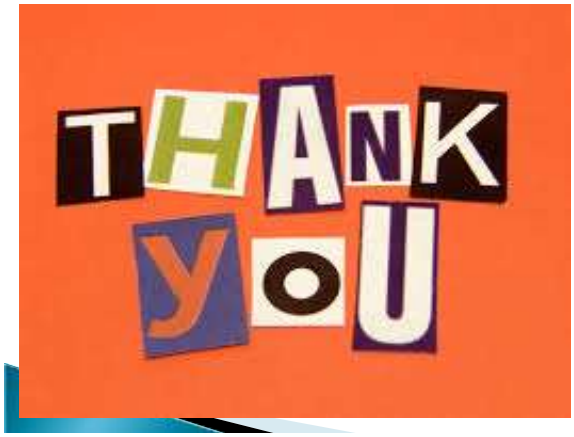
Implementation

- Establish safety precautions- side rails, lighting, and noise control.
- Provide client teaching. Instruct the client to take the prescribed drug as ordered. Emphasize the client to avoid alcohol, caffeine and tobacco.
- Alcohol can intensify the hypotensive effects, caffeine increases the cathecolamine levels and tobacco can promote vasoconstriction.
- Instruct the client to report side effects including dizziness, faintness, and nausea and vomiting.

Anti-Arrhythmias

Evaluation

- Evaluate the effectiveness of the prescribed anti-dysrhythmic by comparing heart rates with baseline heart rate.
- Assess the client's response to the drug.
- Monitor for adverse effect- sedation, Hypotension, cardiac arrhythmias, respiratory depression, CNS effect.
- Evaluate the effectiveness of the teaching plan. Monitor the effectiveness of comfort measures and compliance to regimen.


THANK
YOU