

Comprehending Cardiac Medications: Foundational Cardiac Concepts That Must Be Understood

Cardiac Output (CO) ... $CO=SV \times HR$ (stroke volume x heart rate)

- **Cardiac output:** The amount of blood the heart pumps through the circulatory system in a minute. The amount of blood put out by the left ventricle of the heart in one contraction is called the stroke volume. The stroke volume and the heart rate determine the cardiac output. A normal adult has a cardiac output of 4.7 liters (5 quarts) of blood per minute (normal range 4–8 liters/minute).
- **Stroke volume:** Stroke volume (SV) is the volume of blood pumped from one ventricle of the heart with each beat. The term stroke volume can apply to each of the two ventricles of the heart, although it usually refers to the left ventricle. The stroke volumes for each ventricle are generally equal, both being approximately 70 mL in most adults.
- Stroke volume is influenced by the amount of blood that returns to right atrium from venous circulation (RIGHT SIDE) and left atrium (LEFT SIDE) from pulmonary veins.
 - Higher SV increases workload of the heart...typically due to too much VOLUME.
 - Tachycardia INCREASES cardiac O₂ demands, workload, and DECREASES stroke volume because there is less time for the ventricles to fill.
 - Goal of medication therapy with any cardiac patient is to DECREASE cardiac workload through manipulating SV, HR, preload, afterload, and contractility.

Preload

Preload is the filling pressure of the right ventricle (RV) and left ventricle (LV). It is influenced by how full the body's tank is and amount of venous return. Higher venous return (fluid volume overload) will result in higher SV. Fluid volume deficit will result in lower SV. If monitored with a central venous line, normal values are 2–6 mm/Hg. This is also the same as a central venous pressure (CVP).

- Pressure/Stretch in ventricles end diastole just before contraction.
- In a healthy heart, as you increase preload, you will increase stroke volume which will increase force of contraction (Starling's law).
- Only up to a certain point will this continue, then further stretching may actually decrease contractility. This is what will happen with patients with chronic heart failure.
- Drugs that cause venous dilation (nitrates)-DECREASE preload.
- Diuretics that eliminate excess fluid volume DECREASE preload.

Starling's Law of the Heart:

- If preload is increased, greater quantity of blood that is ejected during systole due to increased stretch of the myocardium and larger amount of circulating blood volume present.
- But only up to a maximal point. Greatest force of contraction is when the muscle fibers are stretched 2 ½ times their normal length.

- Overstretch of cardiac muscle is like an overstretched rubber band; it will DECREASE cardiac contractility and efficiency over time. This is why an enlarged heart due to heart failure is NOT a good thing!

Afterload

- Force of resistance that the LV must generate to open aortic valve.
- Correlates w/SBP—How much pressure is needed to push blood out of the LV, and into systemic arterial circulation.
- Influenced by resistance of blood vessels in the body—Are the arteries dilated or constricted? If arterial vasodilation is present, afterload is DECREASED and workload of the heart is DECREASED. If arterial vasoconstriction is present, afterload is INCREASED and workload of the heart is INCREASED.
- Arterial vasodilators (Ca⁺⁺ channel blockers [CCB], ACE inhibitors) then DECREASE afterload and decrease the workload of the heart.

3) Contractility: Contractility of the cardiac myocardium independent of Starling mechanism

- Ability of heart to change force of inherent contraction strength as needed.
- Influenced by Ca⁺⁺ in action potential...Therefore, how will calcium channel blockers (CCB) influence contractility? (They will DECREASE contractility and must be used with caution in those with heart failure).
- Decrease in contractility (inotropic effect) DECREASES cardiac workload and O₂ demands.
- Negative inotropic meds: CCB, beta blockers
- Positive inotropic: Digoxin, Dopamine & Epinephrine gtt's.

Ejection Fraction—% (EF) on echocardiogram

- 60–70% normal.
- With each contraction 60–70% of the blood in the LV is ejected into circulation.
- As this percent goes down, it is reflecting the loss of cardiac contractility and degree of heart failure.
- 30–35% EF is half normal cardiac output.
- 10–15% EF end-stage heart failure-terminal.

I. Angiotensin Converting Enzyme (ACE) Inhibitors...(end in “pril”)

- Captopril (Capoten)
- Enalapril (Vasotec)
- Lisinopril (Prinivil)

Mechanism of Action: A plasma protein-angiotensinogen is secreted by the kidneys when BP falls. This converts inactive liver protein angiotensinogen to angiotensin I. The conversion to angiotensin II is enhanced by angiotensin-converting-enzymes (ACE) from the lungs to be one of the most potent vasoconstrictors in the body. The effects of angiotensin II are:

- Vasoconstriction of arterioles and veins (increases afterload).
- Stimulation of the sympathetic nervous system (SNS) (increases workload of heart).
- Retention of water by the kidneys due to aldosterone a hormone secreted by the adrenal glands (increases preload).

In heart failure (HF), each of these normal physiologic actions will worsen underlying HF and must be counteracted. This is why ACE-I are the first medication used in HF.

ACE inhibitors block the conversion of angiotensin I to angiotensin II. This inhibition decreases angiotensin II concentrations and causes:

- Vasodilation which decreases SBP (decreases afterload) and decreases sodium and water retention (decreases preload).

Uses: HTN, HF, coronary artery disease (CAD)

Side Effects–Most Common: hypotension, dry cough, dizziness

- Angioedema (life threatening) Laryngeal swelling that can cause asphyxia. Facial swelling also concerning precursor and a clinical RED FLAG.

