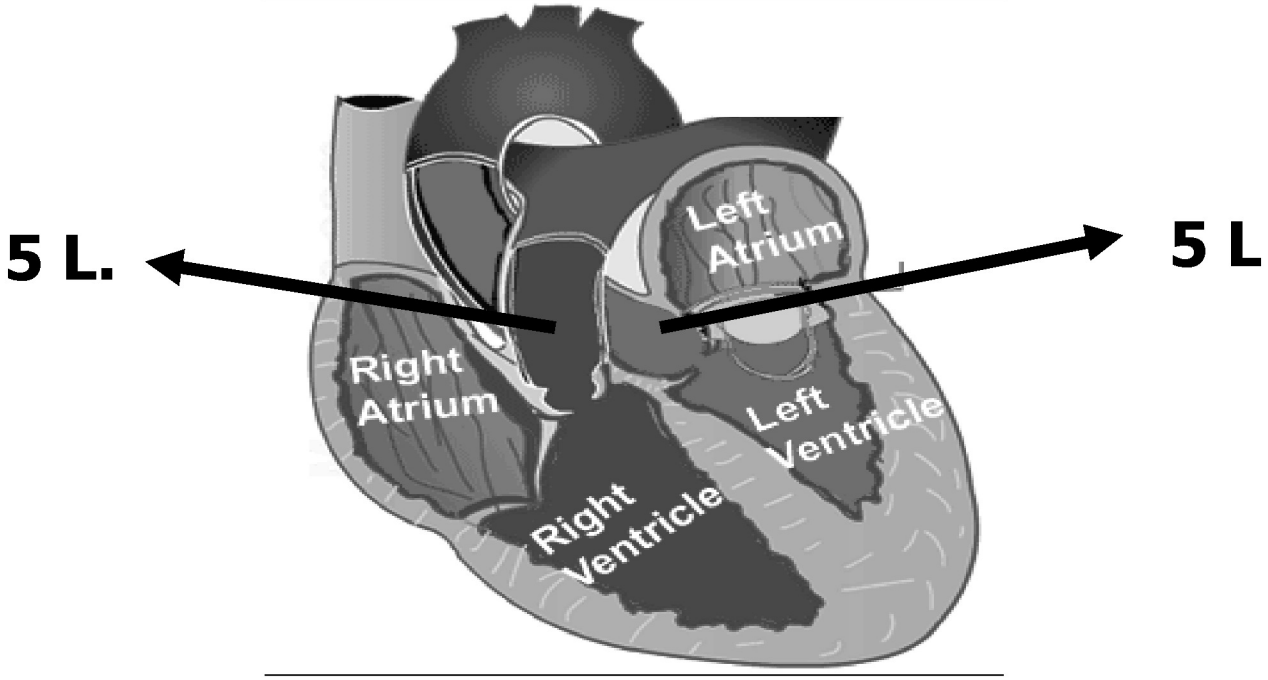
# Cardiac Output



# ❖Cardiac Output

❖**Definition**-It is the amount of blood pumped out by **each ventricle** in one minute. Also called **minute volume**

Normal value- 5 lit/min

- **Cardiac Index** is cardiac output per square meter of body surface area. It is 3L/$m^2$ /min in adult weighing 70 kg.

# Stroke Volume

- **Definition- Is output per ventricle per beat**
- Normal value = 70 ml/ beat
- **Stroke vol. Index-** Is volume per square meter of body surface area & is ~ 47ml.

# ❑Distribution of Cardiac Output

| Body organ | Blood flow ml/min |
|---|---|
| • Liver | 1500 |
| • Kidney | 1300 |
| • Brain | 750 |
| • Lungs | 500 |
| • Heart | 250 |

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| Organ | Blood flow |
| --- | --- |
| • Skeletal musc.+ | |
| Other organs | 1000 |
| • Skin | 500 |

❖About 75% goes to liver, kidney, brain, lungs & heart. i.e. to the vital organs. Rest 25%goes to other organs

# Cardiac Reserve

- Is the maximum amount of blood that heart can pump. It is physiological limit of heart
- Normal value= **30 to 40 L/min** during exercise

# ❑Control of Cardiac Output

- **Cardiac output** = HR x SV

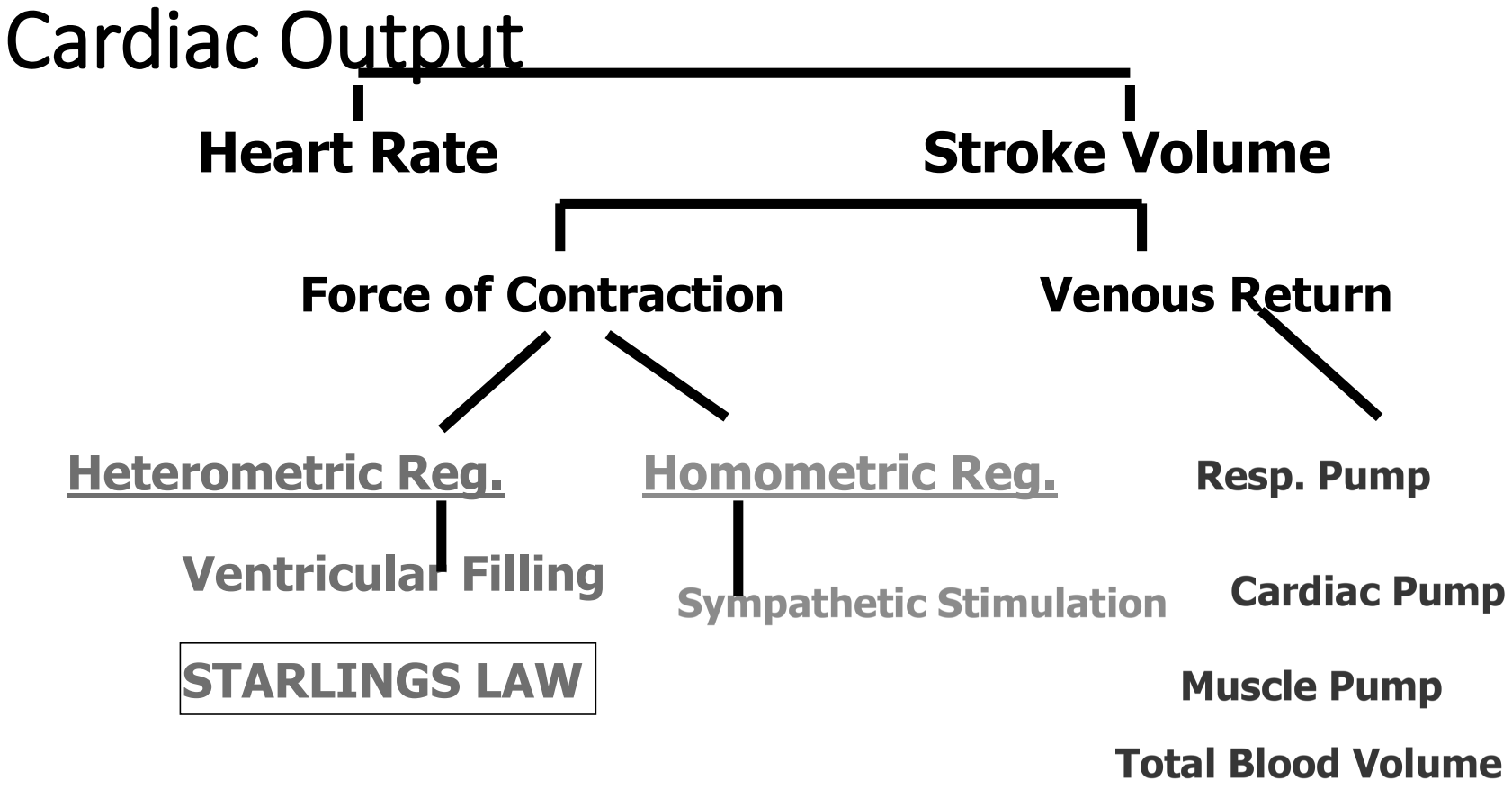
  Normal HR = 70 beats/min

  Stroke volume = 70 ml / beat

Cardiac output = 70 X 70 = 4900 ml/min. or about 5L/ min

# Factors

- 1. Heart rate
- 2. Force of contraction
- 3. Venous return
- 4. Peripheral resistance



