Blood flow and Blood pressure [Control]

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Assot Professor

Learning objectives

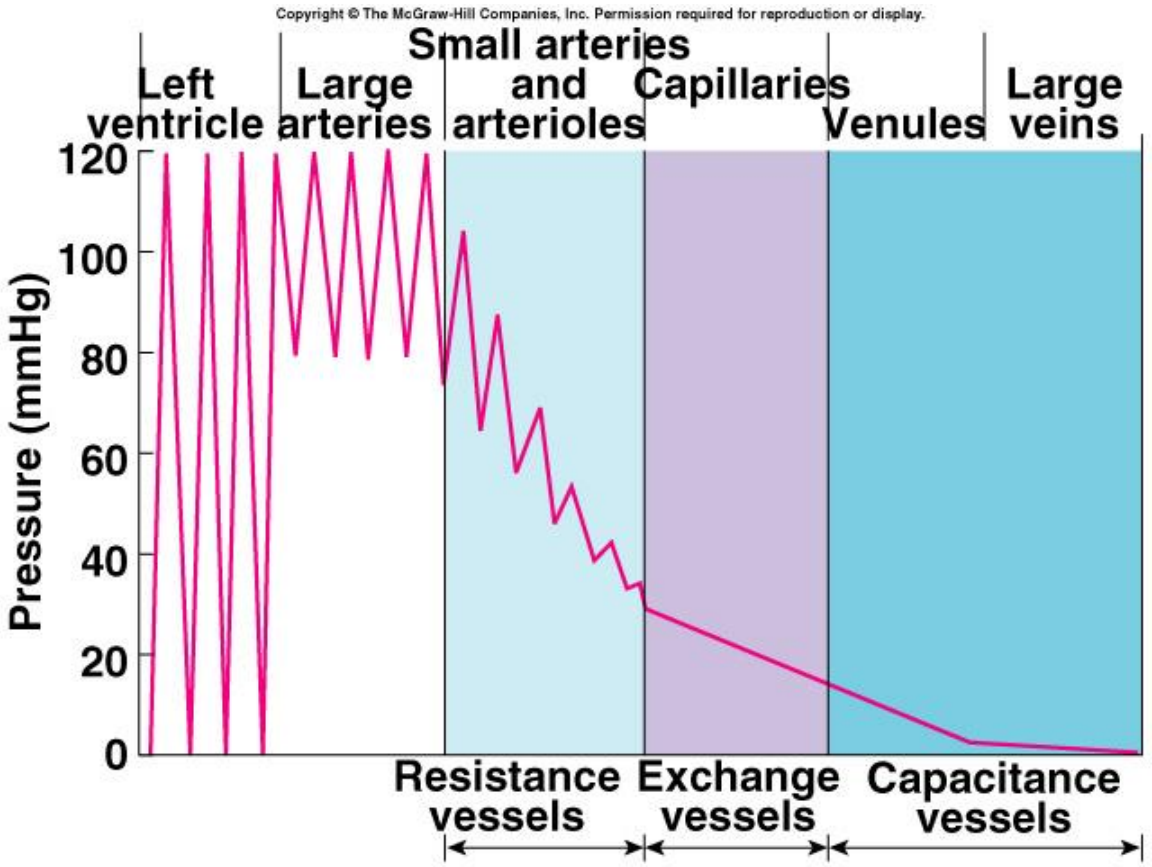
- Local Blood flow
- Blood pressure – measure, regulation
- Baroreceptor reflex
- Orthostatic hypotension (3 minutes)
- Circulatory shock
- Heart failure

Vascular Resistance to Blood Flow

- Physical laws describing blood flow:
  - The flow of blood through the vascular system is due to the difference in pressure at the two ends (ΔP).
- Flow = $\Delta P/R$
  - R = TPR (sum of all vascular resistance within the systemic circulation).
    - Blood flow directly proportional to pressure differences.
    - Inversely proportional to resistance.

Resistance

- Opposition to blood flow.
- Resistance is directly proportional to length of vessel and to blood viscosity.
- Inversely proportional to 4th power of the radius of the vessel.
- $R = \frac{L\eta}{r^4}$
  - L = length of the vessel
  - η = viscosity of blood-eta
  - r = radius of the vessel
    - Vessel length and blood viscosity do not vary significantly.



- Major regulators of blood flow through an organ are:
  - Mean arterial pressure.
  - Vascular resistance to flow

Extrinsic Regulation of Blood Flow

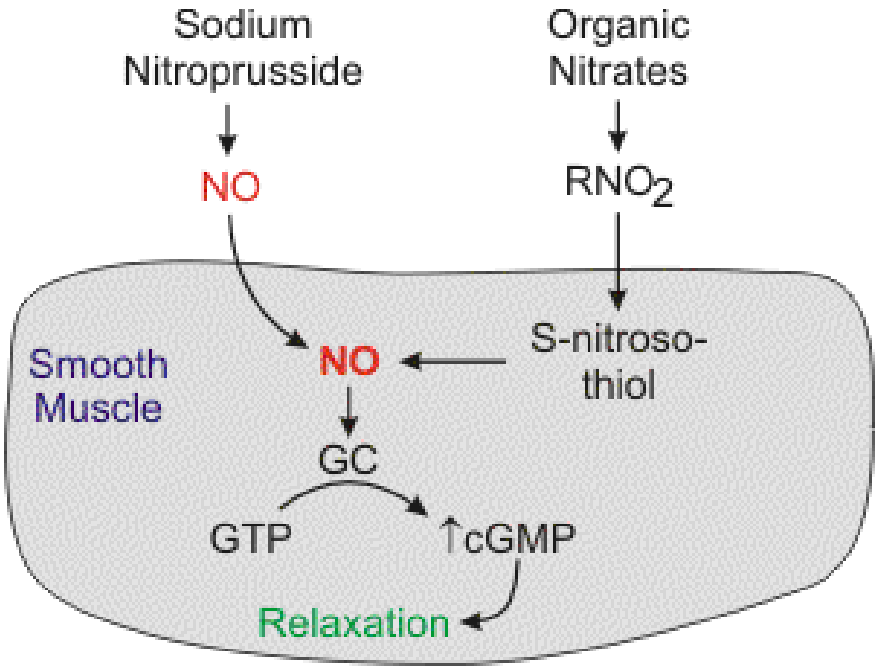
- Sympathoadrenal:
  - Increase CO.
  - Increase TPR:
    - Alpha-adrenergic stimulation:(**usually**)
      - **Vasoconstriction** of arteries in skin and viscera.
    - Cholinergic sympathetic fibers:(**exception**)
      - Vasodilate to skeletal muscles.(more blood = energy to muscles)
- Parasympathetic nervous system:
  - Innervation is limited.
    - Promotes vasodilation to the digestive tract ,external genitalia, and salivary glands.(more blood = energy to digest + absorb food)
  - Less important than sympathetic nervous system in control of TPR.
    - Parasympathetic endings in arterioles promote **vasodilation**.

Paracrine Regulation of Blood Flow

- **Endothelium produces several paracrine regulators:**
  - **Endothelium of arterioles contains eNOS-enzyme, which produces NO: Nitric oxide synthase**
    - **NO diffuses into smooth muscle**
      - **Activates guanylate cyclase:**
        - **Converts GTP to cGMP (2nd messenger).**
        - **Lowers cytoplasmic $[Ca^{2+}]$ = vasodilation**
        - **general effect of calcium is to stimulate smooth muscle contraction, so if no calcium= dilatation**

        Used in Vascular remodeling, angiogenesis

- **Production of NO can be increased indirectly by ACh:**
  - **Stimulates opening of Ca^{2+} channels.**
    - **Ca^{2+} binds to calmodulin.**
    - **Calmodulin complex activates an enzyme to produce NO.**
    - **In response to rise in ca, NOS enzyme is released**



Intrinsic Regulation of Blood Flow (Autoregulation)

- **1) Myogenic control mechanism:**
  - Occurs because of the stretch of the vascular smooth muscle (inherent property in response to stretch)
    - A increase in systemic arterial pressure causes cerebral vessels to contract
      - Maintains adequate flow.
      - Important in preventing excessive stretch of blood vessel when blood pressure is increased.

      Also in kidneys -tubuloglomerular feedback, macula densa juxtaglomerular apparatus
    - A **decrease** in systemic arterial pressure causes **cerebral vessels** to **relax(dilate)** = stress relaxation effect- Maintains adequate flow.

- **2) Metabolic control mechanism:**
  - Intrinsic receptors sense chemical changes in environment due to metabolism.
  - Vasodilation:
    - **Decreased 0_2:**
      - **Due to Increased metabolic rate.**
    - **Increased $C0_2$:**
      - **Due to Decreased ventilation.**
    - **Decreased pH:**
      - **Rise in Lactic acid.**
    - **Increased adenosine or increased K^+, H +:**
      - **From tissue cells.**

      Histamine- vasodilator

Aerobic Requirements of the Heart

- **Survival requires that the heart and brain receive adequate blood supply uninterrupted at all times.**
  - **Coronary arteries supply an enormous # of capillaries.**
    - **Each myocardial cell is within 10 μm of a capillary distance.**
  - **Systole contracts the coronary blood vessels.**
  - **Diastole increases blood flow to the heart muscle.**

Hypoxia- ischemia- ATP degrades – Adenosine released- vasodilator of coronary arteries

- **Ischemia > 30 min- infarction(dead cardiac cells- irreversible)**

- **Myocardium contains large amounts of myoglobin.**
  - **Myoglobin stores 0_2 during diastole to release during systole.**
    - **Heart muscle contains increased number of mitochondria and aerobic respiratory enzymes.**

Examples of mediators

- Vasoconstrictors- Norepinephrine and Epinephrine, Angiotensin II, *Vasopressin*, also called *antidiuretic hormone*, Endothelin, thromboxane A2- platelets, serotonin – platelets, leukotrienes
- Vasodilator Agents – Bradykinin, Histamine from *mast cells* in the damaged tissues and from *basophils* in the blood.
- It is deficiency of tissue oxygen or other nutrients, or both, that leads to formation of the vascular growth factors (also called "angiogenic factors").
- Collateral circulation- When an artery or a vein is blocked in any tissue of the body, a new vascular channel develops around the blockage - allows partial resupply of blood to the affected tissue – long term.

