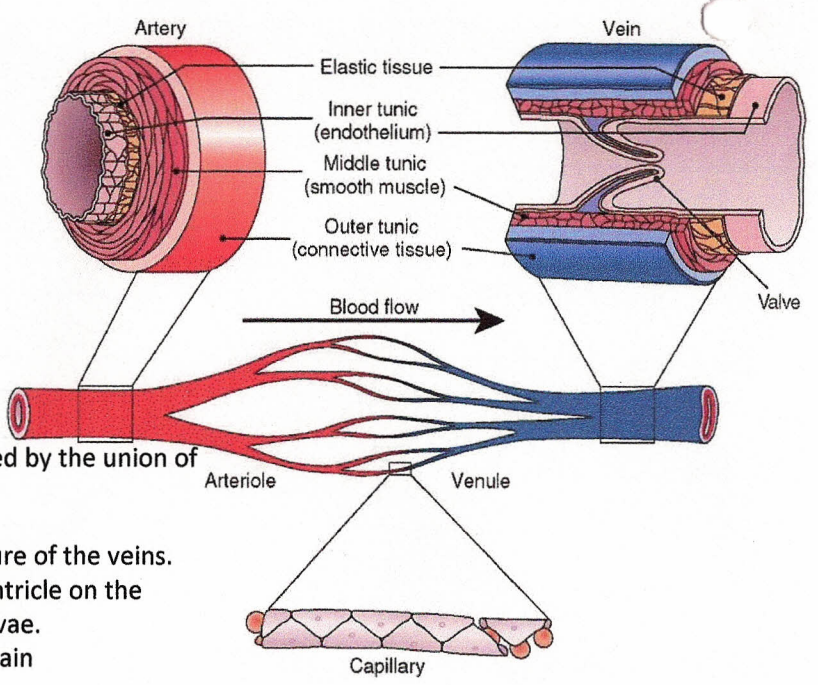
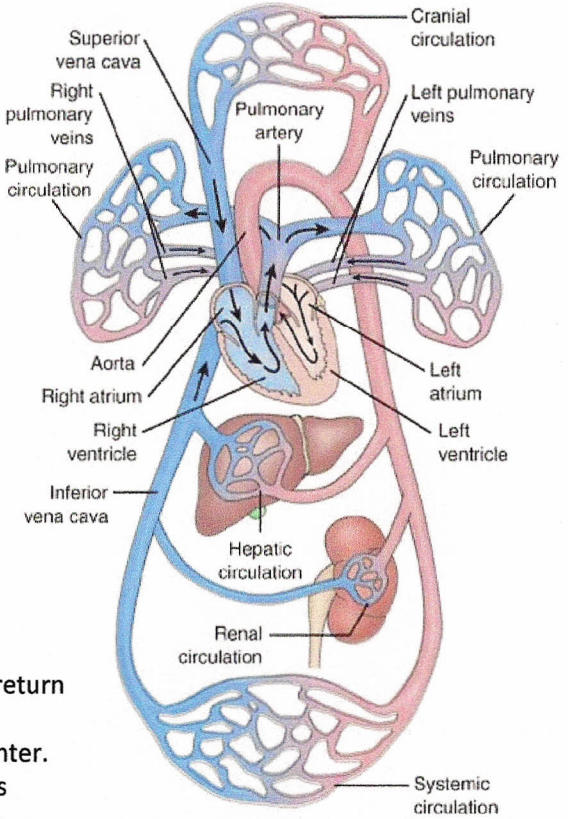
VASCULAR A&P

- Arteries carry blood away from the heart and toward the tissues.
- Arterioles are small subdivisions of the arteries. They carry blood into the capillaries.
- Capillaries : made up of a single layer of endotlhelial cells.
- Venules : sm vessels that receive blood from capillaries & begin its transport back to heart.
- Veins are vessels formed by the merger of venules. They finish blood transport back to heart
  transport of blood until it is returned to the heart
  some veins have valves
  vein musculature is inervated by the SNS
  75% of tot blood volume is in veins



Anastomoses A communication between two vessels
By means of arterial anastomoses, blood reaches vital organs by more than one route.
Some examples of such end-artery unions are as follows:
The circle of Willis, The superficial palmar arch, The mesenteric arches, Arterial arches are formed by the union of tibial artery branches in the foot. There are similar anastomoses in other parts of the body.