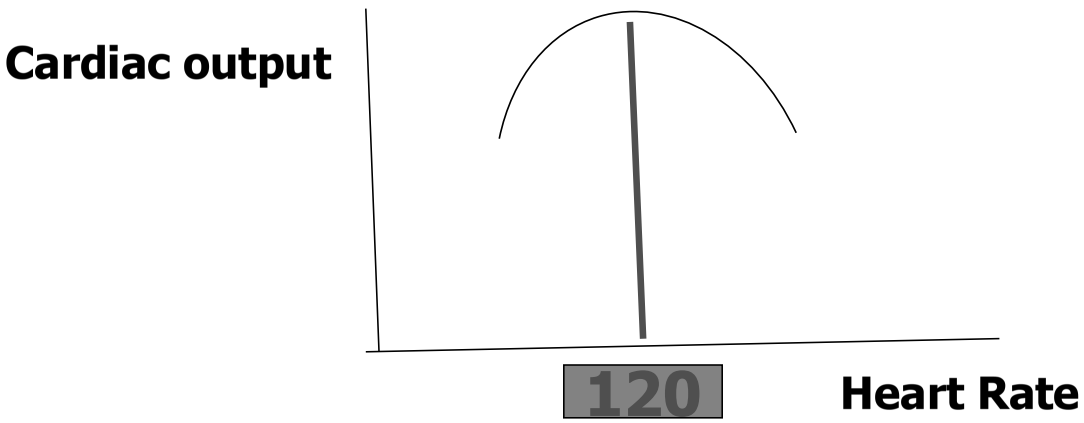
# 1. Effect of Heart Rate

Heart rate has a direct relationship with cardiac output

- ↓ HR leads to fall in cardiac output
- ↑ HR leads to a but it has a upper limit of 180 beats /minute. provided venous return is adequate.

- Normal heart can increase cardiac output ~ 13L/min. (Frank-Starlings law) without excess nervous stimulation. Venous return is limiting factor

# Heart rate & cardiac output



# 2. Force of contraction

Is regulated by two ways:

1 Heterometric regulation

2 Homometric regulation

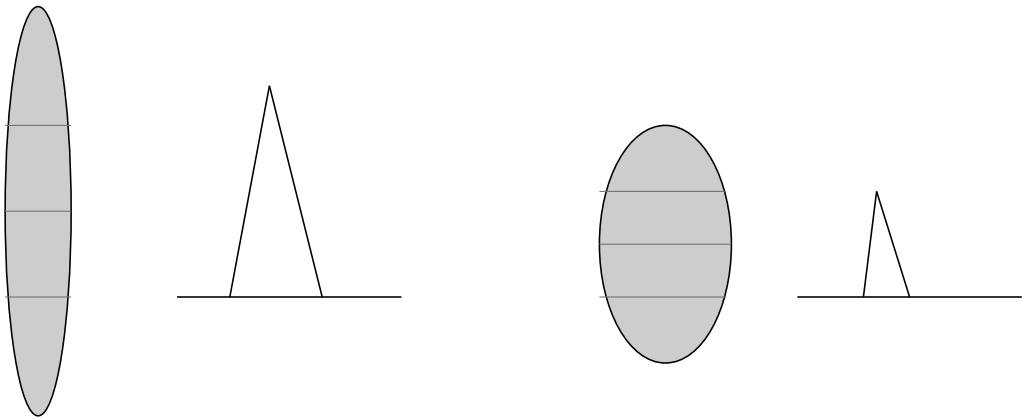
**Heterometric Regulation**

Hetero – change  meter—length

- An increase in initial length of muscle fiber leads to ↑ force of contraction

# ❑Frank-Starlings Law

- Frank-Starlings Law- “With in physiological limits the force of cont. of cardiac muscle is proportionate to the initial length of muscle fiber”.



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- In ventricles initial length depends on the ventricular filling / End Diastolic Volume / Pre Load of the heart &
- It depends on venous return.

Initial length depends on End-diastolic volume

**More Filling**

**More Initial Length of muscle fiber**

# Clinical significance

1. Keeps output of two ventricles equal
2. Stroke volume is constant even if the peripheral resistance ↑
3. Life saving in cardiac failure

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- 4. After Heart Transplant—It is the only mechanism to regulate cardiac output during exercise. Increase is small but appreciable.

# Homometric regulation

- Seen in sympathetic stimulation
- No change in initial length of muscle fib.
- Vigor of contraction increases

# 3.Venous Return

Factors affecting venous return:

- 1. Respiratory pump
- 2. Cardiac pump
- 3. Muscle pump
- 4. Blood volume
- 5. Sympathetic discharge
- 6. Gravity

# Respiratory Pump

Normally

- End expiratory intrathoracic pressure is

  -2mm of Hg

- End inspiratory intrathoracic pressure is

  -5mm of Hg

- 1.This ↓ Intrathoracic pressure decreases pressure in inferior venacava

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- 2. During inspiration diaphragm descends, & intra abdominal pressure rises
- 1 & 2 together ↑ blood flow in Rt. Atrium
- Res. Pump operates strongly in forced respiration like during exercise

# Cardiac Pump

- Generates - Two type of forces
- Vis-a-tergo, force from behind
- Vis a fronte, suction force from front
- Vis-a-tergo- It results from myocardial contractility & is propelling force which pushes blood in the Aorta

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- Vis-a-fronte-- Ventricular contraction & respiratory pump produce a suction effect which draws blood in heart

Again it can be of two types

- 1-Ventricular systolic suction during systole AV ring is pull down – ↓intra atrial pressure & blood is sucked from inf. venacava