1.Regulation of Blood Flow for Skeletal Muscles

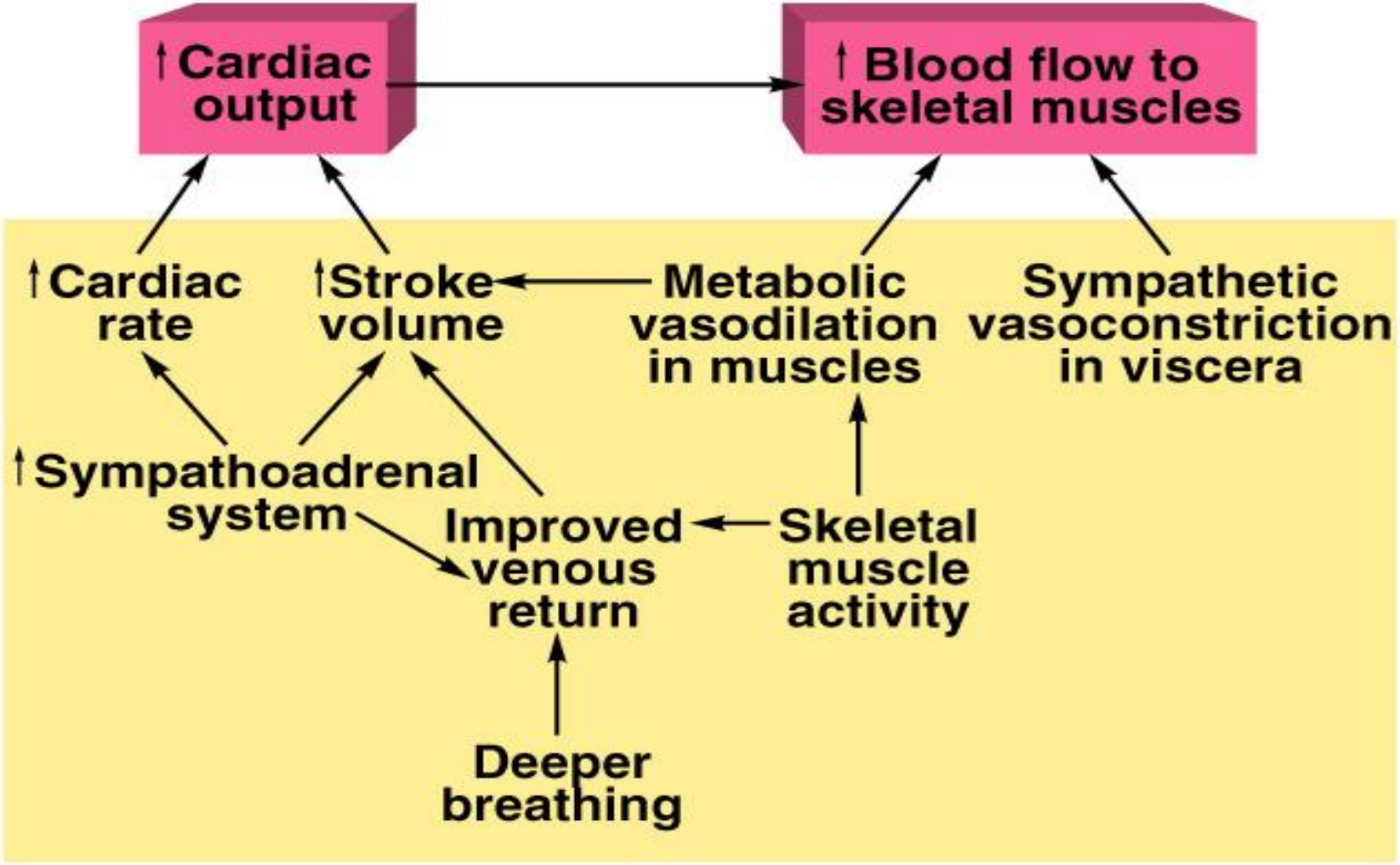
- **Decreased blood flow when muscles contract and constrict arterioles**

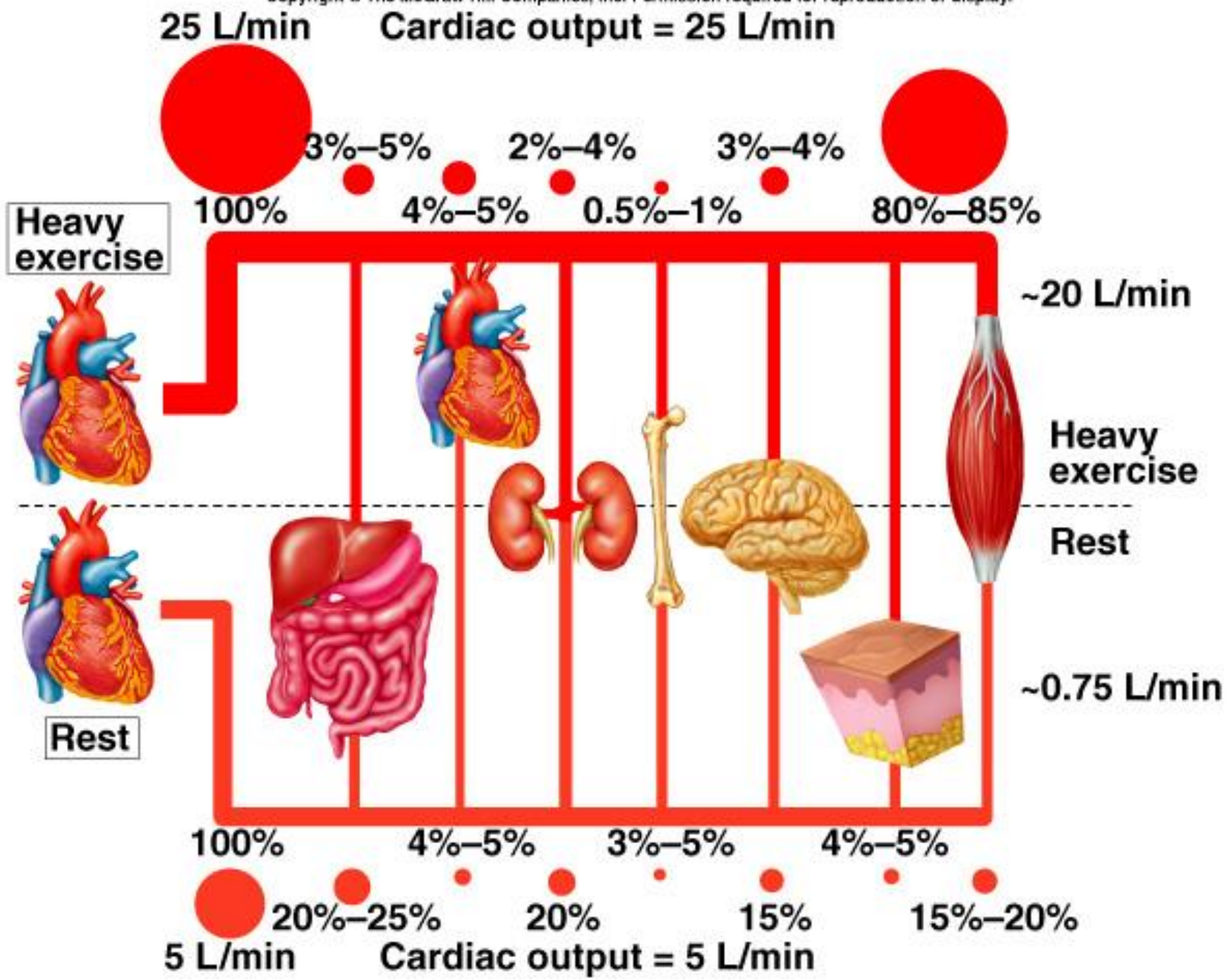
- **Sympathetic:**
  - **α–adrenergic receptors:**
    - **Vasoconstrict at rest.**
- **Sympathetic cholinergic and β–adrenergic receptors:**
  - **Vasodilate.**
- **Intrinsic control are primary mechanisms.**

- As exercise progresses, following changes occur:
  - At beginning, heart – sym stimulated - supply the increased blood flow required by the muscles, - same time blood flow through most non muscular areas of the body is temporarily reduced
  - muscle walls of the *veins (in muscles) and other capacitative areas of the circulation are contracted powerfully* - increase in venous return of blood to the heart - increasing the cardiac output.
  - Increased accumulations of $C0_2$, K^+, and adenosine; decreased 0_2. = Vasodilate arterioles and precapillary spinctors in skeletal muscles.

Circulatory Changes During Exercise

- Vascular resistance decreases to skeletal muscles.
  - Blood flow to skeletal muscles increases.
- SV and CO increase.
  - Blood flow to brain, kidney stays same.
  - Metabolic vasodilation in skel muscles
  - Diversion of blood away from vicera and skin towards muscles
- HR increases to maximum of 190 beats/min.
  - ❑Ejection fraction increases due to increased contractility.
- Vascular resistance:
  - Decreases to skeletal muscle.
  - Increases to GI tract and skin.