Venous Sinuses large channel that drains deoxygenated blood, but doesn't have tubular structure of the veins.
coronary sinus, receives most of the blood from the heart wall, lies bt the left atrium and left ventricle on the heart's posterior surface, and empties directly into the right atrium, along with the two venae cavae.
cranial venous sinuses, which are located inside the skull and drain the veins from all over the brain
The largest of the cranial venous sinuses are the following:
two cavernous sinuses, situated behind the eyeballs, drain the eyes' ophthalmic veins, and give rise to the **petrosal sinuses**, which drain into the jugular veins. The **superior sagittal** sinus is a single long space located in the midline above the brain and in the fissure between the two cerebral hemispheres. It ends in an enlargement called the **confluence of sinuses**.
The two **transverse sinuses**, also called the **lateral sinuses**, are large spaces between the layers of the dura mater. They begin posteriorly from the confluence of sinuses and then extend laterally. As each sinus extends around the skull's interior, it receives additional blood, including blood draining through the **inferior sagittal sinus** and **straight sinus**. Nearly all of the blood leaving the brain eventually empties into one of the **transverse sinuses**. Each sinus extends anteriorly to empty into an internal jugular vein, which then passes through a channel in the skull



lymphatic vessels complex network of thin-walled vessels similar to the blood capillaries.
•This network collects lymphatic fluid from tissues and organs and transports the fluid to the venous circulation.
•The lymphatic vessels converge into two main structures: the thoracic duct and the right lymphatic duct.
•These ducts empty into the junction of the subclavian and the internal jugular veins.
•The right lymphatic duct conveys lymph primarily from the right side of the head, neck, thorax, and upper arms.
•The thoracic duct conveys lymph from the remainder of the body.
•Peripheral lymphatic vessels join larger lymph vessels and pass through regional lymph nodes before entering the venous circulation.
•The lymph nodes play an important role in filtering foreign particles.
•The lymphatic vessels are permeable to large molecules and provide the only means by which interstitial proteins can return to the venous system.
•W/ muscular contrxn, lymph vessels become distorted to create spaces bt the endothelial cells, protein and particles enter.
•Muscular contrxn of the lymphatic walls & surrounding tissues aids in propelling lymph toward venous drainage points

Adequate blo flow depends on the efficiency of heart as a pump, pat y & responsiveness of blood vessels, and adequacy of cir ating blood volume. Nervous system activity, blood viscosity, and the metabolic needs of tissues influence the rate and adequacy of blood flow.
Contraction of the ventricles is the driving force of unidirectional circulation.
When metabolic needs (tissues, organs) increase or decrease blood vessels vasodilate or vasoconstrict accordingly.
If the blood vessels fail to dilate in response to the need for increased blood flow, tissue ischemia (deficient blood supply to a body part) results.
The mechanism by which blood vessels dilate and constrict to adjust for metabolic changes ensures that normal arterial pressure is maintained

Neural and Hormonal Regulation

The **medulla**, located in the brainstem above the spinal cord, is the primary site in the brain for regulating **sympathetic** and **parasympathetic (vagal)** outflow to the heart and blood vessels. The heart and vasculature are regulated, in part, by neural (autonomic) and humoral factors.

Neural mechanisms primarily involve **SNS adrengenic** and **PNS cholingeric** branches of the autonomic nervous system.
In general, the **SNS stimulates the heart and constricts blood vessels** resulting in a rise in arterial pressure.
The **PNS depresses cardiac function and dilates selected vascular beds**.

Humoral mechanisms (circulating or hormonal) includes **catecholamines (epinephrine and norepinephrine), renin angiotensin system, ADH, ANP, & endothelin**.
Epinephrine : ↑ HR and ionotropy (contractility) and vasoconstriction in most systemic arteries and veins. At low doses it vasodilates muscle and liver vessels.
Norepinephrine: ↑ HR and ionotropy (contractility) and vasoconstriction in most systemic arteries and veins. But doesn't shunt to muscle and liver vessels.
Each of these humoral systems directly or indirectly alter cardiac function, vascular function, and arterial pressure.