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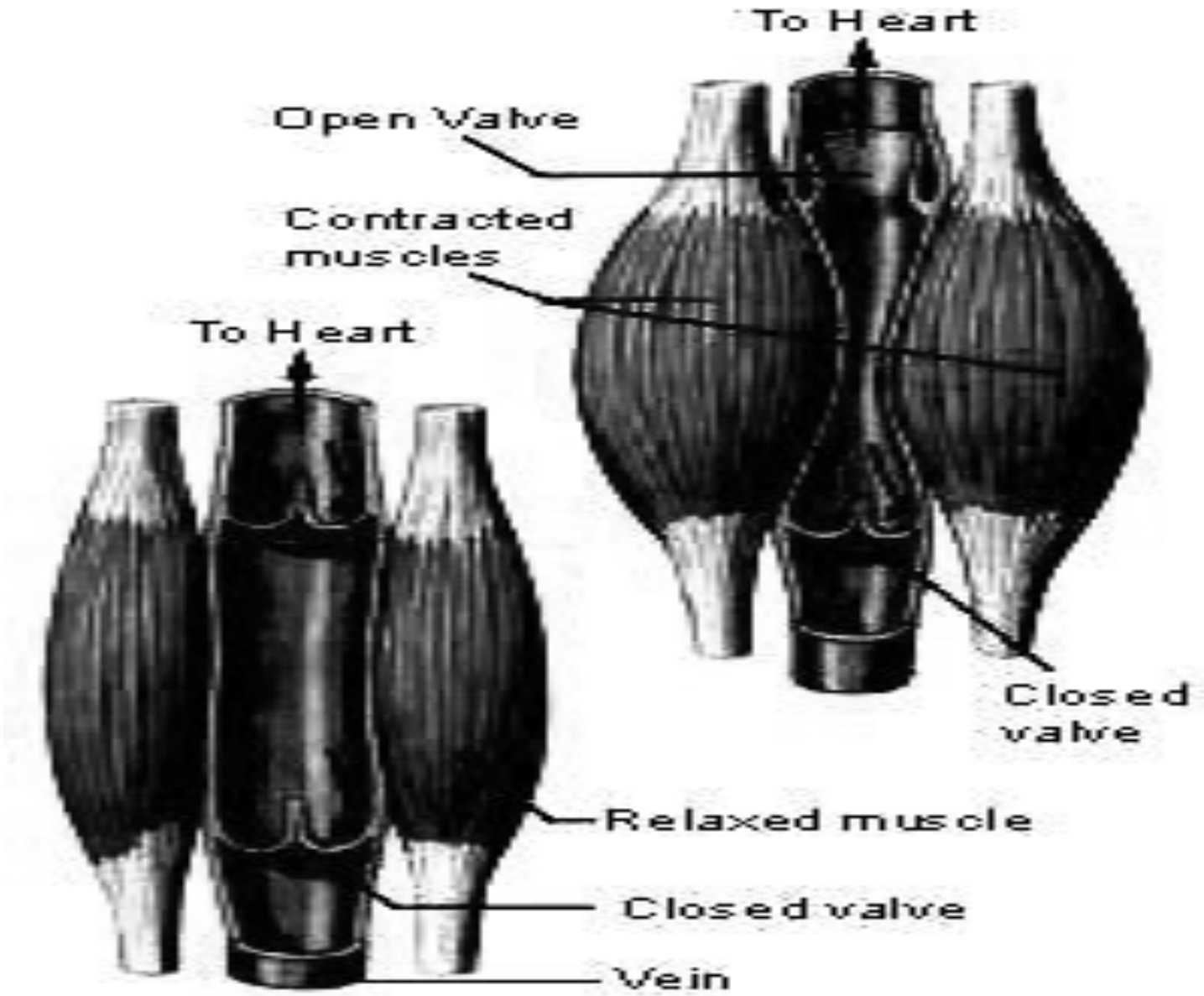
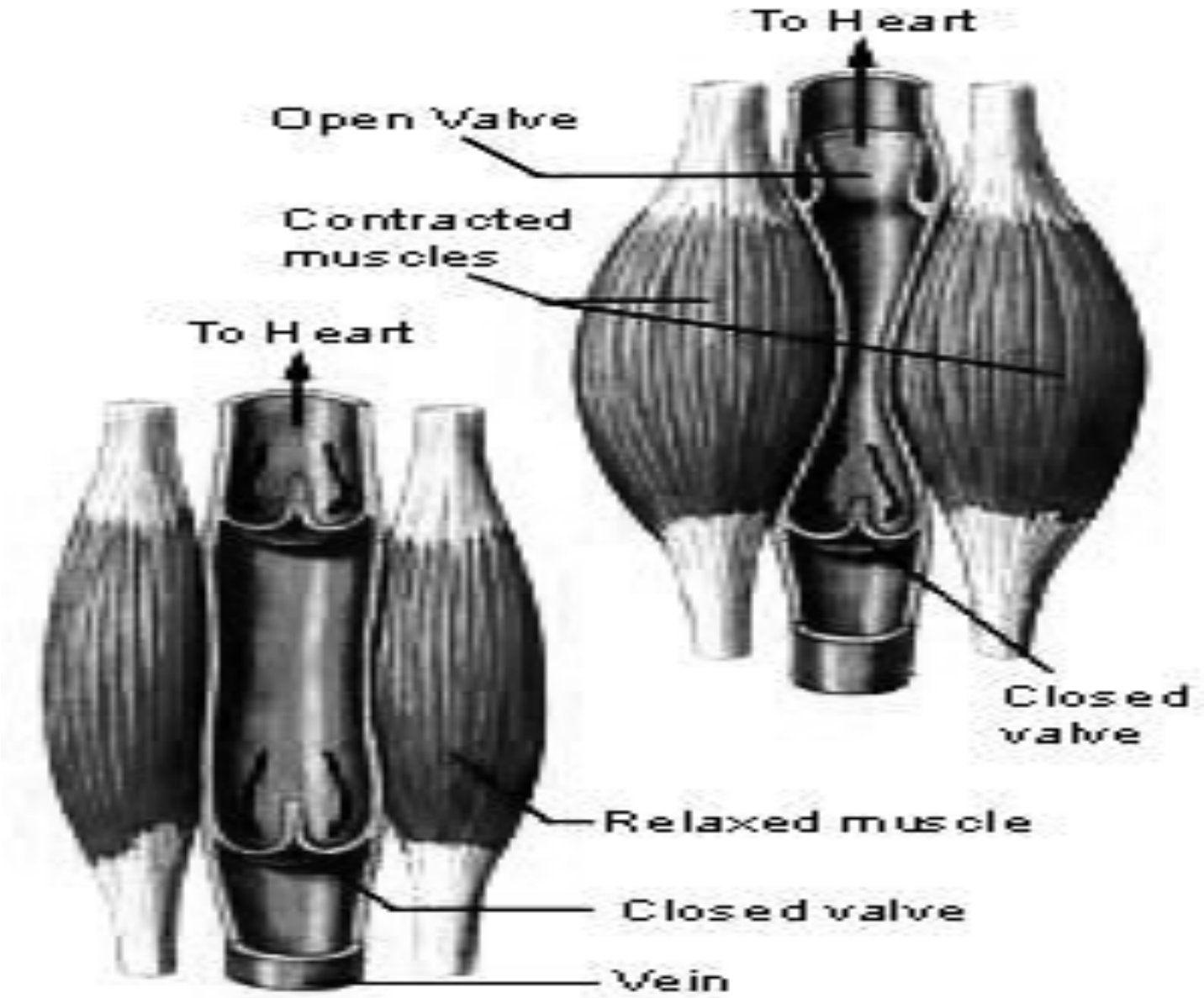
- 2-Ventricular diastolic suction—Opening of AV valve leads to sudden ↓in intra atrial pressure & blood is sucked from great veins

# Muscle Pump

- Contraction of muscles have squeezing effect on blood vessels. It helps in venous return. Specially imp. during exercise





# 4. Peripheral Resistance

- Heart maintains a constant cardiac output & blood flow even against increased peripheral resistance

# Measurement: Methods

Can be measured by

- Heart Lung preparation
- Cardiometers

**Other methods are**

1. Based on Ficks Principle
2. Indicator or dye dilution method
3. Thermodilution

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3. Thermodilution
4. Based on inhalation of inert gases
5. Echocardiography
6. Ballistocardiography
7. Cinecardiographic tech.

# Variations in Cardiac output

Physiological: normal heart without any excess nervous stimulation can increase  output up to 13 L/min.

Pathological:

- Hypoeffective heart &
- Hypereffective

# Hypoeffective heart

Factors:

- Myocardial damage or toxicity
- Congenital heart diseases
- Abnormalities of Rate & Rhythm
- Myocarditis
- Increased arterial pressure

# Hypereffective heart

Factors:

- Nervous stimulation: Sympathetic stimulation can ↑ cardiac out put up to 25 l/min.
- Hypertrophy of heart: is increase in mass & contractility of heart e.g. heavy exercise.