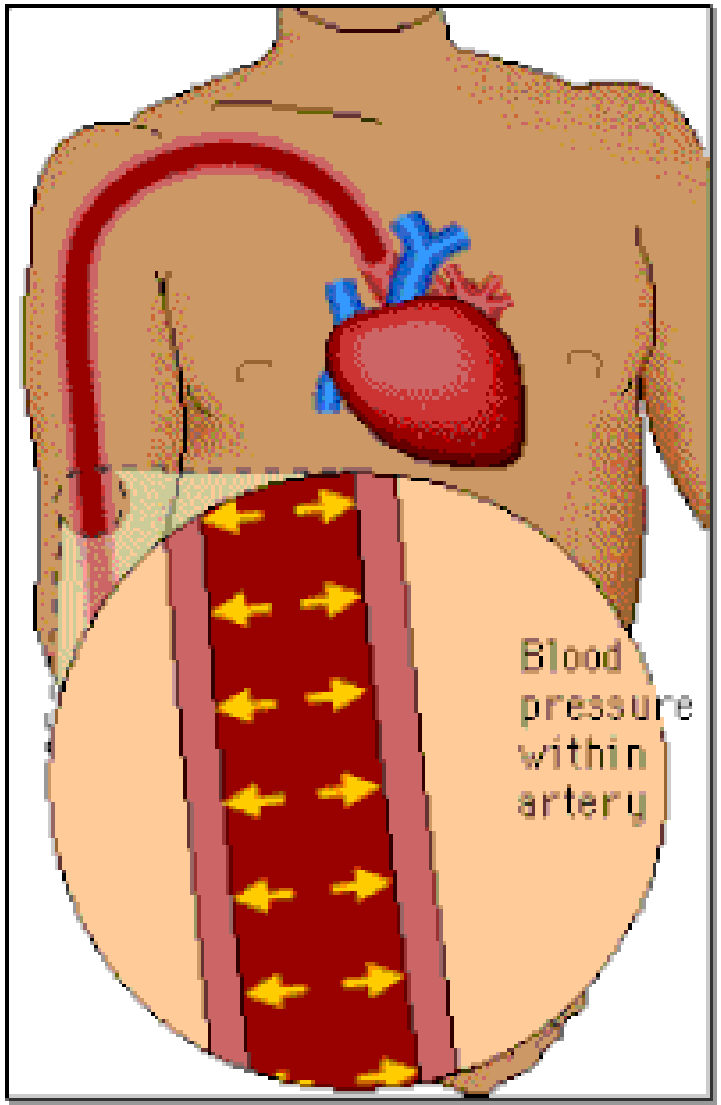




2. Cerebral Circulation

- Cerebral blood flow is not normally influenced by sympathetic nerve activity.
- Normal range of arterial pressures:
  - Cerebral blood flow regulated exclusively by intrinsic mechanisms:
    - **Myogenic**:
      - Dilate in response to decreased blood pressure.
      - Cerebral arteries also sensitive to $[C0_2]$.
        - » Dilate due to decreased pH (rised H +)of cerebrospinal fluid.
    - **Metabolic**:
      - Sensitive to changes in metabolic activity.
        - » Areas of brain with high metabolic activity receive most blood.
        - » May be caused by $[K^+]$.

Blood pressure



- **DEFINITION**:-The pressure exerted by circulating blood upon the walls of blood vessels,

- Blood pressures are usually categorised into three groups:-

1. Low (**90/60 mmHg** or lower),

2. Normal (values above **90/60** mmHg and below **130/80mmHg**), and

3. High (**140/90mmHg** or higher).

Function of Blood Pressure:

- **Provides pressure head for blood flow and exchange of materials across capillary endothelium.**

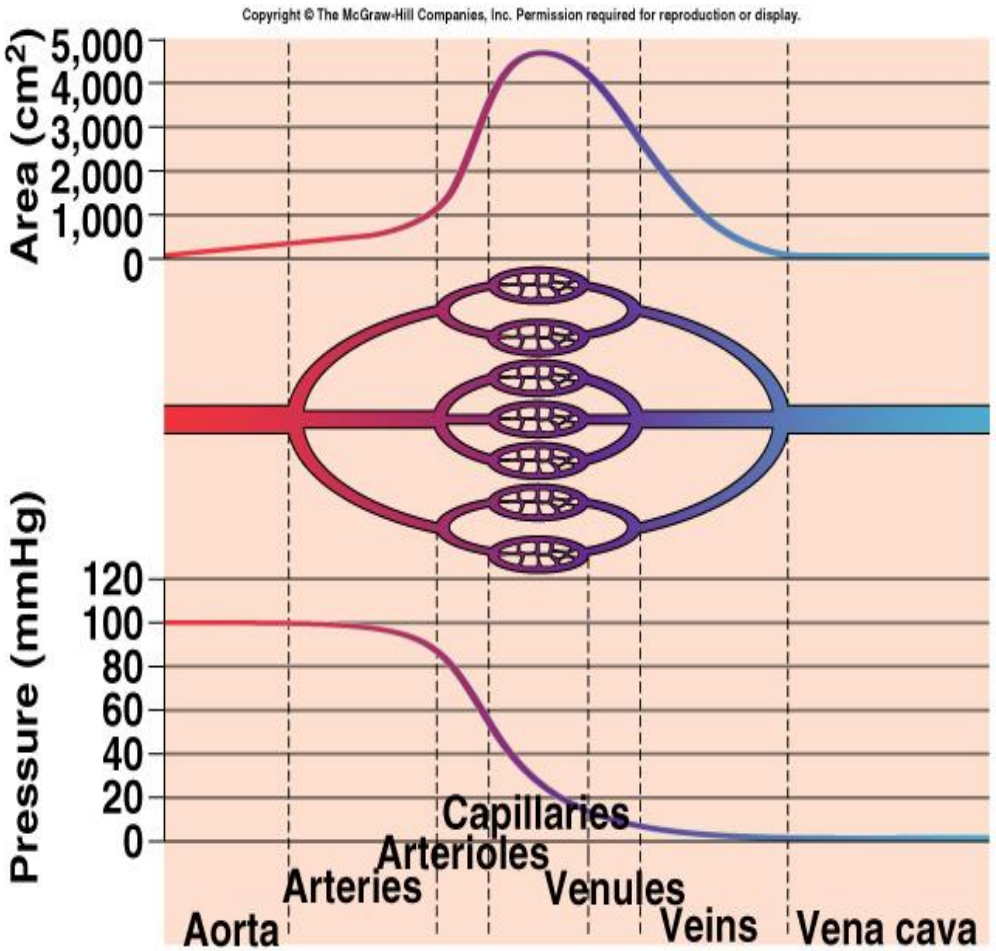
- **Arterial Blood Pressure is usually measured in mm Hg**

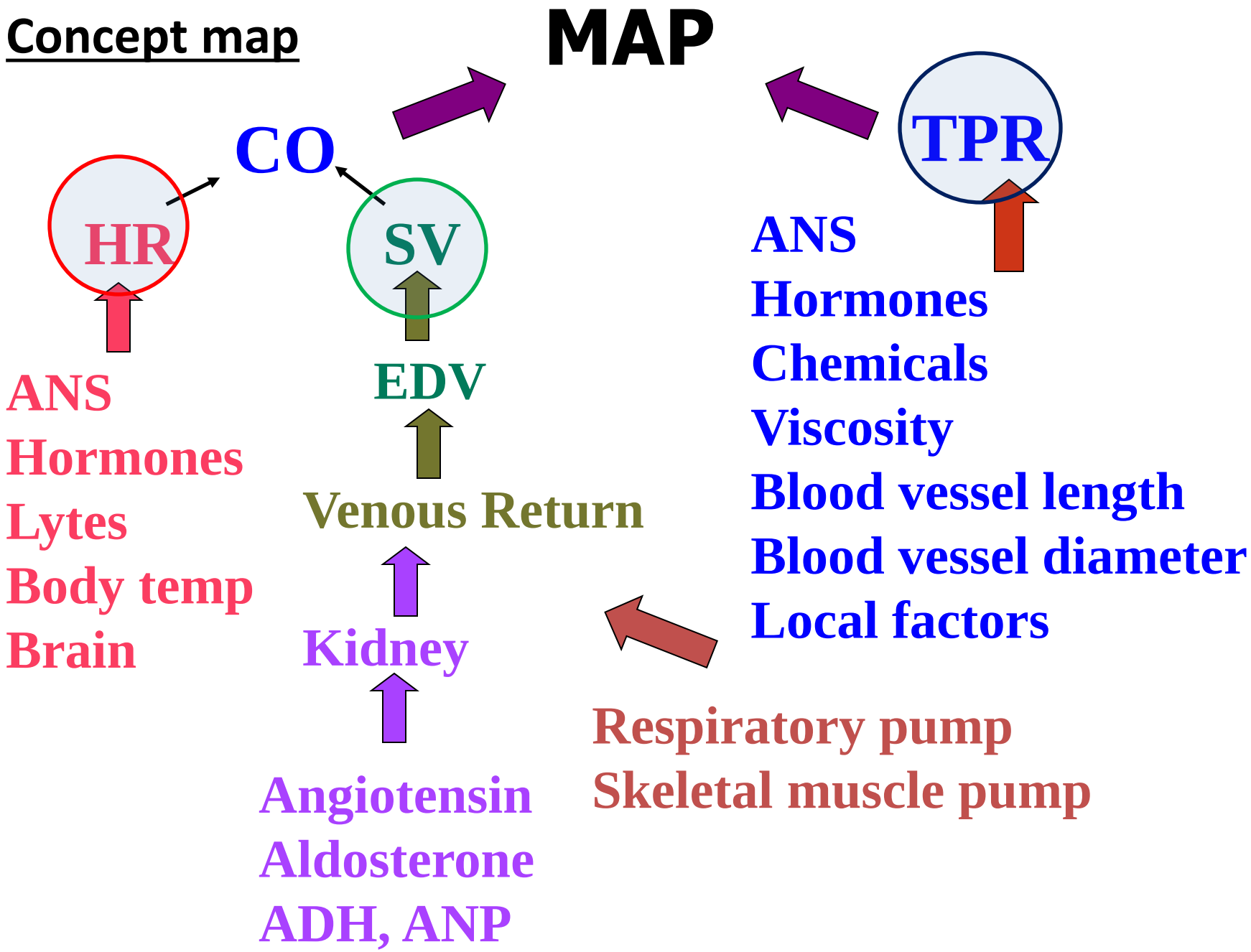
- **Systolic Blood Pressure (SBP): Maximum arterial blood pressure during heart contraction (i.e. systole). Average: 90 – 130 mm Hg in humans.**

- **Diastolic Blood Pressure (DBP): Minimum arterial blood pressure during heart relaxation (i.e. diastole). Average: 60 – 80 mm Hg in humans.**

Blood Pressure (BP)

- Pressure of arterial blood is regulated by blood volume, TPR, and cardiac rate.
  - MAP=CO × TPR
- Arteriole resistance is greatest because they have the smallest diameter.
- Capillary BP is reduced because of the higher total cross-sectional area.
- 3 most important variables are HR, SV, and TPR.
  - Increase in each of these will result in an increase in BP.
- BP can be regulated by:
  - Kidney and sympathoadrenal system.