Vessels

Large arteries branching off aorta (e.g., carotid, mesenteric, renal arteries) distribute the blood flow to specific organs.
Capable of constricting & dilating, but serve almost no role in the reg of pressure and blood flow under normal conditions.
Smaller arteries distribute blood flow w/in the organ. These vessels continue to branch and become **arterioles**.
Both are referred to as **resistance vessels.**
Small arteries & arterioles are the primary vessels involved in the reg of arterial BP as well as blood flow within the organ.
Both are highly innervated by autonomic nerves, and respond to changes in nerve activity and circulating hormones by constricting or dilating.
As arterioles become smaller in diameter, they lose their smooth muscle. Vessels that have no smooth muscle are termed **capillaries**, and represent the smallest vessels within the microcirculation. Capillaries are the primary **exchange vessels** within the body. Across the capillary endothelium, oxygen, carbon dioxide, water, electrolytes, proteins, metabolic substrates and by-products (e.g., glucose, amino acids, lactic acid), and circulating hormones are exchanged between the plasma and the tissue interstitium surrounding the capillary. When capillaries join together, they form **postcapillary venules**, which also serve as exchange vessels, particularly for large macromolecules as well as fluid. As postcapillary venules join together and form larger venules, smooth muscle once again appears. These venous vessels, like the resistance vessels, are capable of dilating and constricting, and serve an important function in regulating capillary pressure. Venules form larger **veins** that serve as the primary **capacitance vessels** of the body - i.e., the site where most of the blood volume is found and where regional blood volume is regulated. For example, constriction of the veins decreases venous volume and increases venous pressure, which alters CO. The final venous vessels are the inferior and superior **vena cava.**

BLOOD'S RETURN TO THE HEART Blood flows in a closed system and must continually move forward as heart contracts.
However, by the time blood arrives in the veins, little force remains from the heart's pumping action.
Also, because the veins expand easily under pressure, blood tends to pool in the veins.
Finally, the force of gravity works against upward flow from regions below the heart.
Mechanisms help to overcome these forces and promote blood's return to the heart in the venous system. These are:
Contraction of skeletal muscles, Valves in the veins prevent back flow and keep blood flowing toward the heart.
Breathing. Pressure changes in the abdominal and thoracic cavities during breathing also promote blood return in the venous system.
Together, these actions serve to both push and pull blood through these cavities and return it to the heart.

Hemodynan (Pressure, Flow, and Resistance) the physical factors .tha vern blood flow.

This hemodynamic relationship can be summarized by:

$$F = \frac{\Delta P}{R} = \frac{(P_A - P_V)}{R}$$

where F= Flow, P = Pressure, A= Artery, V= Vein, and R = Resistance. The equation is the same for the heart blood flow but the ΔP = the difference bt pressure on either side of the valve, i.e. intraventricular Piv- Pao aortic pressure where R is the size of the valve.

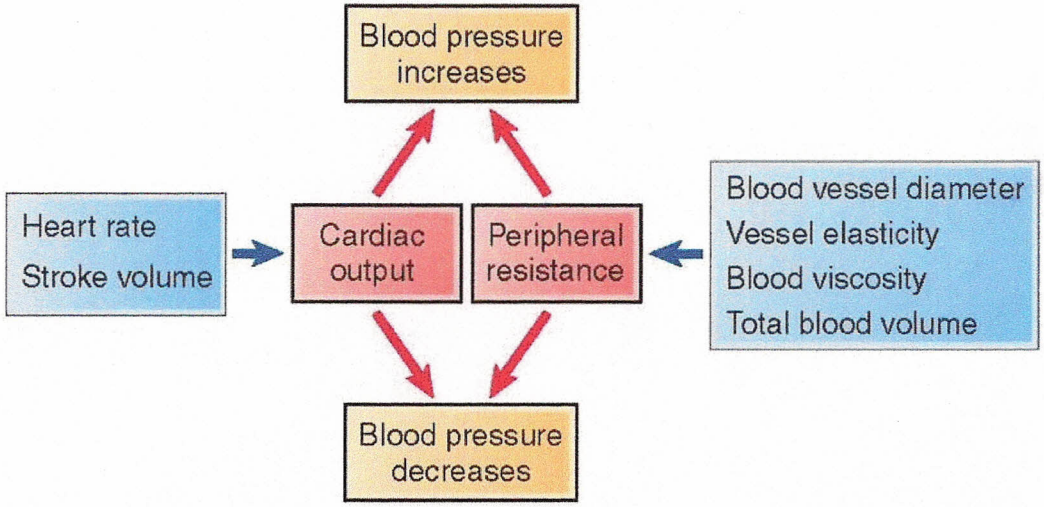
The Pulse The ventricles regularly pump blood into the arteries about 70 to 80 times a minute. The force of ventricular contraction starts a wave of increased pressure that begins at the heart and travels along the arteries. This wave, called the pulse, can be felt in any artery that is relatively close to the surface. Normally, the pulse rate is the same as the heart rate, but if a heartbeat is abnormally weak, or if the artery is obstructed, the beat may not be detected as a pulse

