## Vascular System Pathophysiology- Reduced blood flow (venous insufficiency) through peripheral blood vessels characterizes all peripheral vascular diseases.

Effects of altered blood flow depend on extent of tissue demands exceeding supply of O2 & nutrients W/ high tissue needs, even slightly reduced blood flow may be inadequate to maintain tissue integrity.

Tissues fall prey to ischemia, become malnourished, & ultimately die unless blood flow is restored.

**Pump Failure**

An inefficient heart causes inadequate Inadequate peripheral blood flow

**Left-side HF** (ventricular failure) (CHF) causes pulmonary venous congestion & reduction in forward flow or cardiac output, resulting in inadequate arterial blood flow to the tissues.

**Right-side HF** (ventricular failure) causes systemic venous congestion and a reduction in forward flow

### Alterations in Blood and Lymphatic Vessels

**Arteries** can become damaged or obstructed as a result of atherosclerotic plaque, thromboemboli, chemical or mechanical trauma, infections or inflammatory processes, vasospastic disorders, and congenital malformations.

Sudden arterial occlusion causes profound and often irreversible tissue ischemia and tissue death. When they develop gradually, then less risk of sudden tissue death because collateral circulation may develop, giving that tissue the opportunity to adapt to gradually decreased blood flow.

**Venous** blood flow can be reduced by a thromboembolus, incompetent venous valves, mechanical trauma, reduced pumping action of surrounding muscles.

If ↓venous blood flow then ↑venous pressure, subsequent ↑ in capillary hydrostatic pressure = edema

Lymphatic vessels can get obstructed by tumor, mechanical trauma or inflammation = edema.

Edematous tissues don't get adequate O2/Nutrition so get breakdown, injury, and infection

### Circulatory Insufficiency of the Extremities

Many types of peripheral vascular diseases exist, most result in ischemia and produce some of the **same symptoms: pain, skin changes, diminished pulse, and possible edema.**

Peripheral vascular disease is categorized as arterial, venous, or lymphatic.

### Arteriosclerosis and Atherosclerosis

**Arteriosclerosis:** most common disease of the arteries; means "hardening of the arteries." It is a diffuse process where muscle fibers and the endothelial lining of the walls of small arteries and arterioles become thickened.

**Atherosclerosis:** affects intima of the large and medium-sized arteries.

Deposits of lipids, calcium, blood components, carbohydrates, and fibrous tissue on the intimal layer of the artery.

Called atheromas or plaques.

Pathology of arteriosclerosis & atherosclerosis differ, but usually occur together, terms used interchangeably.

Atherosclerosis is a generalized disease of the arteries, when present in extremities, then usually present elsewhere

#### Risk Factors for Atherosclerotic Vascular Disorders

**Modifiable lifestyle factors:** Dyslipidemia, obesity, sedentary lifestyle,smoking, stress

**Modifiable Conditions**: DM, HTN, and Metabolic Syndrome (↑ triglycerides, ↑ cholesterol).

**Non-modifiable**: age, family history, gender, and race.

**Labs**: Elevated C-reactive protein, Hyperhomocysteinemia

**S&S (really more like complications)** depend on organ or tissue affected.

Coronary atherosclerosis (heart disease), angina, and acute MI.

Cerebrovascular diseases: TIA, stroke.

Atherosclerosis of the aorta, including aneurysm, and atherosclerotic lesions of the extremities

Renovascular disease (renal artery stenosis and end-stage renal disease)

**Medical Management**

Modification of risk factors, controlled exercise program to improve circulation & function, medication therapy, and

## PYRAMID TERMS

**afterload** The force against which the heart has to pump (peripheral resistance) to eject blood from the left ventricle. Factors and conditions that would impede blood flow increase left ventricular afterload.

**arterial pressure** The pressure of the blood against the arterial walls. Pressure can be measured indirectly by sphygmomanometer or directly by arterial catheter. Readings are expressed as systolic over diastolic. Arterial pressure increases when the cardiac output, peripheral resistance, or blood volume increases.