Concept map
MAP
CO
HR
SV
TPR
ANS
Hormones
Chemicals
Viscosity
Blood vessel length
Blood vessel diameter
Local factors
ANS
Hormones
Lytes
Body temp
Brain
EDV
Venous Return
Kidney
Angiotensin
Aldosterone
ADH, ANP
Respiratory pump
Skeletal muscle pump

Regulation of BP:

- **Neural- rapid control**

1. Baro receptor

2. Chemo receptor

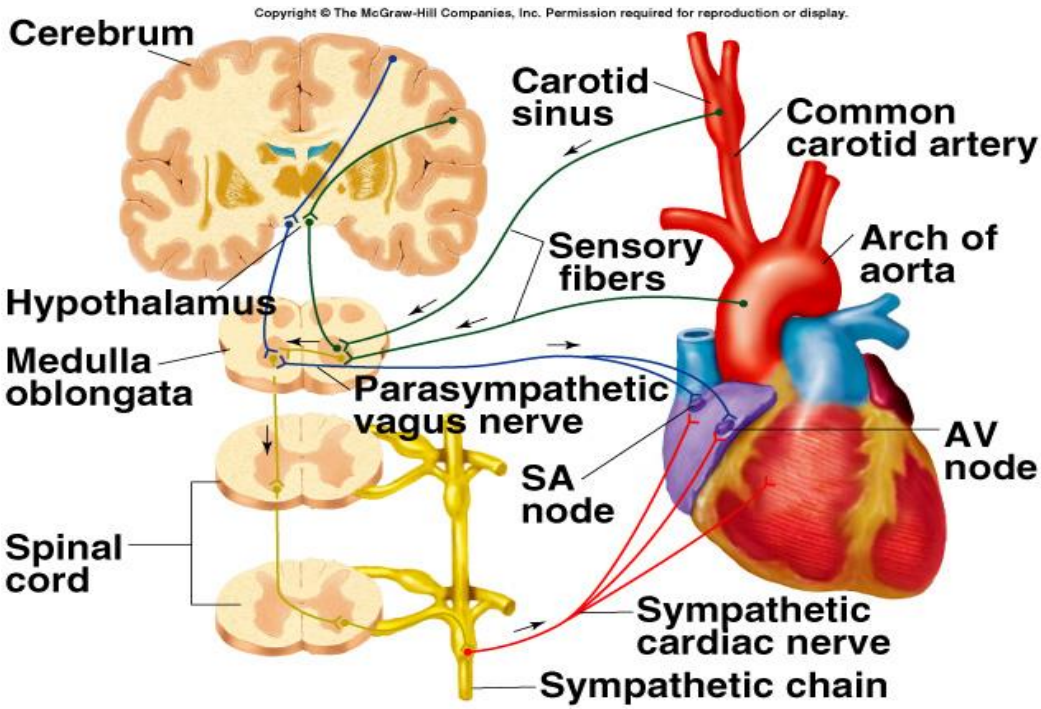
3. CNS ischemic response

- **Hormonal- slow**

1. **1. Nor adrenaline-adrenaline system**
2. **2. Renin angiotensin aldosterone system**
3. **3. Vasopressin system**

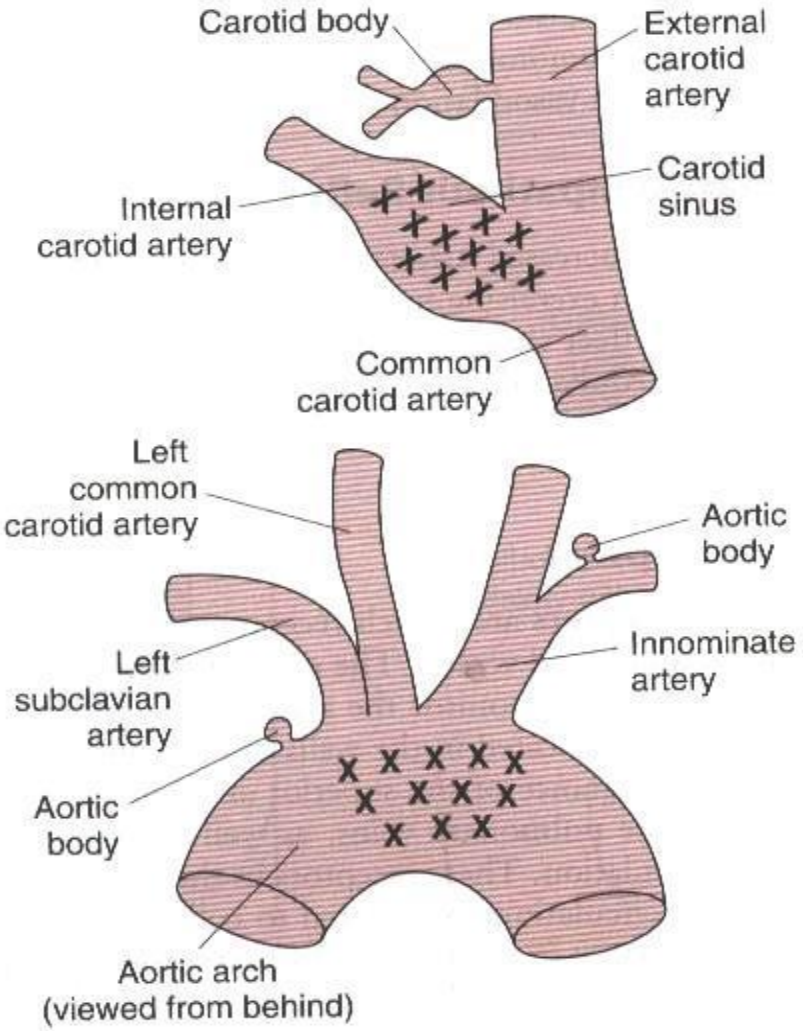
1. Baroreceptor Reflex

- **An increase in pressure causes the receptors (aortic arch-X nerve and carotid sinuses- IX nerve) to stretch, increasing frequency of Aps through- buffer nerves.**
- **Baroreceptors send APs to vasomotor center and cardiac vagal control centers in the medulla.**
- **Baroreceptor reflex activated with changes(fall) in BP(40 mm Hg to 160mm Hg)= working range.**
- **More sensitive(60- 120 mm) to decrease in pressure and sudden changes in pressure- help in BP homeostasis**



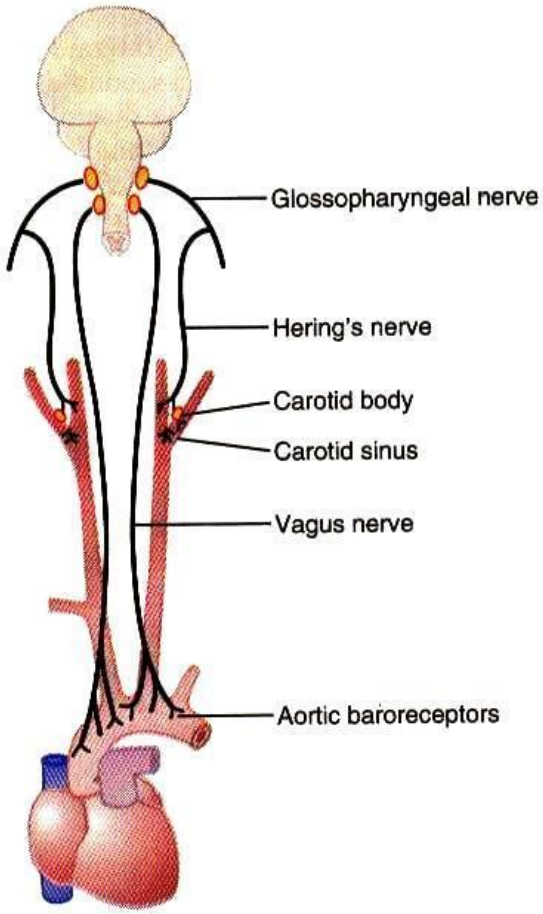
Follow the arrows

Buffer nerves



Baroreceptor areas in the carotid sinus and aortic arch. X, sites where receptors are located.