Blood Pressure the force exerted by the blood against the walls of the vessels. Blood pressure is determined by the heart's output and resistance to blood flow in the vessels. If either of these factors changes and there are no compensating changes, blood pressure will change

Resistance to blood flow: opposition to blood flow owing to friction generated as blood slides along the vessel walls. Because the effects of resistance are seen mostly in small arteries and arterioles that are at a distance from the heart and large vessels, this factor is often described as peripheral resistance. Resistance in the vessels is affected by the following factors:

Vasomotor changes: vasoconstriction increases resistance to flow and vasodilation lowers resistance.
The medulla's vasomotor center controls vessel diameter, responds mainly to impulses from baroreceptors in the carotid arteries and the aorta.
When stretched by increased BP (or blood volume), they transmit signals that result in vasodilation.
Simultaneously, central controls slow the heart rate to reduce cardiac output.
With less stretching, the sympathetic nervous system causes the vessels to constrict and causes the heart rate to increase.
Elasticity of blood vessels. Arteries normally expand to receive blood and then return to their original size.
f vessels lose elasticity, as by atherosclerosis, they offer more resistance to blood flow.
Blood vessels lose elasticity with aging, thus increasing resistance and blood pressure.
Viscosity, increased blood viscosity will increase blood pressure.
Increased numbers of red blood cells, as in polycythemia, or a loss of plasma volume, as by dehydration, will increase blood viscosity.
Total blood volume A loss of blood volume, as by hemorrhage, will lower blood pressure.
An increase in blood volume will generate more pressure within the vessels. It will also increase cardiac output by increasing venous return of blood to the heart.

Blood pressure = cardiac output × peripheral resistance



Stroke Volu (SV) is the difference between the ventricular EDV and the ESV. The)V is the filled volume of the ventricle prior to contraction and the ES the residual volume of blood remaining in the ventricle after ejection. In a typical heart, the EDV is about 120 ml of blood and the ESV about 50 ml of blood. The difference in these two volumes, 70 ml, represents the SV. Therefore, any factor that alters either the EDV or the ESV will change SV.

SV = EDV - ESV

For example, an increase in EDV increases SV, whereas an increase in ESV decreases SV.

There are three primary mechanisms that regulate EDV and ESV, and therefore SV.

Preload

Increase in venous return to the heart increases the filled volume (EDV) of the ventricle, which stretches the muscle fibers thereby increasing their preload. This leads to an increase in the force of ventricular contraction and enables the heart to eject the additional blood that was returned to it.

Therefore, an increase in EDV results in an increase in SV.

Conversely, a decrease in venous return and EDV leads to a decrease in SV by this mechanism.

Afterload

R/T the pressure that the ventricle must generate in order to eject blood into the aorta.

Changes in afterload affect the ability of the ventricle to eject blood and thereby alter ESV and SV.

Ex: an increase in afterload (e.g., increased aortic pressure) decreases SV, and causes ESV to increase.

Conversely, a decrease in afterload augments SV and decreases ESV.

It is important to note, however, that the SV in a normal, non-diseased ventricle is not strongly influenced by afterload.

In contrast, the SV of hearts that are failing are very sensitive to changes in afterload.

Inotropy

Changes in ventricular inotropy (contractility) alter the rate of ventricular pressure development, thereby affecting ESV and SV.

Ex: an increase in inotropy (e.g., produced by sympathetic activation of the heart) increases SV and decreases ESV.

Conversely, a decrease in inotropy (e.g.,HF) reduces SV and increases ESV.

It is important to note that the effects of changes in EDV and ESV on SV are not independent.

For example, an increase in ESV usually results in a compensatory increase in EDV.

Furthermore, if SV is increased by increasing EDV, this can lead to a small increase in ESV because of the influence of increased afterload on ESV caused by an increase in aortic pressure.