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- Combined effect of above 2 factors can increase cardiac out put up to **30 to 35 ml/min**

# ❖Blood Pressure

❑Definition— It is arterial pressure & is

**lateral pressure** exerted by flowing blood on the walls of the vessels

- It is exerted at right angles to the direction of flow
- Lateral pressure-represents potential energy

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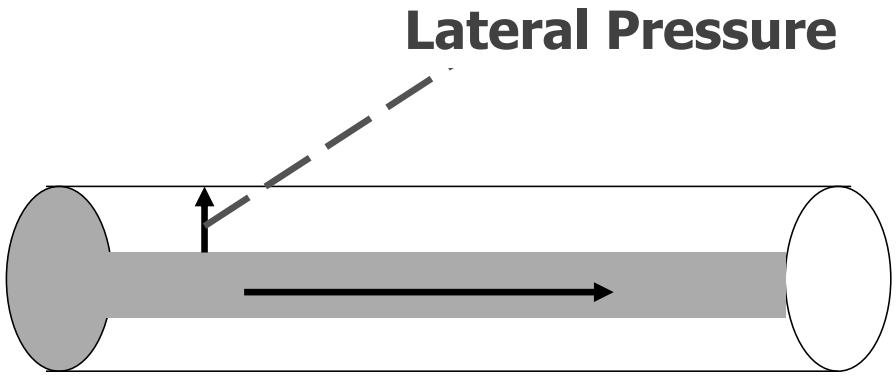
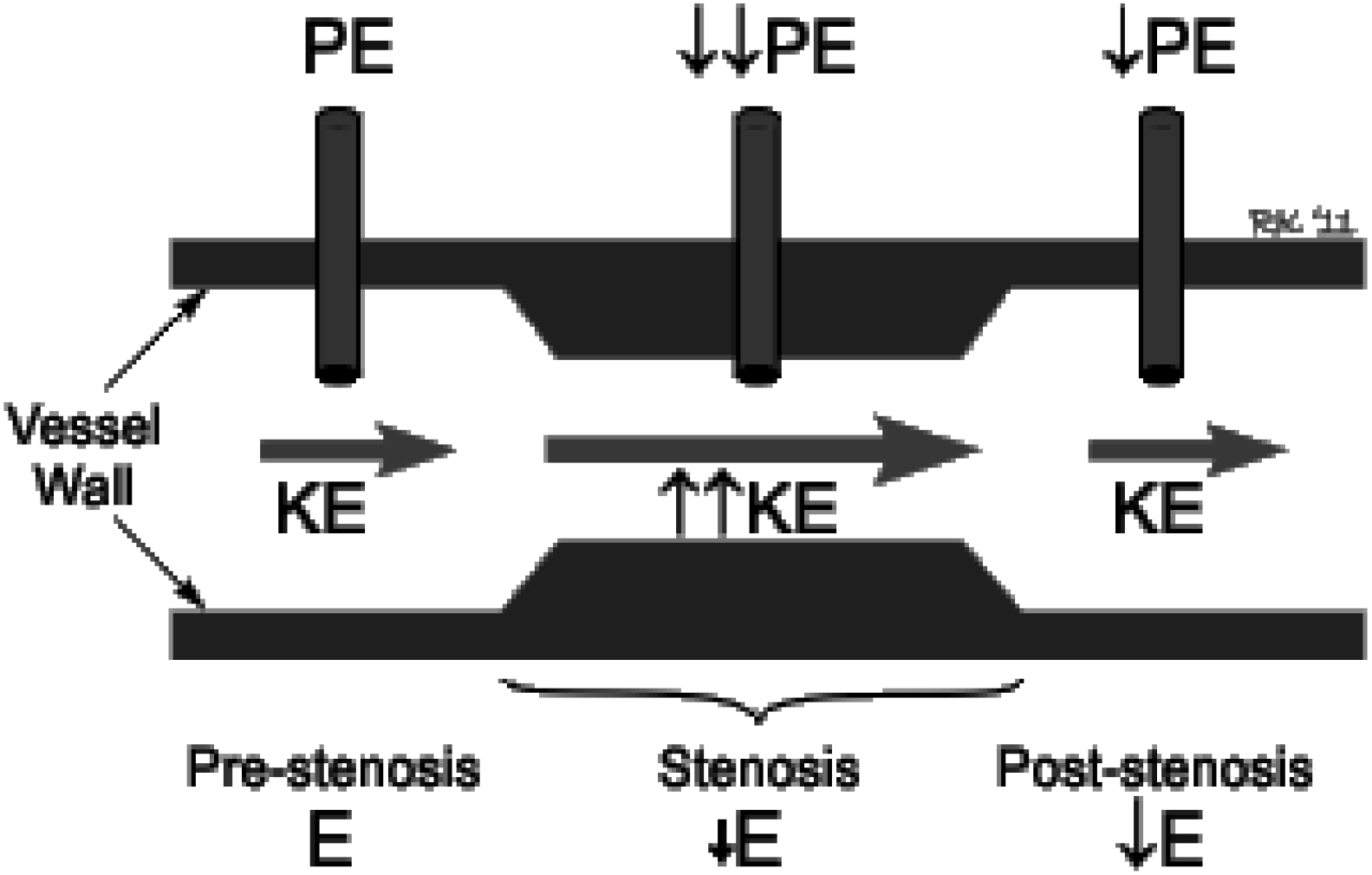
- Another is **End pressure** also called perfusion pressure – represents total energy (KE + PE)
- Kinetic E. depends on blood velocity
- If velocity is constant than lateral press indicates a rise in perfusion pressure.

# Arterial pressure





# Blood Pressure

- Systolic Pressure
- Diastolic
- Pulse
- Mean

# Systolic Pressure

Systolic pressure-Maximum pressure during systole of heart

- Normal value—120 mm Hg (105—135)
- It depends on **cardiac output**
- Indicates extent of work heart is doing
- Shows considerable fluctuation

# Diastolic Pressure

Diastolic Blood Pressure-Minimum pressure during diastole of heart

- Normal value -- 80mmHg (60—90)
- It depends on total **peripheral resistance**
- Indicates constant load of heart
- Undergoes much less fluctuation

# Pulse & Mean Pressures

- Pulse pressure—systolic – diastolic
  120 - 80= 40 mmHg
- It indicates stroke vol.
- Mean Arterial pressure

  Diastolic + 1/3 of pulse press

  80 + 13= 93mmHg

# ❑Mean Pressure

Mean Arterial Pressure very important

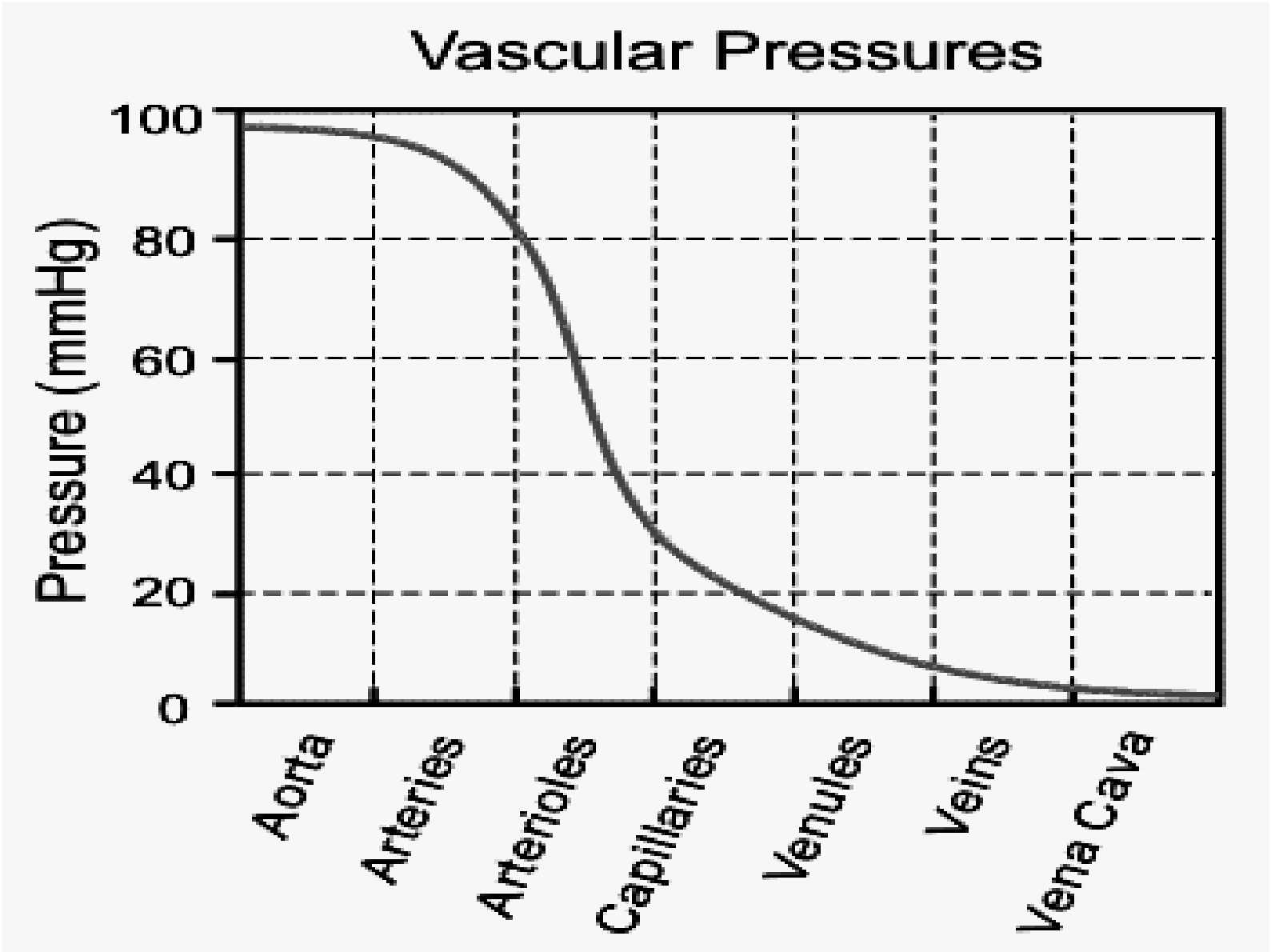
- Determines regional blood flow of that organ
- All cardiovascular reflexes are sensitive to mean arterial pressure