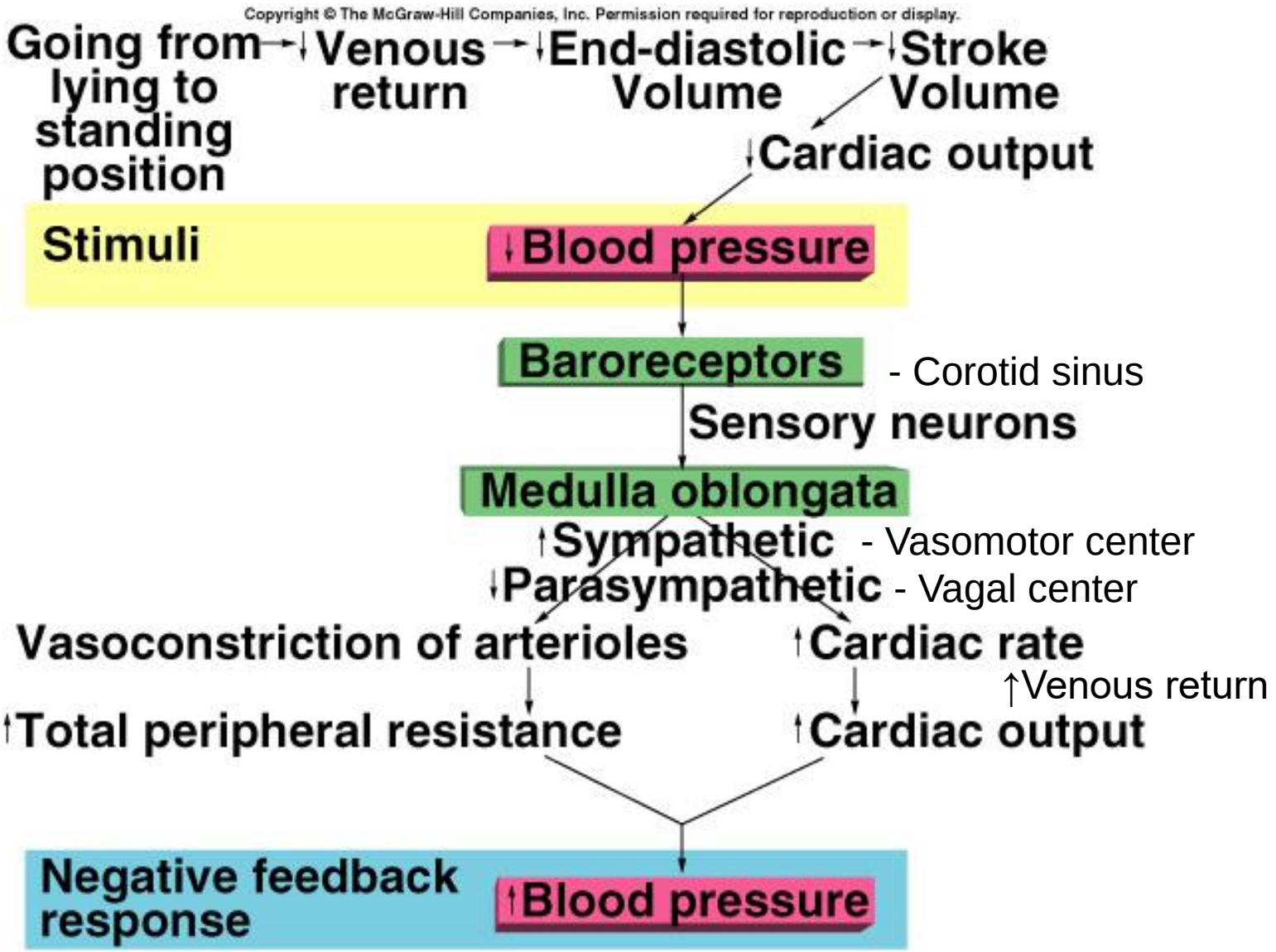
Ganong textbook



The baroreceptor system for controlling arterial pressure.

Pal & Pal textbook

Baroreceptor Reflex



2. Chemo receptor reflex- ↓ in BP 40-80 mm Hg

Hypotension, hypercapnia, acidosis in blood - stimulate - Carotid body, aortic body- excite VMC- rise in BP + resp rate

3. CNS ischemic response -

BP < 50 mm – ischemia, low blood flow to **VMC-** excites intense discharge to **sym nerves**- good vaso constriction- rise in BP

(Because neurons **very sensitive to hypoxia**)

Cushing's reflex- If the above response is activated in response to rise in intracranial pressure eg- intra cranial hemorrhage due to head injury

Atrial Stretch Reflexes

- Respond to rise in BP
- Located in the atria of the heart.
- Receptors activated by increased venous return.
    - Stimulate reflex tachycardia.
    - Inhibit ADH release= causes diuresis
    - Promote secretion of ANP= causes natriuresis
    - So loss of sodium = water follows = fall in blood volume and pressure (Osmosis)

Other regulation

- Bradykinin – VD decreases BP
- Histamine - VD
- Endothelins - VC
- Catecholamines - VD
- Atrial stretch reflex – increased venous return – type B receptors – VD – fall in BP

Conversely if increased blood volume – opp effect

Bain bridge reflex – infuse saline/blood in to veins = tachycardia – TPAR – atrial wall

Vascular mechanisms of BP

- Capillary fluid shift- shift of fluid occurs between interstitial tissue space and capillary lumen depending on "where" the reduction in hydrostatic pressure happens.

- Stress relaxation – acute stretch on vessel wall, Smooth muscle relaxes.
- Reverse stress relaxation – fall in BP – causes contraction of smooth muscles in vessels

- **Myogenic theory** applies to blood flow

BP measurement

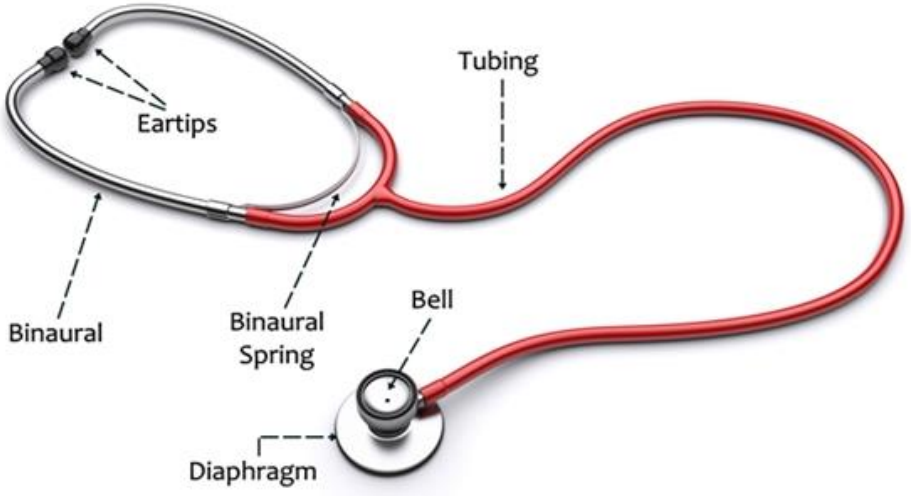
- Direct method to measure BP-
- Artery blood – anti coagulant in cannula- manometer
- Records only end pressure
- Animals - experimental

Indirect method to measure BP

- **Auscultation:**
  - **Art of listening with a stethoscope - invented by ?**
- **Laminar flow:**
  - **Normal blood flow, lamina over lamina**
  - **Blood in the central axial stream moves faster than blood flowing closer to the artery wall.**
    - **Smooth and silent.**
- **Turbulent flow and vibrations in flow are produced in the artery when cuff pressure is greater than diastolic pressure and lower than systolic pressure.**

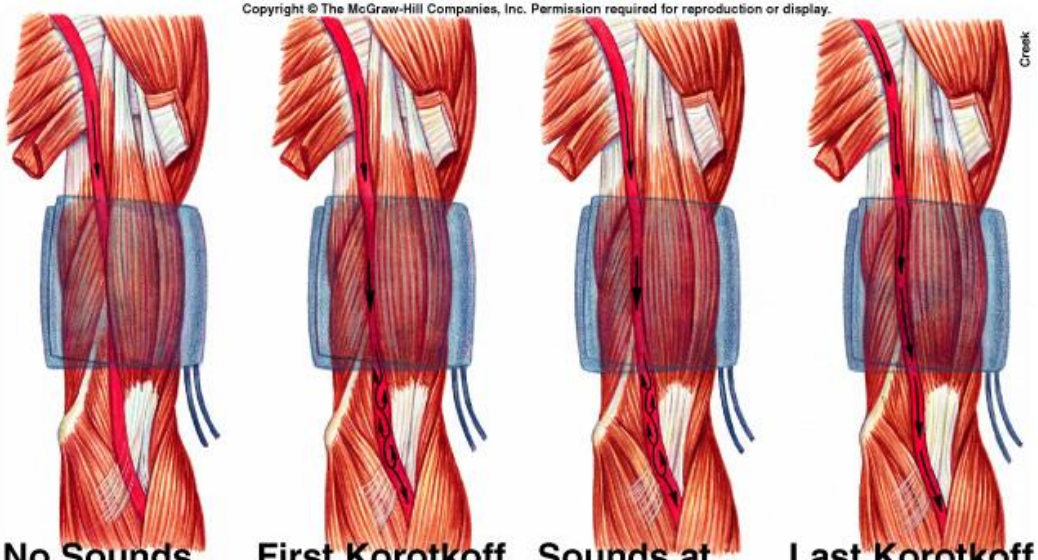
- **Eg-Block (cuff pressure) and slowly release bit by bit – initially turbulent – later calms down to normal laminar flow when fully open**

Stethoscope



Mercury sphygmomanometer

- Sphygmomanometer- cuff, Hg reservoir, 1 way air flow hand pump, vertical scale of 0- 300 mm Hg
- Blood pressure cuff is inflated above systolic pressure(140 mm Hg), occluding the brachial artery (tied) above cubital fossa.
- As cuff pressure is lowered (2 mm Hg per sec), the blood will flow only when systolic pressure is above cuff pressure, producing the sounds of Korotkoff (5 Phases) under lying principle
- Korotkoff sounds will be heard until cuff pressure equals diastolic pressure, causing the sounds to disappear later after DBP.