Therefore, while the primary effect of a change in preload, afterload or inotropy may be on either EDV or ESV, secondary changes can occur that can partially compensate for the initial change in SV.

Ejection Fraction (EF) is the fraction of blood ejected by the ventricle relative to its EDV. Therefore, EF is calculated from: **$EF = (SV / EDV) \cdot 100$**

where SV = stroke volume, EDV = end-diastolic volume

Ejection fraction is most commonly measured using **echocardiography,** non-invasive technique that provides good estimates of EDV and ESV, and stroke volume

In HF, particularly in dilated cardiomyopathy, EF can become very small as SV decreases and EDV increases. In severe heart failure, EF may be 20% or less. EF is often used as a clinical index to evaluate the the inotropic status of the heart. However, it is important to note that there are circumstances in which EF can be normal, yet the ventricle is in failure. One example is diastolic dysfunction caused by hypertrophy in which filling is impaired because of low ventricular compliance and stroke volume is therefore reduced. In this case, both SV and EDV can be reduced such that EF does not change appreciably. For this reason, low ejection fractions are generally associated with systolic dysfunction rather than diastolic dysfunction.

Glossary

anastomosis
junction of two vessels

aneurysm
a localized sac or dilation of an artery formed at a weak point in the vessel wall

angioplasty
an invasive procedure that uses a balloon-tipped catheter to dilate a stenotic area of a blood vessel

ankle-brachial index (ABI) or ankle-arm index (AAI)
ratio of the ankle systolic pressure to the arm systolic pressure; an objective measurement of arterial disease that provides quantification of the degree of stenosis

arteriosclerosis
diffuse process whereby the muscle fibers and the endothelial lining of the walls of small arteries and arterioles thicken

atherosclerosis
inflammatory process involving the accum of lipids, calcium, blood components, carbohydrates, and fibrous tissue on the intimal layer of a large or medium-sized artery

bruit
sound produced by turbulent blood flow through an irregular, tortuous, stenotic, or dilated vessel

dissection
separation of the weakened elastic and fibromuscular elements in the medial layer of an artery

duplex ultrasonography
combines B-mode gray-scale imaging of tissue, organs, and blood vessels with capabilities of estimating velocity changes by use of a pulsed Doppler

intermittent claudication
a muscular, cramplike pain in the extremities consistently reproduced with the same degree of exercise or activity and relieved by rest

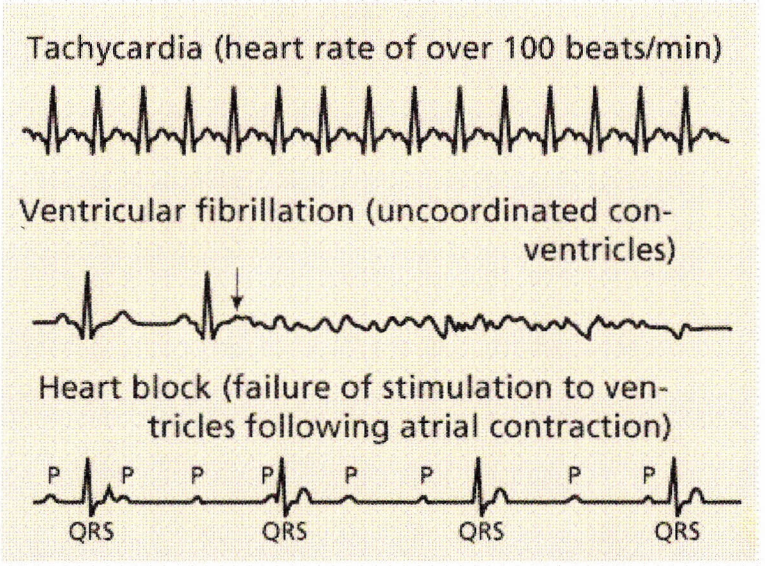
ischemia
deficient blood supply

rest pain
persistent pain in the foot or digits when the patient is resting, indicating a severe degree of arterial insufficiency

rubor
reddish blue discoloration of the extremities; indicative of severe peripheral arterial damage in vessels that remain dilated and unable to constrict