# Expression of Blood Pressure

Conventional way of expression

- Systolic / Diastolic: 120 / 80 mmHg.

- **Central Venous Pressure:** is pressure in rt. atrium because all the systemic veins open here



# Factors Affecting BP

- Physiological factors
- Pathological factors

Physiological Factors:

- 1. Age- Both ↑ with age
- 2. Sex- Before menopause BP is less in females by 4-6mmHg (estrogen).

  No diff. after menopause

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- 3. Meals- After meals

  Systolic ↑ by 4-6 mmHg, lasts for about one hr.
- Diastolic shows no change or ↓ as there is fall in PR because of vasodilatation in splanchnic vessels
- 4. Emotions: fear, anxiety--↑ BP(↑Symp)

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- 5. Temp.

  Cold ↑ BP because of ↑ PR due to cutaneous vasoconstriction (action through hypothalamus

  Hot ↓ BP because of ↓ PR caused by cutaneous vasodilatation (through hypothalamus)

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- 6 Diurnal variation-BP ↓ in morning and ↑ in after noon, by 6-10 mmHg
- 7 Exercise- Generally systolic ↑ and diastolic ↓

  Both returns to normal with in 5 min. of stoppage of exercise

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- 8. Gravity- It is more in vessels below the heart
- And less in vessel above heart
- For every one cm. change in height BP changes by 0.77 mmHg
- 9. Posture- In standing position dia. BP ↓ for about 30-60 sec. but it is immediately corrected by barocepters

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- 10. Sleep-

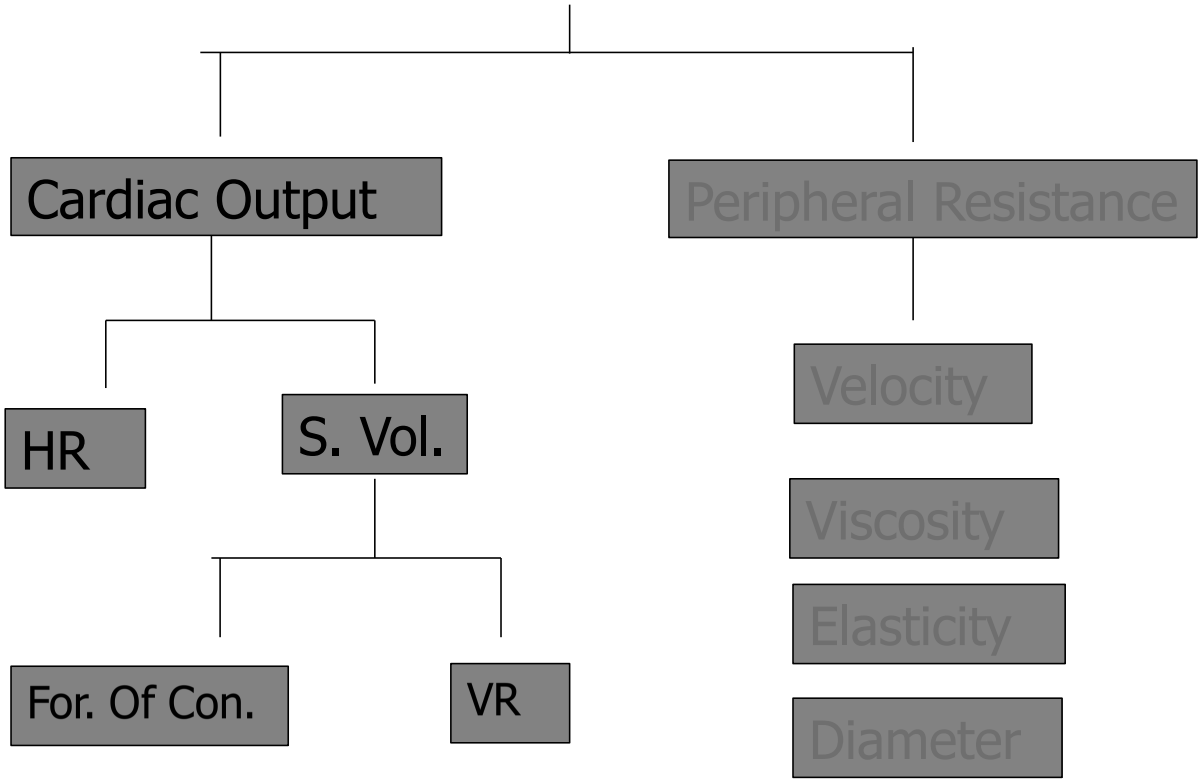
  Early hrs. of sleep- fall by 15-20 mmHg

  In disturbed sleep BP rises as symp. activity is more
- 11. Body built-Sy. BP more in obese. (Some error is because of cuff)

# Determinant of BP

- BP = cardiac output X Peripheral Resist.
- Cardiac output = HR X Stroke Volume
- Stroke volume = Force of contraction X Venous Return
- Change in any of these factors will affect BP

# BLOOD PRESSURE



# Peripheral Resistance

It is the resistance which blood has to overcome while passing through the periphery. The chief seat of PR is the arterioles

Resistance depends on :

- 1. Velocity of blood flow
- 2. Viscosity  of blood

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- 3. Elasticity of vessel wall
- 4. Diameter of blood vessels

  **Velocity:** rapid flowing blood will have more frictional effect than a slower one
- Hence pressure is high in Aorta

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- **Viscosity**-more viscid blood will have a higher friction & high BP
- **Elasticity** Due to elastic property arteries can accommodate considerable amount of blood with little rise in BP
- In old age arterial walls become stiff & BP changes

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**Diameter of vessel:** Has inverse relation with BP