No Sounds

Cuff pressure = 140

First Korotkoff sounds

Cuff pressure = 120

Systolic pressure = 120 mmHg

Sounds at every systole

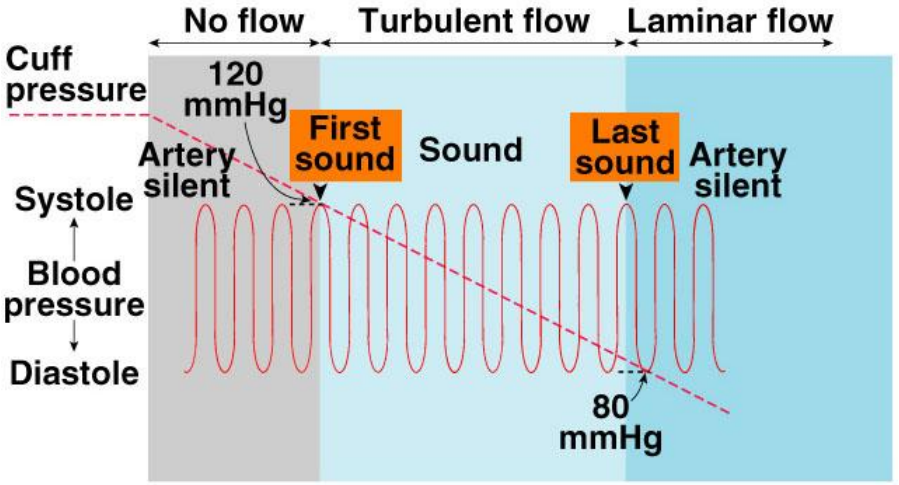
Cuff pressure = 100

Last Korotkoff sounds

Cuff pressure = 80

Diastolic pressure = 80 mmHg

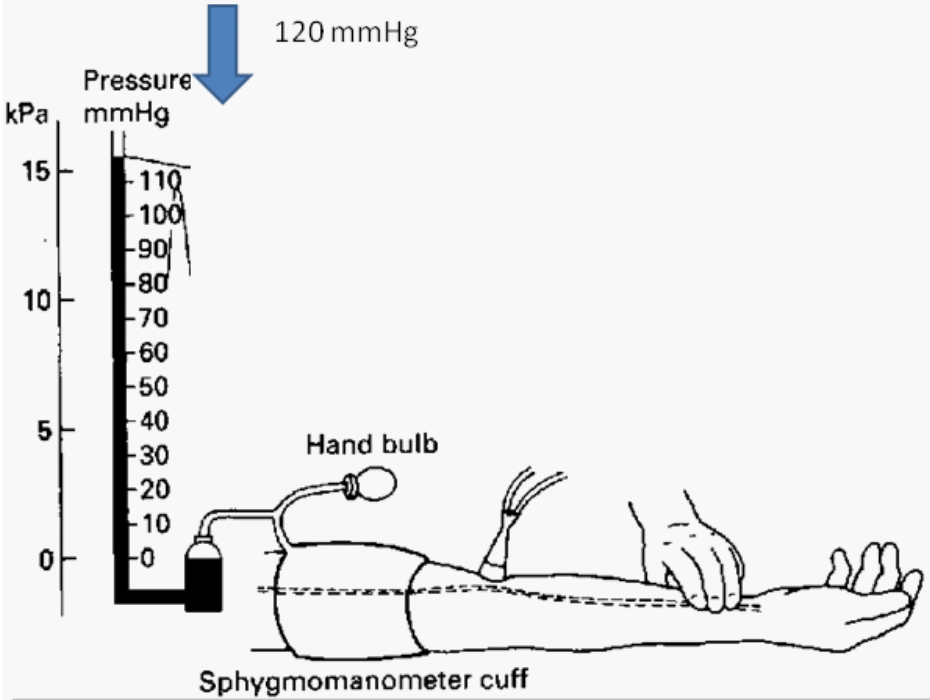
Blood pressure = 120/80



Measuring BP at a medical camp

Try palpating the diastolic blood pressure

It is not unscientific !



Tactile impulses can be more sensitive , further it is immune from ambient noise contamination !

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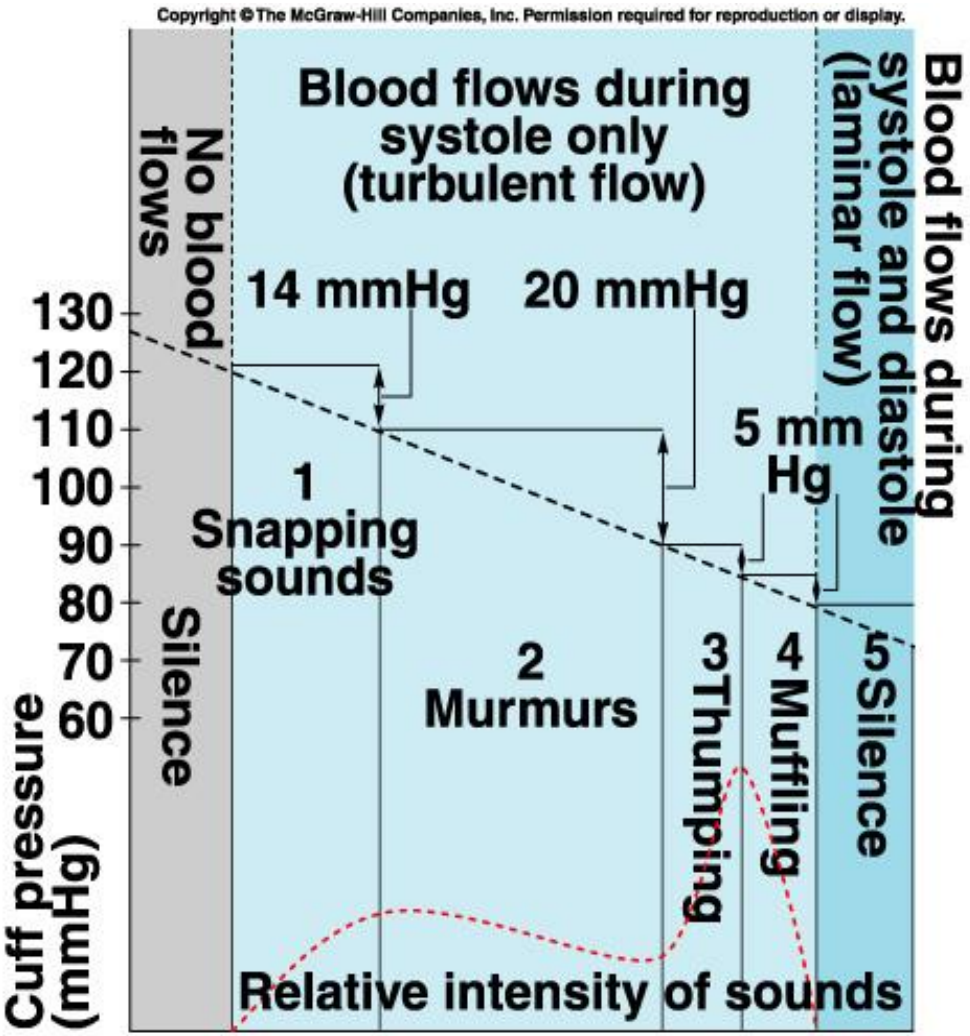
Colour white = empty, black = blood flowing

- Different <u>phases(5)</u> in measurement of blood pressure are identified on the basis of the <u>quality</u> of the <u>Korotkoff sounds</u>.
- Average arterial BP is <u>120/80 mm Hg.</u>
- [Average pulmonary BP is 22 to 8 mm Hg]
- Sounds appear at SBP=120 mm
- Disappear at DBP=80 mm
- 5 phases of sounds



- SBP- highest pressure recorded when heart is contracted- systole
- DBP- lowest pressure – heart – dilated- diastole
- Pulse Pressure- Denotes the expansion of the artery in response to the volume of blood ejected by the left ventricle.
- Pulse pressure = systolic pressure – diastolic pressure
- Mean arterial pressure (MAP):
  - Represents the average arterial pressure during the cardiac cycle.
    - Is closer to diastolic pressure, as the period of diastole is longer than the period of systole.
- MAP = diastolic pressure + 1/3 pulse pressure
- Hypotension - BP less than 90/60 mm of Hg


Questions
?

Hypertension (HTN)

- Blood pressure in excess of normal range for age and gender (115/75).

  >120/80 mm Hg now considered pre-hypertension.

  >140/90 mm Hg is high BP

- 1. Primary or essential hypertension-(idiopathic)
  - Is the result of a complex or poorly understood process- unknown cause
- 2. Secondary hypertension:
  - Is a result of a known disease process- renal Cx, renal failure, liver cirrhosis, COPD, adrenal gland diseases, thyroid and para thyroid disorders

  BP increases with more age

Hazards of Hypertension

- Silent killer: many people have high blood pressure for years without being aware of it
- Patients are asymptomatic until substantial vascular damage occurs.
  - Atherosclerosis.
- Increases afterload.
  - Increases workload of the heart.
  - Congestive heart failure.
- Damage cerebral blood vessels.
  - Cerebral vascular accident (**brain stroke** or TIA) no blood supply to brain parts.
  - There are two major types, **embolic or ischemic stroke** and **haemorrhagic strokes**.

Physiological basis of treating of Hypertension

- Modification of lifestyle:
  - Regular exercise- aerobics, swimming
  - Cessation of smoking.
  - Moderation in alcohol intake.
  - Weight reduction (BMI).
  - Reduction in Na^+ intake.
  - Diet high in K^+ like tomatoes, spinach

- Medications:
  - Diuretics (lasix) :
    - Increase water loss in urine
  - Beta-blockers:
    - Decrease HR.
  - Calcium antagonists:
    - Block Ca^{2+} channels – no smooth mus contraction
  - ACE inhibitors:
    - Inhibit conversion to angiotensin II.
  - Angiotension II-receptor antagonists:
    - Block receptors.