stenosis
narrowing or constriction of a vessel

Some abnormal EKGs

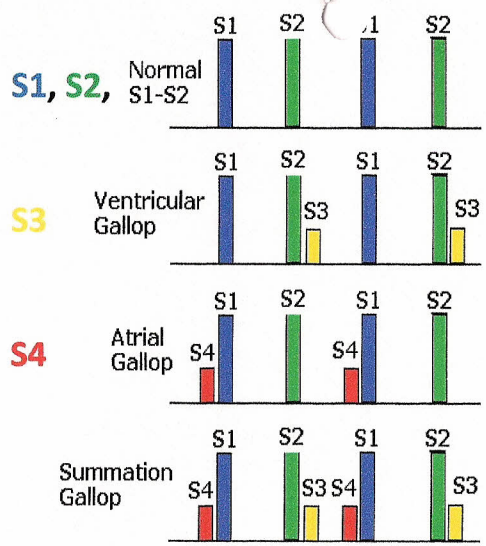


SSESSMENT : HEMODYNAMIC

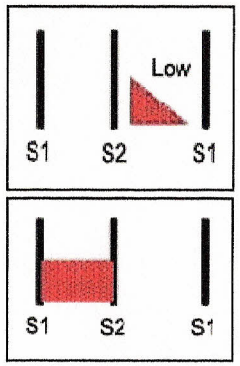
art Sounds

– normal - end of atrial systole, ventricle systole starts and tricuspid and bicuspid (Mitral) valves shut - heard best at apex
– normal - end of ventricle systole, semilunar valves shut – best heard at base
- ventricular gallop sound occurs when blood enters non-compliant ventricles during early diastole.
A suddenly occurring S3 is said to be an early sign of heart failure.
–atrial gallop sound occurring when blood enters from the atrium with atrial contraction into non-compliant ventricular ambers at the end of ventricular diastole. (don't need S4 for exam, probably don't need below either but...)
occurring w/ S3 is called a summation gallop, said to be **a sign of severe heart failure.**

hich valves are closing with S1 and S2? S1 tricuspid & Mitral (AV valves)
here does S1 and S2 fall in the cardiac cycle? S1 End of atrial sytole. S2 end of ventriculare systole.



urmurs - gentle blowing, swooshing sounds
eflect turbulent blood flow through the valves
nditions that can result in murmurs include:
elocity of blood increases
iscosity of blood decreases
structural defects in the valves
nusual openings occur in the chambers
rmurs are described by their :
ocation,Timing,Grading,Pitch,Quality,Radiation
only done by CCU RN)



CO = SV X HR

CO = Stroke Volume (SV) x Heart Rate (HR)
***Normal for CO : 4 to 8 L/minute**
***Normal for SV : 60 to 120 ml/beat**
CO -directly measured by advanced invasive monitoring devices like a Pulmonary Artery Catheter.

icks, Snaps, Rubs (abnormal)

licks –An ejection click can occur with septal fects,abrupt dilation of the aorta, rceful opening of the aortic valve, opening or closing of prosthetic valve.
naps - An abnormal motion of a stenotic mitral valve can cause an opening snap hat is often heard at the beginning of a mitral murmur.
ericardial Friction Rub - A friction sound occurring with myocardial contraction

Preload, Afterload, & Ejection fraction

SV is changed by an alteration in any of the following:
Preload – amt of stretch in atria before it contracts (how full it is)
Load – not a real term - EVDV
Afterload – pressure in the aorta at the heart (my definition)
Contractility (ionotropy) – weakness or strength of contraction
Ejection Fraction: % of blood that actually leaves ventricle at the end ventricular systole – evals L ventricle **55-65% is normal**

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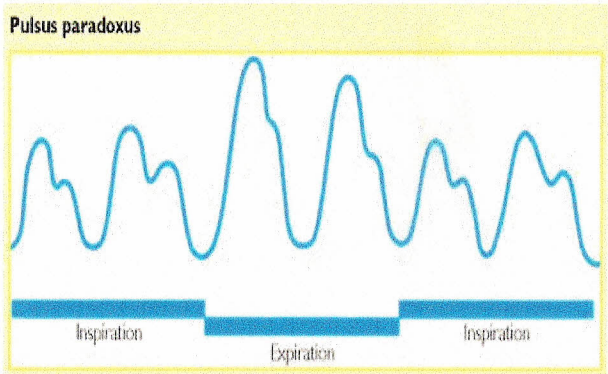
ood Pressure

=CO X Peripheral Resistance
mean arterial pressure
Systolic + Diastolic /2 = MAP
should be > 50 and at ~65
pulsus paradoxus – HTN
pulse pressure
orthostatic measurement
supne, sitting, standing