Nursing Considerations:

- Obtain BP before administering—hold typically if SBP <90.
- Change position slowly—especially with elderly to prevent orthostatic changes.
- Monitor for decreased WBC count, hyperkalemia as well as liver function, and creatinine (metabolized by liver-excreted by kidneys).

II. Beta Blockers...(end in “lol”)

- Metoprolol (Lopressor)
- Atenolol (Tenormin)
- Propranolol (Inderal)
- Labetolol (Trandate)

Mechanism of Action: Selectively inhibits beta (sympathetic/fight or flight) receptors primarily of the heart (Beta 1). Sympathetic nervous system (SNS) stimulation normally causes:

- Increased heart rate.
- Increased BP.
- Increase myocardium contractility and oxygen demand.
- ALL THESE EFFECTS INCREASE WORKLOAD OF THE HEART!

By blocking the SNS it effectively causes the opposite effects and DECREASES workload of the heart...decreased heart rate, decreased BP, and decreased contractility. This is why it must be used with caution in those with history of HF. They have excessive activation of SNS which will worsen CHF over time. B-Blockers very effective to manage this complication of HF, and will commonly be used in addition to ACE-I.

Uses: HTN, HF, acute MI, CAD, ventricular dysrhythmias

Side Effects–Most Common: bradycardia, hypotension, fatigue, weakness

Nursing Considerations:

- Obtain BP and HR before administering—hold typically if SBP <90. HR <60.
- Change position slowly—especially with elderly to prevent orthostatic changes.
- Contraindicated in worsening HF, bradycardia, heart block...use with caution in diabetes, liver disease (1)

III. Calcium Channel Blockers

- Diltiazem (Cardizem)
- Verapamil (Calan)
- Nifedipine (Procardia)
- Amlodipine (Norvasc)

Mechanism of Action: Inhibits or “blocks” the influx of extracellular calcium into the membranes of the heart and vascular smooth muscle cells of heart and body. This blocking of calcium ions is responsible for causing the following:

- Dilation of coronary and systemic arteries w/resultant decrease in BP (improves cardiac O₂ supply & decreases afterload).
- Decrease of myocardium contractility (decreases workload).
- Slows AV node conduction (decreases heart rate).
- Diltiazem most commonly used to slow HR and AV node conduction in atrial fibrillation.

Uses: HTN, angina, controlling rapid heart rate in SVT or atrial fibrillation.

Side Effects–Most Common: peripheral edema, hypotension, constipation.

Nursing Considerations:

- Change position slowly—especially with elderly to prevent orthostatic changes.
- Measure I&O closely and fluid status due to potential for edema.
- Monitor liver and kidney function (metabolized in liver-excreted by kidneys).
- Obtain BP and HR before administering—hold typically if SBP <90. HR <60 (1).

IV. Nitrates

- Nitroglycerine sublingual (Nitrostat)—SHORT acting
- Isosorbide Mononitrate (Imdur)—LONG acting
- Isosorbide Dinitrate (Isordil)—LONG acting
- Nitroderm topical patches—LONG acting

Mechanism of Action: Able to relax and dilate both coronary arterial vessels and systemic venous smooth muscle.

- Dilation of veins reduces amount of blood that returns to heart (decreases preload). With less blood to pump, cardiac output is reduced and workload of the heart decreased lowering myocardial oxygen demand.
- Dilation of coronary arteries can help improve blood flow to heart as well as well as relieve angina caused by coronary artery vasospasm.

Uses: Angina

Side Effects–Most Common: hypotension, tachycardia, dizziness, headache (dilates cerebral vessels causing mini migraine)

Nursing Considerations:

- Tolerance common and serious problem with long acting nitrates. Nitrates lose their effectiveness if transdermal patches remain on continually. Patches must be taken off at night and then reapplied in the morning.

- Contraindicated if client taking any erectile dysfunction meds as these are a similar nitrate that improves blood circulation to the penis—synergistic effect can cause dramatic hypotension (1).

V. Diuretics

- Furosemide (Lasix) -most potent
- Bumetanide (Bumex) -most potent
- Hydrochlorothiazide (HCTZ)-mod. potency
- Spironolactone (Aldactone)-mild potency-K⁺ sparing

Mechanism of Action:

- **Loop diuretics**—Most potent diuretics that prevent sodium reabsorption in the loop of henle causing rapid/large diuresis with resultant loss of K⁺, and smaller amounts of Mg⁺, and Na⁺. Because of their potency are not used for maintenance but when large diuresis is desired such as in acute HF exacerbation (decreases preload).
- **Thiazide diuretics**—Increases excretion of sodium and water by inhibiting sodium reabsorption in distal tubule. Most common maintenance diuretic. K⁺ loss needs to be monitored.
- **Potassium sparing diuretics**—Weakest of all diuretics due to not blocking reabsorption of sodium. Used when client is at risk for developing hypokalemia. Must also assess for development of hyperkalemia.

Uses: HTN, HF, renal insufficiency/acute renal failure

Side Effects—Most Common: dehydration, orthostatic hypotension, hypokalemia, hyponatremia, hypomagnesemia with loop and thiazide diuretics, potential for hyperkalemia w/spironolactone (Aldactone).

Nursing Considerations: *Change position slowly-especially with elderly to prevent orthostatic changes.

- Change position slowly-especially with elderly to prevent orthostatic changes.
- Obtain BP before administering—hold typically if SBP <90.
- Monitor sodium and K⁺ levels closely as well as GFR and creatinine.
- Aldactone and ACE inhibitors can cause resultant hyperkalemia.
- If on Aldactone—make sure does not use potassium based salt substitutes or foods rich in K⁺ (1).