Orthostasis

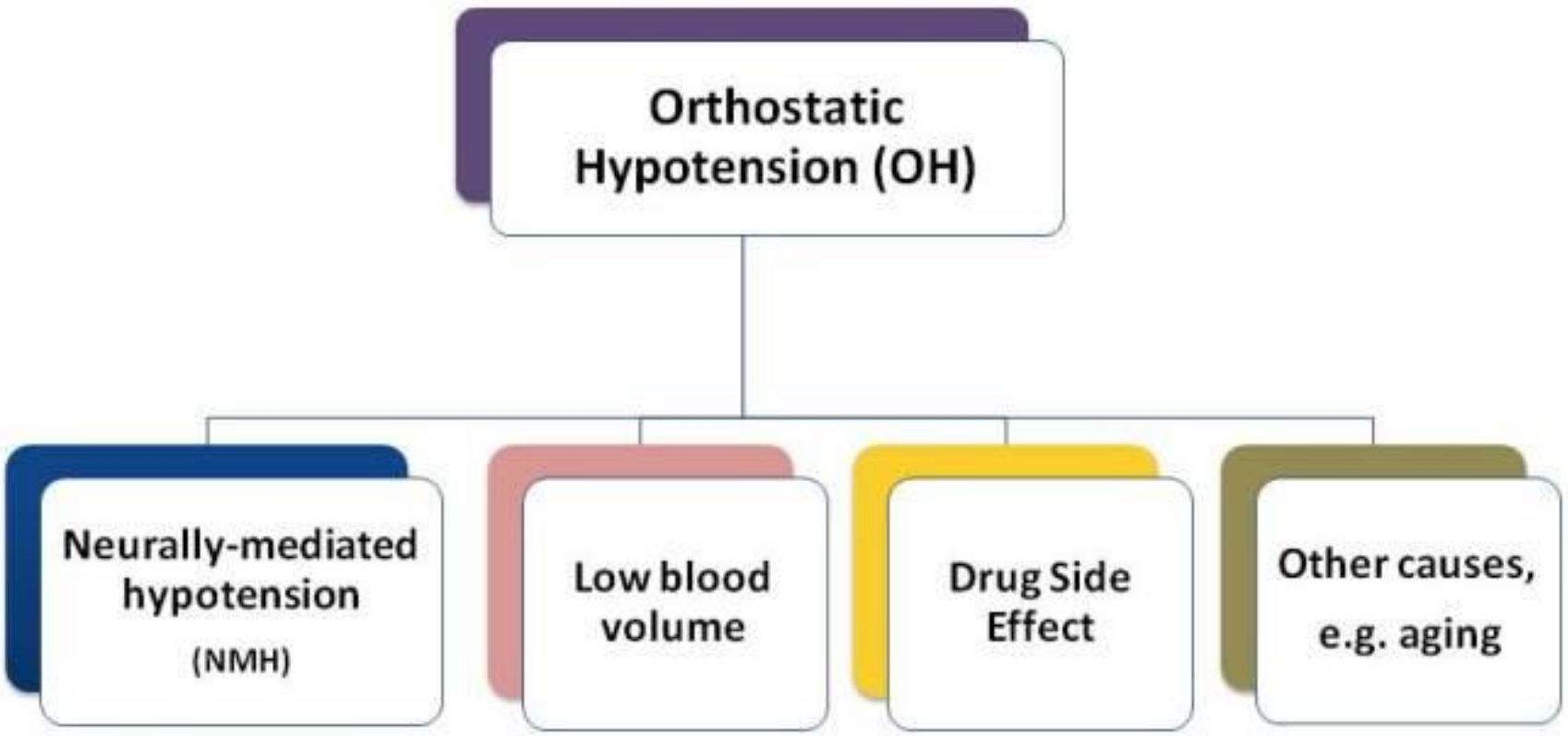
- In standing position, usually the venous pooling of 300-500ml of blood - in lower extremities - results in decreased venous return - cardiac output - blood pressure is - reduced. A drop in the BP in carotid sinus and aortic arch within seconds triggers the baroreceptor mediated compensatory reflex mechanism which causes following changes:
- Increase in heart rate (chronotropic effect)
- Increase in force of cardiac contraction (ionotropic effect).
- Increase in peripheral resistance (VMC stimulated)

Definition of Orthostatic Hypotension :

- The consensus committee of the **American Autonomic Society** and the **American Academy of Neurology** defined the orthostatic hypotension as a [*systolic blood pressure decrease 20mmHg* ***or*** *a diastolic10mmHg*] **with in three minutes** of standing up.
- Measurement- in standing position- measure BP at heart level (arm)
- Hazards- postural syncope or falls
- Treatment- vasopressor drugs, drink more water

OH-standing BP

Hypotension



Circulatory Shock = low blood flow to organs

Types

I. Cardiogenic shock (due to heart problems)
II. Hypovolemic shock (caused by too little blood volume)
III. Anaphylactic shock (caused by allergic reaction)
IV. Septic shock (due to infections)
V. Neurogenic shock (caused by damage to the nervous system)

Stages of shock

Characteristics of circulatory shock change with different degrees of severity, shock is divided into the following three major stages:

1. nonprogressive stage (*compensated stage*), in which the normal circulatory compensatory mechanisms eventually cause full recovery **without help from outside therapy.**

2. progressive stage – ***recovers with*** **therapy,** the shock becomes steadily worse until death.

3. irreversible stage - has progressed - all forms of known therapy are inadequate to save the person's life- for the moment, the person is still alive – **going to die tomorrow**

A) Hypovolemic shock:

- Circulatory shock that is due to low blood volume.
- Decreased CO and blood pressure.
  - Bleeding, dehydration, trauma and burns.

- **Compensation mechanisms (physiological):**
  1. Baroreceptor reflex:
     - Tachycardia.
     - Vasoconstriction to GI, skin, kidneys, and muscles.
  2. Kidneys stimulate production of renin-angiotensin-aldosterone system.
     - Vasoconstriction.
  3. Increase in ADH.

B) Septic shock: Dangerously low blood pressure as a result of sepsis.

- **– Occurs through the action of endotoxin.**
  - **Endotoxin activates nitric oxide synthase, producing excess NO.**
  - **NO causes excessive vasodilation = fall in BP.**
- **Treat with drugs that inhibit the production of NO.**

Other Causes of Circulatory Shock

- **Anaphylactic shock:**
  - **Severe allergic reaction.**
  - **Widespread release of histamine.**
  - **Vasodilation.**
  - **Skin infections spreading, peritonitis, kidney infections ascending up**
- **Neurogenic shock:**
  - **Rapid fall in BP.**
    - **Sympathetic tone is decreased (VMC disorder – brain damage).**
    - **Deep general anesthesia,**
- **Cardiogenic shock:**
  - **Cardiac failure (RVH, LVH).**
    - **Cardiac Output inadequate to maintain perfusion.**

Congestive cardiac Failure

- **Cardiac Output is insufficient to maintain the blood flow required by the body- (unable to pump effectively)**
  - **Increased venous volume and venous pressure.**
- **Caused by:**
  - **MI (most common cause).**
  - **Congenital heart defects.**
  - **Hypertension.**
  - **Aortic valve stenosis (small openings).**
  - **Disturbances in electrolyte concentrations.**
    - **↓ K^+ and Ca^{++}.**
- **Initially Body compensations similar to those of hypovolemic shock.**
- **Later stages- decompensated stage occurs**

Types

- Right heart F
- Left heart F
- High output F
- Low output F
- Forward F
- Backward F……..
- **Treated with medications:**
  - **Digitalis, vasodilators, and diuretics.**
  - **Digitalis action - strengthen heart contraction by increasing the quantity of calcium ions released inside muscle fibers – depresses calcium pump in the cell membranes of cardiac muscle = no calcium going out, more cal++ inside**

Ischemic Heart Disease or CAD

Most common cause of death in the West and Malaysia

Causes- *coronary ischemia - coronary occlusion - atherosclerosis*

Small amounts of collateral flow helps – later *myocardial infarction*.

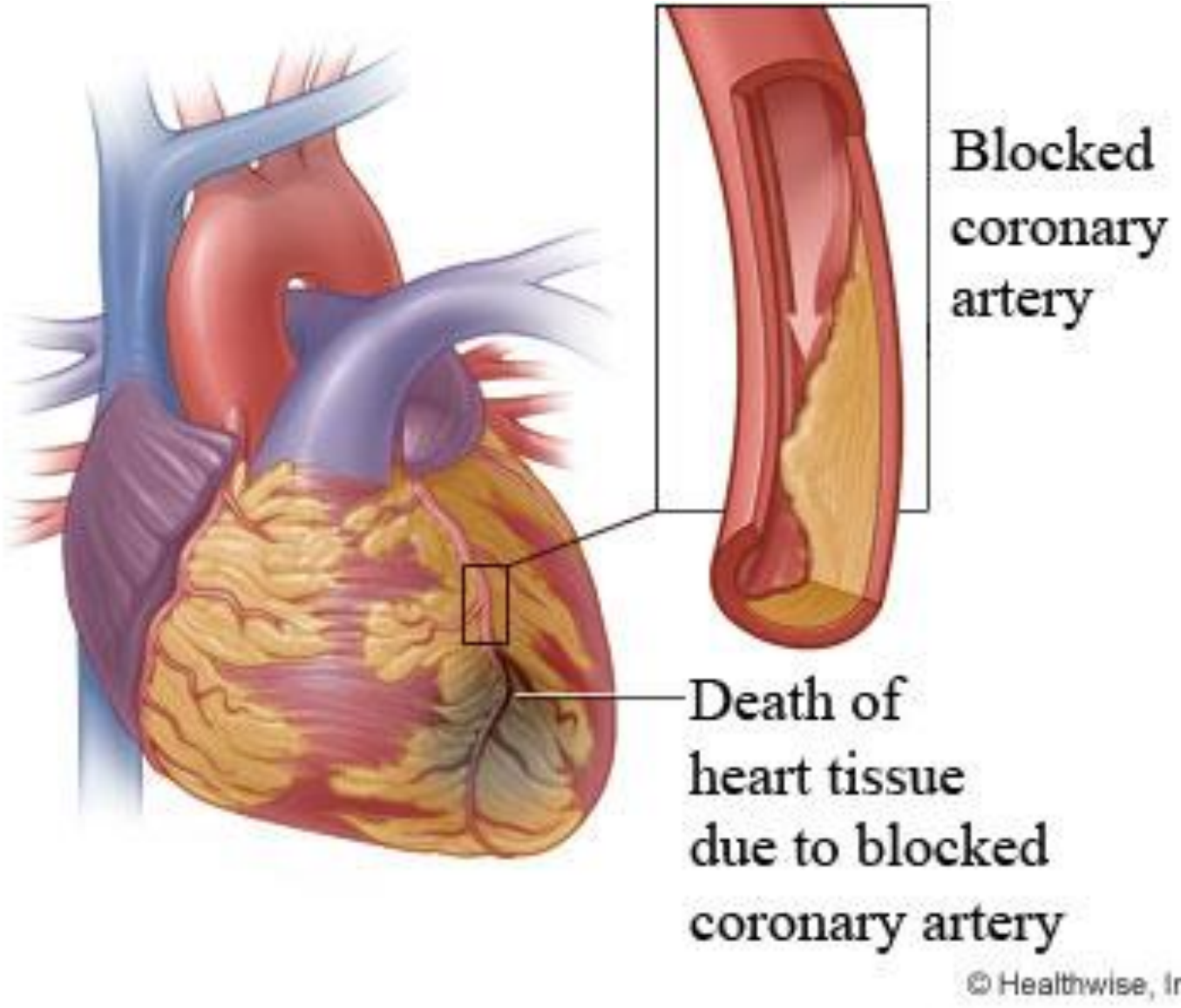
cardiac pain - called *angina pectoris*- felt beneath the upper sternum – referred to distant surface areas of the body- to the *left arm* and *left shoulder* - to the *neck* and *side of the face*.