- Capillaries have small diameter but velocity is also less so BP is not high

# Hagen Poiseuille's Law

$$R = \frac{8nl}{\pi r^4}$$

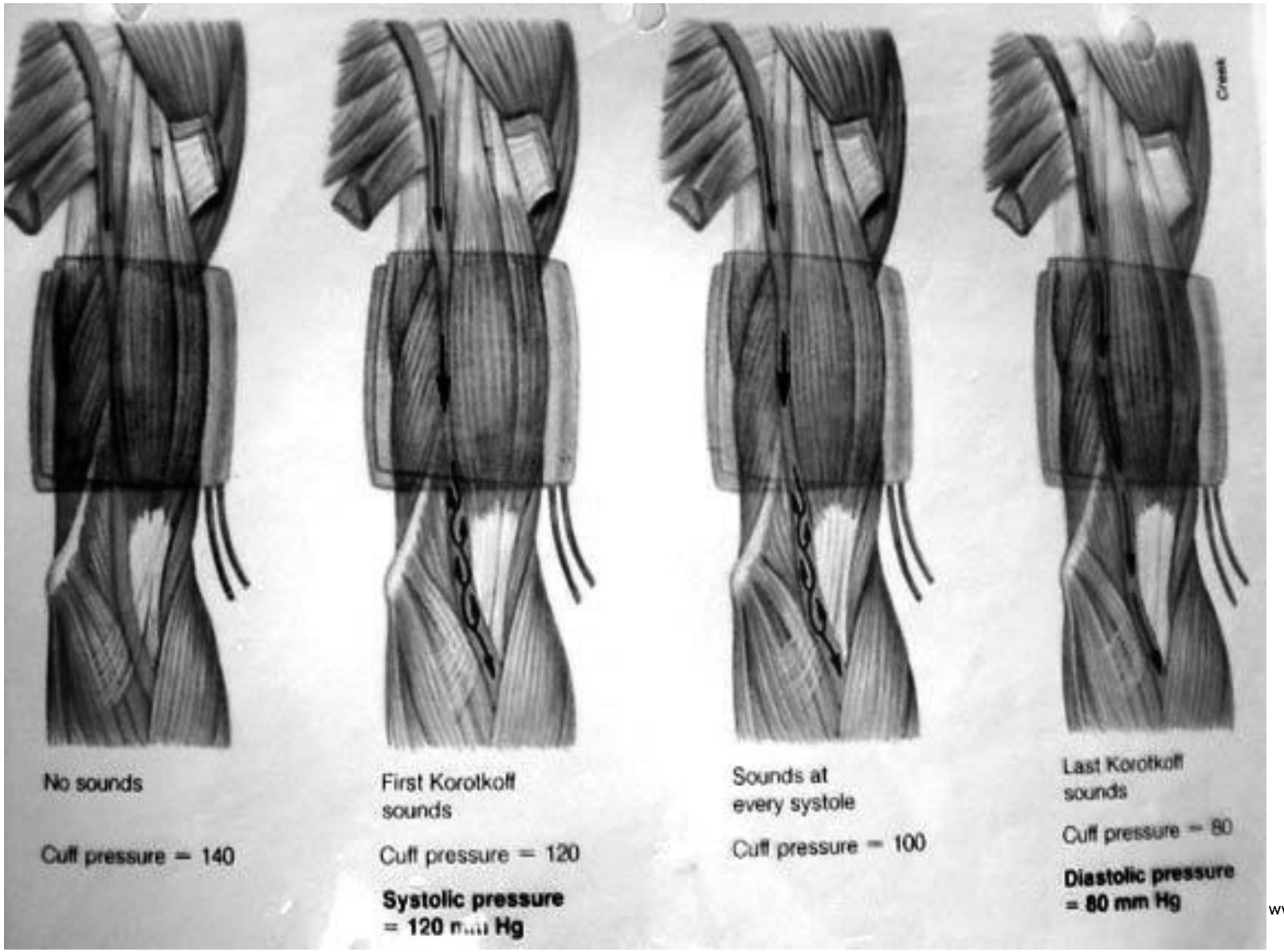
# ❑BP- Measurement

- Direct Method- used in experimental studies
- Indirect Method- Sphygmomanometery

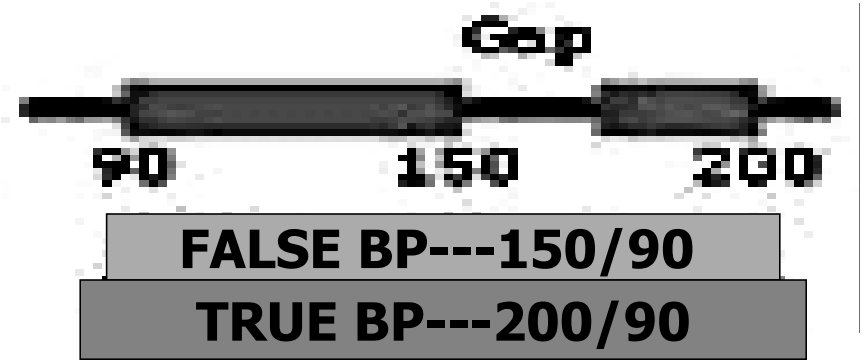
  Palpatory

  Oscillatory

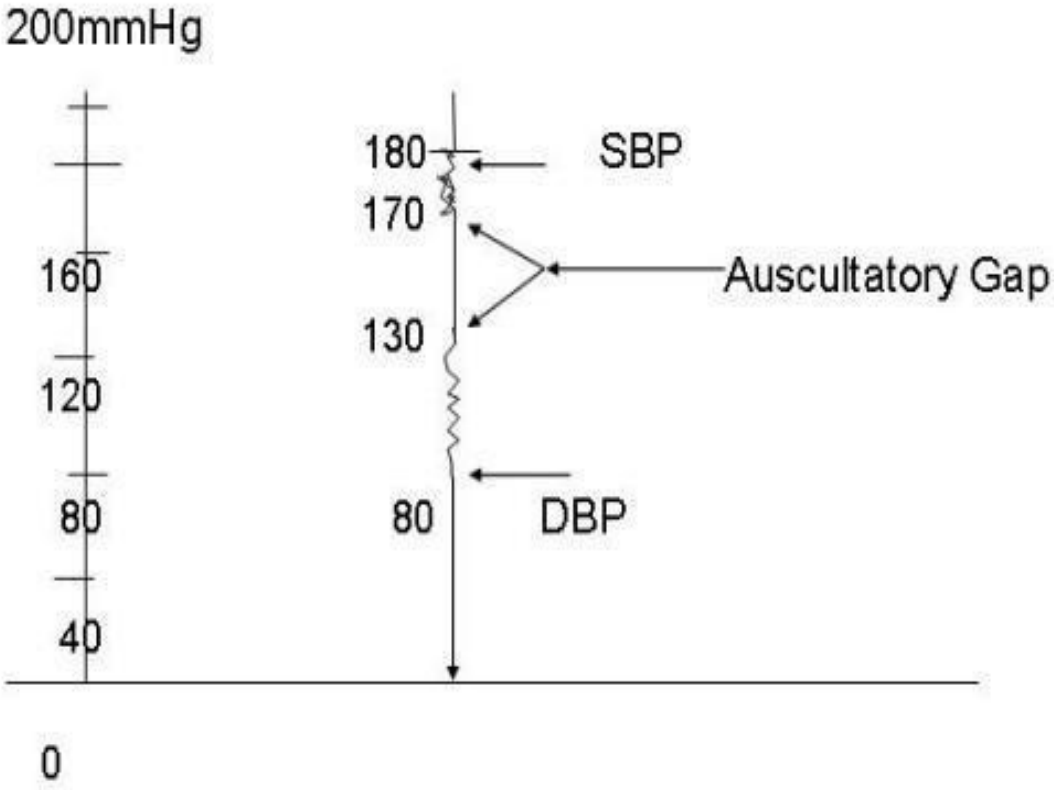
  Auscultatory



# ❑Auscultatory gap



# BP Monitoring: Auscultatory Gap



# Aneroid type

# ❑BP-Regulation

Three main mech.