**automaticity** The ability of cardiac cells to initiate an impulse spontaneously and repetitively without external neurohormonal control. The pacemaker cells have the highest rate of automaticity of all cardiac cells.

**baroreceptors** Specialized nerve endings located in the walls of the aortic arch and carotid sinuses that are affected by changes in the arterial blood pressure (BP). Increases in arterial pressure stimulate baroreceptors and the heart rate and arterial pressure decrease. Decreases in arterial pressure lead to a lessened stimulation of the baroreceptors, vasoconstriction occurs, and the heart rate increases; also called pressoreceptors.

**blood pressure (BP)** The force exerted by the blood against the walls of the blood vessels. If the blood pressure falls too low, blood flow to the tissues, heart, brain, and other organs become inadequate. If the blood pressure becomes too high, the risk of vessel rupture and damage increases.

**capillary pressure or hydrostatic pressure** The pressure exerted by the blood against the capillary wall. Normal capillary pressure is 25 to 30 mm Hg at the arterial end of the capillaries, and 10 to 15 mm Hg at the venous end.

**cardiac output** The total volume of blood pumped through the heart in 1 minute. The normal cardiac output is 4 to 7 L/min. Cardiac output equals stroke volume multiplied by heart rate. Cardiac output can be calculated via the thermodilution method when the client has a pulmonary artery catheter (Swan-Ganz catheter).

**chemoreceptors** Nerve endings located in the aortic arch and carotid bodies that are stimulated by hypoxemia and that subsequently transmit impulses to the central nervous system.

**conductivity** The ability of the heart muscle fibers to propagate electrical impulses along and across cell membranes.

**contractility** The inherent ability of the myocardium to alter contractile force and velocity. Sympathetic stimulation increases myocardial contractility, so stroke volume increases. Conditions that decrease myocardial contractility reduce stroke volume.

**diastole** The phase of the cardiac cycle in which the heart relaxes between contractions. Diastole represents the period of time when the two ventricles are dilated by the blood flowing into them.

**diastolic pressure** The force of the blood exerted against the artery walls when the heart relaxes or fills.

**excitability** The ability of cardiac muscle cells to depolarize in response to a stimulus. Excitability is influenced by hormones, electrolytes, nutrition, oxygen supply, medication, infections, and nerve characteristics.

**mean arterial pressure (MAP)** An approximation of the average pressure in the systemic circulation throughout the cardiac cycle; used in hemodynamic monitoring. Mean arterial pressure must be at least 60 mm Hg for adequate organ perfusion.

**paradoxical blood pressure** An exaggerated decrease in systolic pressure by more than 10 mm Hg during the inspiratory phase of the respiratory cycle. Normal value is 3 to 10 mm Hg.

**postural (orthostatic) hypotension** A blood pressure decrease of more than 10 to 15 mm Hg of the systolic pressure or a decrease of more than 10 mm Hg of the diastolic pressure and a 10% to 20% increase in heart rate. Postural hypotension occurs when the client's blood pressure is not maintained adequately when moving from a lying to a sitting or standing position.

**preload** The volume of blood stretching the left ventricle at the end of diastole. Preload is determined by the total circulating blood volume and is increased by an increase in venous return to the heart.

**pulmonary capillary wedge pressure (PCWP)** The measurement obtained during momentary balloon inflation of a pulmonary artery catheter; it is reflective of left ventricular end-diastolic pressure. The PCWP normally ranges between 6 and 12 mm Hg. Decreased PCWP indicates hypovolemia, whereas increased PCWP indicates hypervolemia, left ventricular failure, or mitral regurgitation.

**pulse pressure** The difference between the systolic and diastolic pressure. Normal pulse pressure is 30 to 40 mm Hg.

**refractoriness** The inability of the heart to respond to a new stimulus while still in a state of contraction from an earlier stimulus. Refractoriness prevents uncontrolled rapid cardiac contractions and helps preserve the heart rhythm.