Pulses

presence or absence – **notify HO if new, undocumented**
Quality – bounding , weak, thready
Symmetry of bilateral pulses in the arms and legs
Pulse Alternans - HTN
Pulse deficit - **Apical HR > radial P (indicates obstruction or poor perfusio**
Assessment of the carotid pulse – ausculcate for bruits (swishing sound)
JVD – is usually first symptom of HF

SSESSMENT : LABS

- ıbs –Overview
- type Natriuretic Peptide (BNP)
- ɔagulation Studies
- ɔmplete Blood Count (CBC)
- ectrolytes
- Iron & Indicators of IDA
- Lipid Profile
- C-Reactive Protein
- Homocysteine
- Type & Cross Match

Blood Draw

- any needed restrictions in food/fluid intake
- any restriction in medication administration
- Collect serial specimens on time (for admin of meds, etc) ie peak and trough
- After blood is drawn monitor venipuncture site for bleeding, hematoma, phlebitis, infection
- Watch for lab results and report any significant abnormalities to the ordering Provider.

type Natriuretic Peptide – normal < 100 pg/mL

neurohormone primarily secreted from the ventricles in response to increased preload with resulting elevated ventricular pressure.

B-type Natriuretic Peptide is a serum marker for heart failure

ynthetic BNP is now available in an **IV form** nesiritide (**Natrecor**)

- used to treat acute exacerbations of heart failure.
- causes arterial and venous dilation & diuresis which clinically results in a decrease in afterload and preload

ɔagulation Studies

PTT - Activated partial thromboplastin time
ɔrmals – APTT 21-35 sec
ıerapeutic APTT (patient on heparin) is 1.5 to 2.5 Xs patient baseline

- Prothrombin time (PT) **11.0-13.0 sec**

R
R <2.0 (not on anticoagulant - normal
R 2.0 to 3.0 – tx for venous thrombosis, PE, and valvular heart disease
R 2.5 to 3.5 – pts w/ mechanical heart valves or being tx'd for recurrent emboli

CBC - Refer NURN 152 CBC

specific to perfusion
Hgb– carries O2
WBC - infection
Platelets - clotting

Miscellaneous
D-Dimer- ↑means need more testing for possible clot
ESR – erythrocyte sedimentation rate
↑may mean inflammation
Dig level- 0.5-2

CBC - NURN 152

- WBC - **4,800 to 10,800/L**
- RBC - **4.2 - 5.4 men**
  3.6 - 5.0 x 106/ul women
- Hct - **42 – 52% men**
  36 - 48% women
- Hgb - **14.0-17.4g/dl men**
  12.0-16.0g/dl women
- Indices - **MCV mean corpuscular v**
  avg size of single RBC
  ↓ Iron-deficiency anemia
- Platelets - **140,000 - 400,000 mm3**

ɔn & Indicators of IAD

Iron
- 50 to 170 *u*g/dL in females
- 65 to 175 *u*g/dL in males

Ferritin
- 20-250 ng/mL in men
- 10 – 120 ng/mL in women <40 yr old
- 12 – 263 ng/mL in women >40 yr old

Transferrin 200-380 mg/dL
TIBC 250 – 350 *u*g/dL

ectrolytes that are most influential in perfusion are:

Sodium 135 – 145 mEq/L
Potassium 3 5 – 5.0 mEq/L
Calcium 4.5 – 5.5 mg/dL (ionized)
Magnesium 1.6 – 2.6 mg/dL

Lipid Profile - Desirable Optimal Levels

- Cholesterol (total) <200 mg/dL
- HDL > 60 mg/dL
- LDL < 100 mg/dL
- Triglycerides <150 mg/dL

C-Reactive Protein 1.0-3.0 mg/L
Made by liver in response to inflammation
May indicate athersclerosis

Homocysteine 8-20 *u*mol/L
Linked to development of athersclerosis

Patients w/ Rh - blood can only receive Rh -blood.
Rh +patients can accept Rh +or Rh-.