- Rapid or short term regulation
- Intermediate control mech.
- Long term regulation

# Short term Regulation

- Is by nervous mechanisms: most rapid mechanisms
  - Baroreceptors
  - Chemoreceptors
  - Atrial reflex
  - CNS ischemic response
  - Abdominal compression Reflex

# ❑Baroreceptors

- Are pressoreceptors & are sensitive to stretch
- Located in the carotid sinus & aortic sinus
- Aortic Sinus Inn. by Vagus nerve
- Carotid sinus by Hering branch of Glossopharyngeal nerve

# Characteristics

- They come into action very quickly with in seconds
- Very important in regulating BP during day to day activities
- *Buffer function: they oppose either increase or decrease of BP. so called buffer sy. & nerves are buffer nerves*

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- Respond more rapidly to changing pressure than to a stationary press.
- Not stimulated between 0 to 60 mmHg.
- Normal operating range is 60 to 180
- Normal arterial pressure is around 100
- Thus they function most effectively in the range where it is most needed

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- They show adaptation quickly with in hours to days. So not useful in long term regulation
- Not helpful in chronic diseases like atherosclerosis or chronic hypertension
- In arteriosclerosis they loose their efficacy

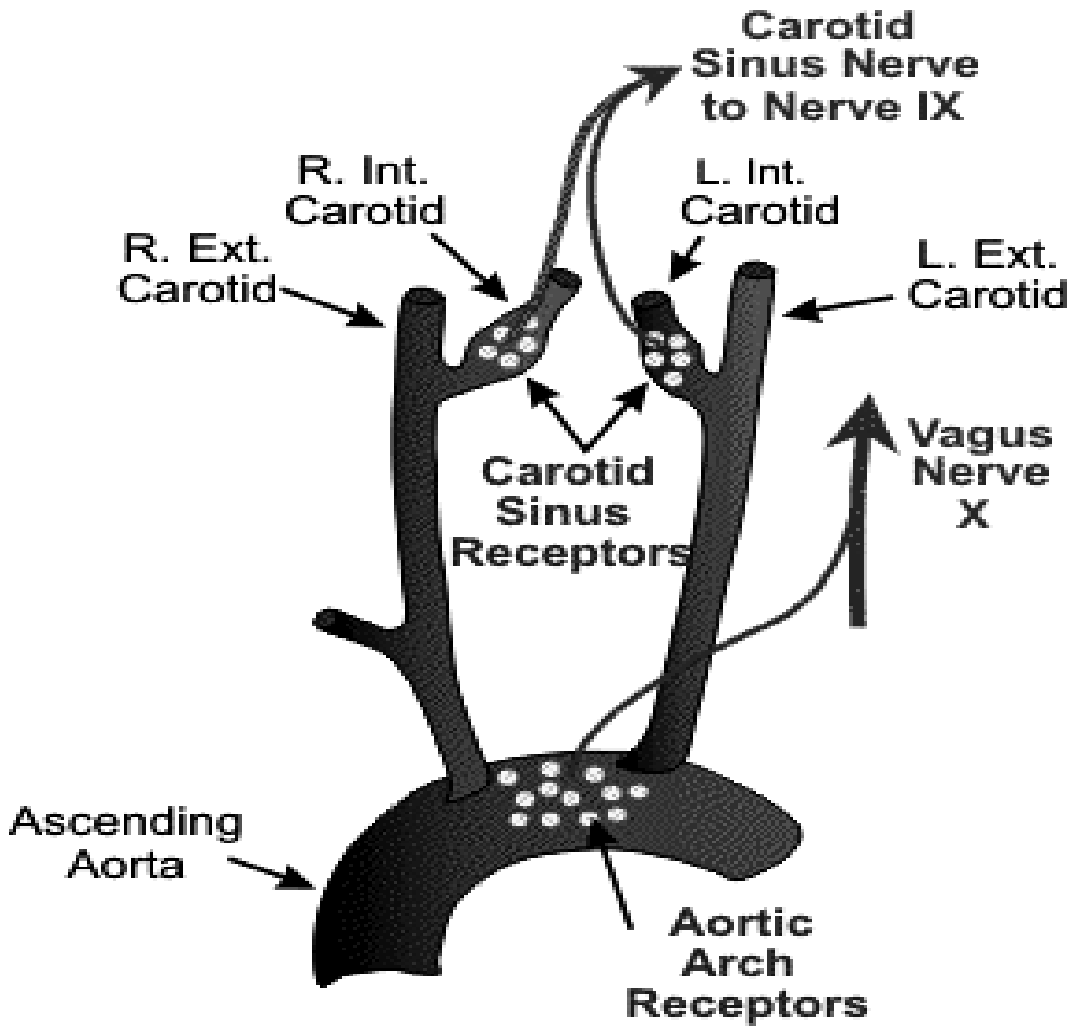
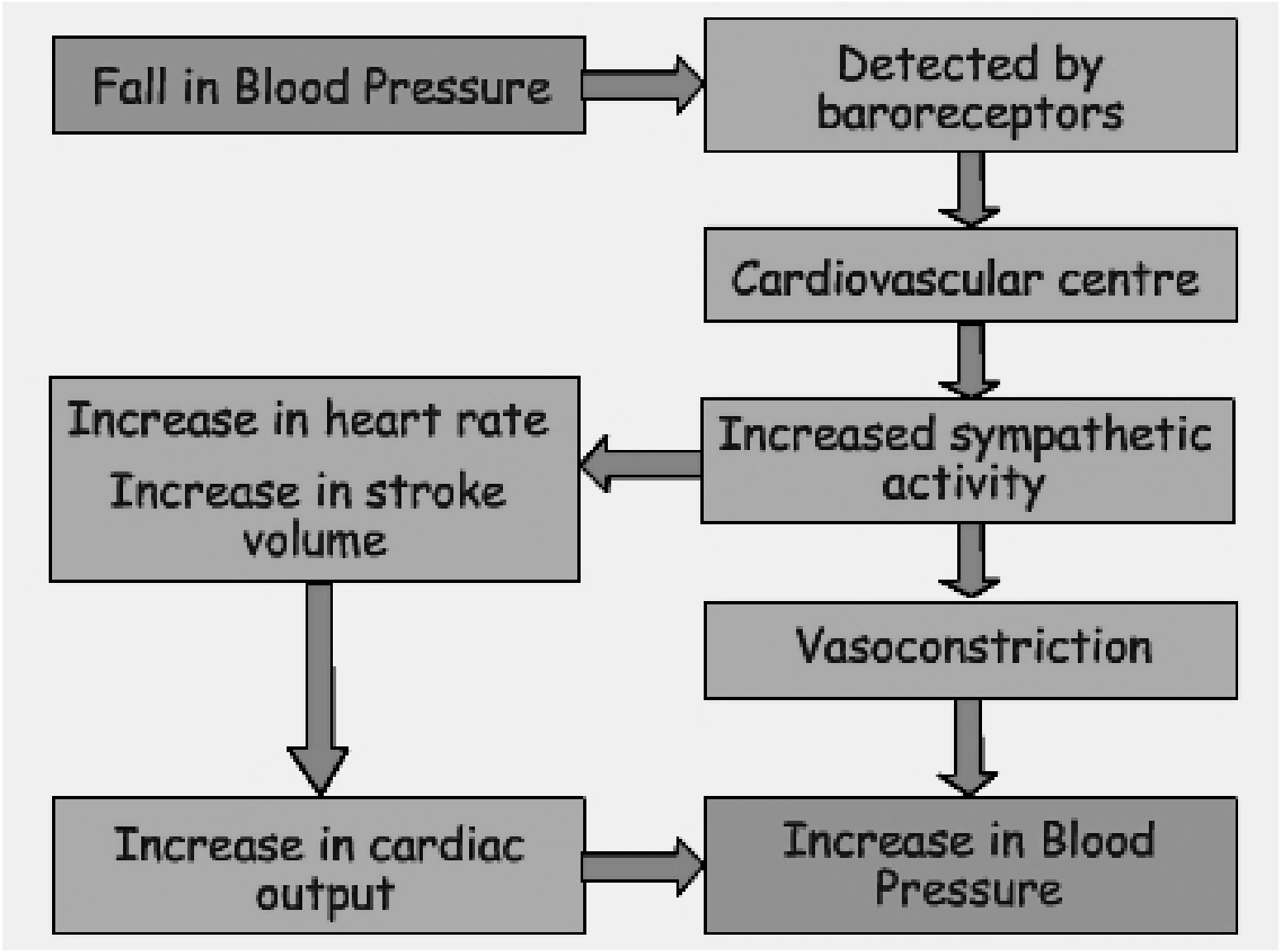



Figure 1. Location and innervation of arterial baroreceptors.