**stretch receptors** Nerve endings located in the vena cava and the right atrium that respond to pressure changes affecting circulatory blood volume. When the blood pressure decreases because of hypovolemia, a sympathetic response occurs, causing an increased heart rate and blood vessel constriction. When the blood pressure increases because of hypervolemia, an opposite effect occurs.

**stroke volume** The amount of blood ejected from the left ventricle with each contraction. The normal stroke volume is 70 to 130 mL/heartbeat. The stroke volume can be affected by preload, afterload, contractility, and the Frank-Starling law.

**systole** The phase of contraction of the heart, especially of the ventricles, during which blood is forced into the aorta and pulmonary artery.

**systolic pressure** The maximum pressure of blood exerted against the artery walls when the heart contracts.

**venous pressure** The force exerted by the blood against the vein walls. Normal venous pressures are highest in the extremities (5 to 14 cm $H_2O$ in the arm), and lowest closest to the heart (6 to 8 cm $H_2O$ in the inferior vena cava).

| Laboratory Test | Implications |
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| **Reference Range** | |
| **Blood Chemistries** | |
| Sodium ($Na^+$) 135–145 mEq/L | Low or high serum sodium levels do not directly affect cardiac function.<br>*Hyponatremia:* Decreased sodium levels indicate fluid excess and can be caused by heart failure or administration of thiazide diuretics.<br>*Hypernatremia:* Increased sodium levels indicate fluid deficits and can result from decreased water intake or loss of water through excessive sweating or diarrhea. |
| Potassium ($K^+$) 3.5–5.0 mEq/L | Potassium has a major role in cardiac electrophysiologic function.<br>*Hypokalemia:* Decreased potassium levels due to administration of potassium-excreting diuretics can cause many forms of dysrhythmias, including life-threatening ventricular tachycardia or ventricular fibrillation, and predispose patients taking digitalis preparations to digitalis toxicity.<br>*Hyperkalemia:* Increased potassium levels can result from an increased intake of potassium (eg, foods high in potassium or potassium supplements), decreased renal excretion of potassium, use of potassium-sparing diuretics (eg, spironolactone), or use of angiotensin-converting enzyme inhibitors (ACE inhibitors) that inhibit aldosterone function. Serious consequences of hyperkalemia include heart block, asystole, and life-threatening ventricular dysrhythmias. |
| Calcium ($Ca^{++}$) 8.6–10.2 mg/dL | Calcium is necessary for blood coagulability, neuromuscular activity, and automaticity of the nodal cells (sinus and atrioventricular nodes).<br>*Hypocalcemia:* Decreased calcium levels slow nodal function and impair myocardial contractility. The latter effect increases the risk for heart failure.<br>*Hypercalcemia:* Increased calcium levels can occur with the administration of thiazide diuretics because these medications reduce renal excretion of calcium. Hypercalcemia potentiates digitalis toxicity, causes increased myocardial contractility, and increases the risk for varying degrees of heart block and sudden death from ventricular fibrillation. |
| Magnesium ($Mg^{++}$) 1.3–2.3 mEq/L | Magnesium is necessary for the absorption of calcium, maintenance of potassium stores, and metabolism of adenosine triphosphate. It plays a major role in protein and carbohydrate synthesis and muscular contraction.<br>*Hypomagnesemia:* Decreased magnesium levels are due to enhanced renal excretion of magnesium from the use of diuretic or digitalis therapy. Low magnesium levels predispose patients to atrial or ventricular tachycardias.<br>*Hypermagnesemia:* Increased magnesium levels are commonly caused by the use of cathartics or antacids containing magnesium. Increased magnesium levels depress contractility and excitability of the myocardium, causing heart block and, if severe, asystole. |
| Blood urea nitrogen (BUN) 10–20 mg/dL | BUN and creatinine are end products of protein metabolism excreted by the kidneys.<br>Elevated BUN reflects reduced renal perfusion from decreased cardiac output or intravascular fluid volume deficit as a result of diuretic therapy or dehydration. |
| Creatinine 0.7–1.4 mg/dL | Both BUN and creatinine are used to assess renal function, although creatinine is a more sensitive measure. Renal impairment is detected by an increase in both BUN and creatinine. A normal creatinine level and an elevated BUN suggest an intravascular fluid volume deficit. |
| Glucose Fasting: 60–110 mg/dL | Glucose levels are elevated in stressful situations, when mobilization of endogenous epinephrine results in conversion of liver glycogen to glucose. Serum glucose levels are drawn in a fasting state. |
| Glycohemoglobin (hemoglobin $A_{1c}$)<br>Nondiabetic: 4.4–6.4% | Glycohemoglobin (hemoglobin $A_{1C}$) is monitored in people with diabetes. It reflects the blood glucose levels over 2–3 mo. The glycemic goal is to maintain the hemoglobin $A_{1C}$ below 7% reflecting consistent near-normal blood glucose levels. |