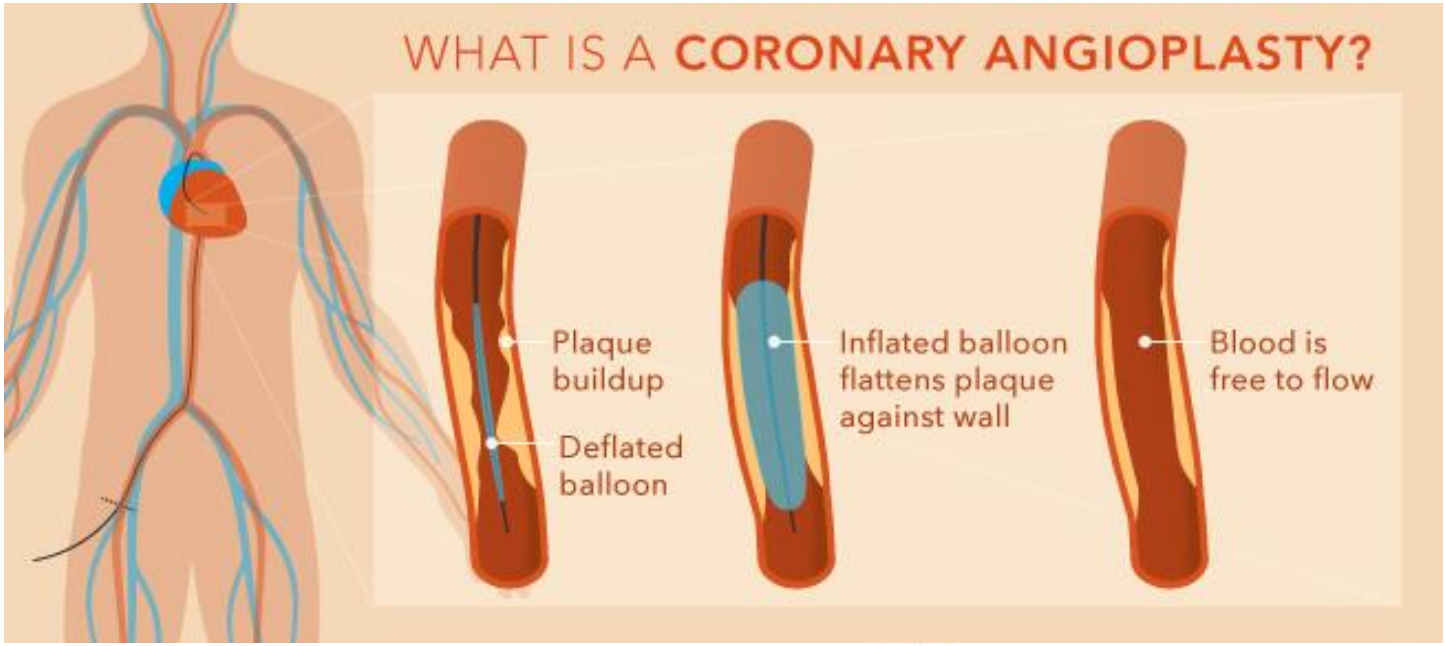
Cause of referred pain ?

Remedy: -vasodilator drugs- *nitroglycerin*

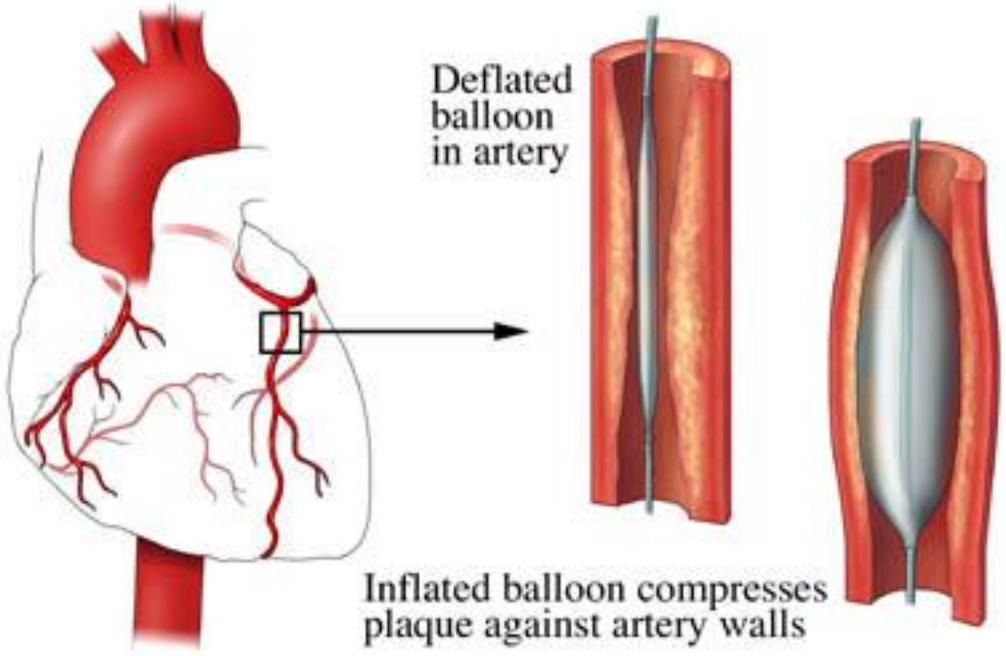
***beta blockers –* propranolol - block sympathetic beta adrenergic receptors - prevents sympathetic increase of heart rate and cardiac metabolism during exercise or emotional episodes.**

Surgical also

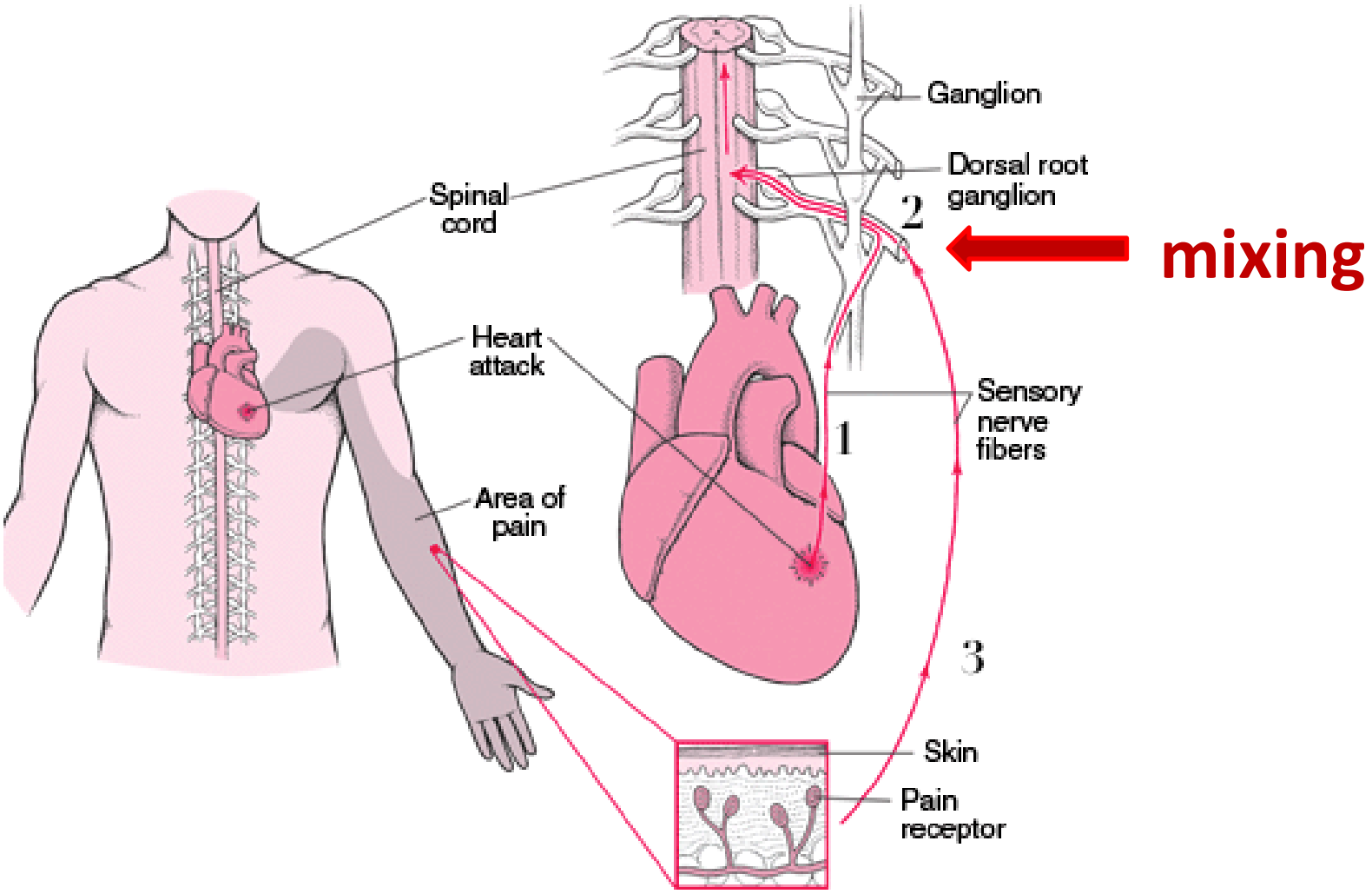




Physiological basis



Referred pain in MI



- Same nerve branches supply to heart and (**embryonic life**) **surface areas of arms on left side body receive pain nerve fibers from the same spinal cord segments.**

ESSENTIALS OF TODAY'S LECTURE

- *Regulation of blood flow to organs*
- *Regulation of blood pressure*
- *Measurement of BP*
- *Hypertension*
- *Hypotension*
- *Circulatory Shock*
- *Ischemic heart disease*
- *Heart failure*

What ever book you may follow in library, what's important is comprehension