Blood Group	Can give blood to	Can receive blood from
AB	AB	AB, A, B, 0
A	A and AB	A and 0
B	B and AB	B and 0
0	AB, A, B, 0	0

SSESSMENT : DIAGNOSTICS Annuplasty etc are under HF section of exemplars

e-test: focused assessment; pre-test checklist; Invasive/Semi-invasive, contrast; - need **consent**, Pt teaching , **Contrast** : contraindicated if allergies - shellfish or iodinated dye; or renal failure. **Kidneys work** (can't flush radioactive contrast post-test)? Safe to leave unit?
During test:: possible sedation, position
Post test: reassess ; if contrast: **monitor for reaction & kidney function.** If invasive: monitor **site**, **monitor for embolus.** If sedation: **monitor vitals, respiration status**, pt safety; teaching.

uclear Scans
ses contrast
e-Procedure IV to inject the contrast
JGA -Multi-gated acquisition scan
RNA - Equilibrium radionuclide angiocardiography
Q Scan -Ventilation & Perfusion Scan

omputed Tomography (CT or CAT) xrays provide cross-sectional images of chest: heart & great vessels.
ay use Contrast
e-test
ay be medicated to prevent renal damage from contrast
ay be NPO depending upon area scanned
eaching regarding the procedure:
sitioned on table while scanner revolves, noninvasive and painless, need to lie perfectly still, IV line if contrast used, machine is very noisy

piral CT (also called helical)
ewest, most accurate, fast. Usually a VQ scan is done first cuz it's cheaper, if that shows moderate to high risk then spiral ct ordered.
old Standard DX for PE
sulting images are three-dimensional rather than two-dimensional

ung Scan
erfusion scanning: injection of a radioisotope, scanning device detects adequacy of the body to pick up the isotope
entilation Scanning: inhalation of radioactive gas (xenon). Pt scanned for lung pick-up of gas. Pt needs to be able to "cooperate" ie ventilate. May be omitted for intubated patients.

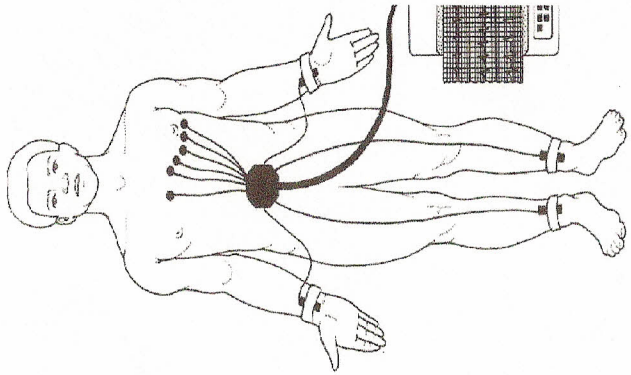
oppler Studies ultrasound device, non-invasive way to hear & eval blood flow.
ses conducting gel
nsducer is slowly moved over vessel area
uplex Ultrasound Imaging use a pulsing Doppler to produce computer generated images – shows stenosis, plaque, clots.

Lead Electrocardiogram

epresents elec al activity of heart at one point in time.

e Procedure

Position supine as flat as possible
Uncover chest & limbs
Identify landmarks
Prepare skin for electrode adherence.
Place electrodes at landmarks
Attach corresponding leads



ost-procedure:

etermine if the EKG appears normal.
epeat EKG needed may leave electrodes on
ave equipment ready for emergency use.

hocardiograph Transthoracic Echocardiography done simultaneously with an ECG. Noninvasive ultrasound , transmission of high-frequency sound waves
oduce images
ed to assess heart valves, direction of blood flow, size and motion of myocardium and heart chambers.
nducting gel applied to the chest wall, transducer applied, moved slowly
holds breath for short periods of time, may lay on left side (better visualization)

ansesophageal Echocardiography (TEE) Invasive. Used to visualize back side of heart.
e-Test - fasts for 6 hours
uring test:
line for sedation and meds
roat sprayed w/ numbing agent
monitored
nscious sedation
st-procedure
onitor for gag reflex return
lverse reactions to sedation
ıy potential injury

giography (cardiac cath) visualization, via fluoroscopy, of the structure and patency of blood vessels **(shows ischemia, lesions, efflusions, occlusions)**
es contrast, invasive
e-test: NPO up to 12 hrs, checklist completed, IV access site groin or antecubital space prepped,
uring: lie prone, cath site is numbed, possible IV conscious sedation
st-test:
sess renal dysfunction/insufficiency – may give mucimix (sp?) pre and post proceedure so pt can have test
sess vitals and pedal pulse q15min, then q30min then every hour.
y flat 2-6 HOURS