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| | coagulation cascade, the complex interactions among phospholipids, calcium, and clotting factors that convert prothrombin to thrombin. The coagulation cascade has two pathways, the intrinsic and extrinsic pathways. Coagulation studies are routinely performed before invasive procedures, such as cardiac catheterization, electrophysiology testing, and cardiac surgery. |
| Partial thromboplastin time (PTT) 60–70 sec | PTT or aPTT measures the activity of the intrinsic pathway and is used to assess the effects of unfractionated heparin. A therapeutic range is 1.5–2.5 times baseline values. Adjustment of heparin dose is required for aPTT <50 sec (↑ dose) or >100 sec (↓ dose). |
| Activated partial thromboplastin time (aPTT) 20–39 sec | |
| Prothrombin time (PT) 9.5–12 sec | PT measures the extrinsic pathway activity and is used to monitor the level of anticoagulation with warfarin (Coumadin). |
| International normalized ratio (INR) 1.0 | The INR, reported with the PT, provides a standard method for reporting PT levels and eliminates the variation of PT results from different laboratories. The INR, rather than the PT alone, is used to monitor the effectiveness of warfarin. The therapeutic range for INR is 2–3.5, although specific ranges vary based on diagnosis. |

| **Hematologic Studies** | |
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| Complete blood count (CBC) | The CBC identifies the total number of white and red blood cells and platelets, and measures hemoglobin and hematocrit. The CBC is carefully monitored in patients with cardiovascular disease. |
| White blood cell (WBC) count 4500–11,000/$mm^3$ | WBC counts are monitored in immunocompromised patients, including patients with heart transplants or in situations where there is concern for infection (eg, after invasive procedures or surgery). |
| Hematocrit<br>Male: 42–52%<br>Female: 35–47%<br>Hemoglobin<br>Male: 13–18 g/dL<br>Female: 12–16 g/dL | The hematocrit represents the percentage of red blood cells found in 100 mL of whole blood. The red blood cells contain hemoglobin, which transports oxygen to the cells. Low hemoglobin and hematocrit levels have serious consequences for patients with cardiovascular disease, such as more frequent angina episodes or acute myocardial infarction. |
| Platelets 150,000–450,000/$mm^3$ | Platelets are the first line of protection against bleeding. Once activated by blood vessel wall injury or rupture of atherosclerotic plaque, platelets undergo chemical changes that form a thrombus. Several medications inhibit platelet function, including aspirin, clopidogrel (Plavix), and intravenous glycoprotein IIb/IIIa inhibitors (abciximab [ReoPro], eptifibatide [Integrilin], and tirofiban [Aggrastat]). When these medications are administered, it is essential to monitor for thrombocytopenia (low platelet counts). |