# Chemoreceptors

- Located in the Carotid bodies & Aortic bodies
- Inn. By Hering nerves & Vagi nerves
- Becomes active when pressure falls below a critical level of 80 mmHg.
- Diminished $O_2$ supply stimulate VMC through chemoreceptors

# Atrial Reflex

- Also called low pressure receptors
- Atria contain stretch receptors
- ↑Atrial pressure causes ↑ heart rate

  Causes are
- Bainbridge Reflex
- Stretching of sinus node

# ❑Bainbridge Reflex

- Stimulation of Atrial Stretch Receptors
- Signals through vagus to Medulla
- Efferent through vagus & sym. nerves

↑ Heart Rate

# ❑CNS Ischemic Response

- Initiated when BP falls below 60mmHg.
- blood flow to VMC (cerebral ischemia)
- Strong stimulation of neurons in VMC
- Due to accumulation of CO2, lactic acid
- Intense & v. powerful vasoconstriction peripheral vessels like renal vessels are totally occluded

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- It is an emergency in fact last ditch response
- If CNS Ischemia is not relieved neurons begin to suffer & can become inactive within 3 to 10 minutes.

# ❑Cushing Reflex

- CSF pressure (equal to arterial press)

- Brain art. compressed (blood supply cut

# Abdominal Compression Ref.

- When VMC is stimulated other reticular areas of brainstem are also stimulated
- Impulses go to skeletal muscles specially that of abdomen
- Abdominal mus. contract & compress the abdominal venous reservoirs
- This leads to increase in HR & BP

# Intermediate Mechanisms

Intermediate controlling mechanism:

- Renin- Angiotensin mechanism
- Stress relaxation & Reverse stress relaxation
- Capillary fluid shift method
- Comes into action after several minutes and reach to maximum in few hrs.

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- They function from few days to few week
- Act by altering the blood volume

# Longterm Regulation

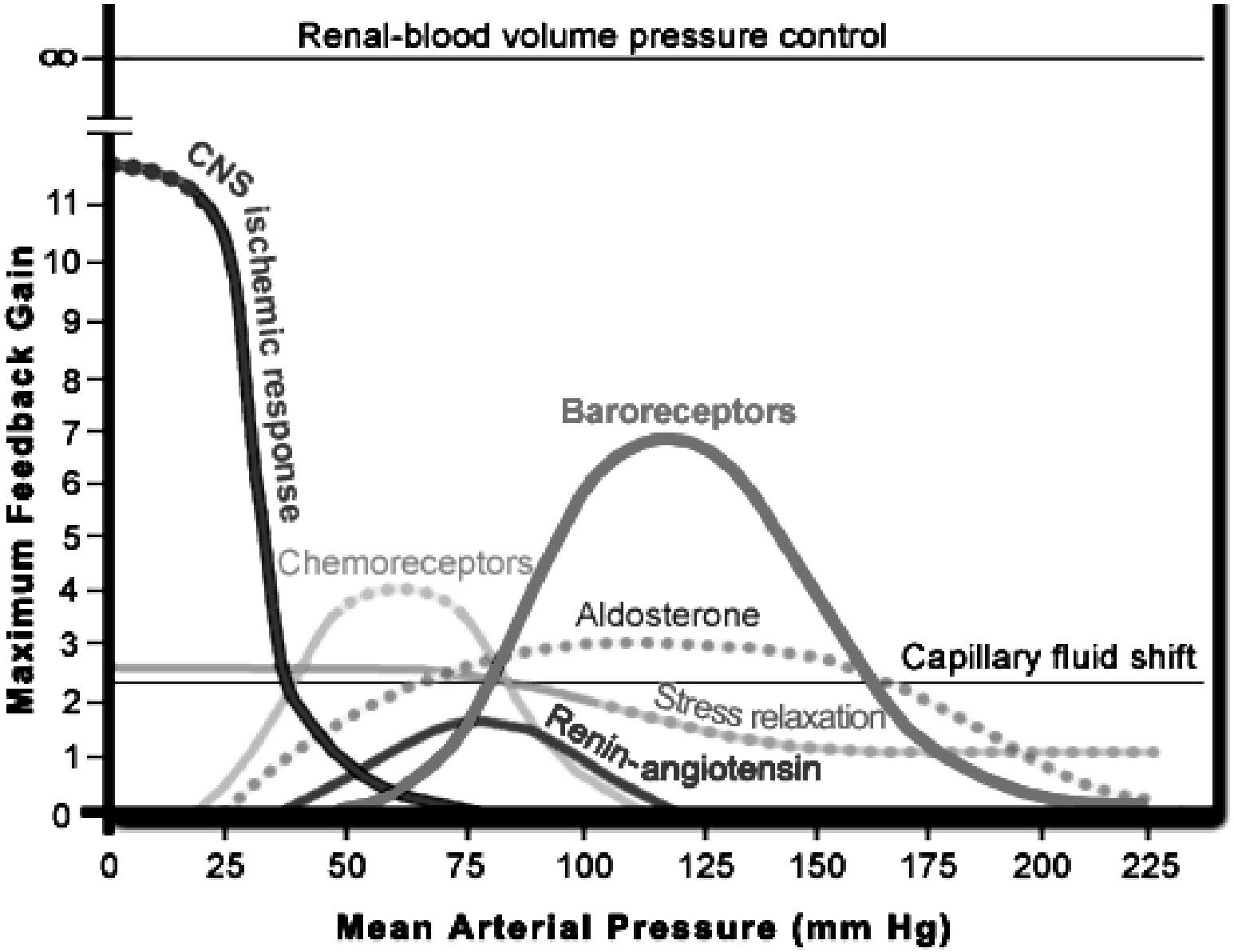
Long term regulation:

Is by kidneys

- Direct- renal body fluid feedback mech.
- Indirect- via hormones

  Aldosterone

  Renin angiotensin mech.



# Other Mechanisms

Includes:

- fluid absorption from GIT
- Conservation of water & salt by kidneys
- ↑ desire to take salt
- ↑ thirst

# ❑Conclusion

- To conclude rapid control of BP begins with life saving measures of nervous reflexes, continue with sustaining characteristics of the intermediate controlling mechanisms and finally BP is stabilized by long term mechanisms.

# Effect of Art. BP. on tissue blood flow

- Tissue blood flow remains almost normal, if BP. variations are between 75 to 175 mmHg. It is called **Autoregulation**

# ❑ Autoregulation

Is ability of tissue to regulate thier own blood supply

- Two theories:
- Myogenic theory
- Metabolic theory

# Myogenic theory

- Blood vessels have a basal / resting myogenic tone
- Stretch of vessel by high arterial pressure causes vasoconstriction & reduces the blood flow
- Low BP has opposite effect
- Effect is caused by contraction or relax. of smooth muscles

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- Myogenic Theory-

  Vascular smooth muscles

  **Stretching** ↓

  Contraction of muscles ↓

# Metabolic theory

- **Local vasdialator metabolites** are formed with increase in tissue activity
  1. ↑$pCO_2$
  2. ↓$O_2$
  3. ↑Temp.
  4. Accumulation of $K^+$

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- 5. Lactate
- 6. Increase in blood osmolarity
- 7. Adenosine in cardiac muscle not in skeletal muscle
- 8. Histamine released from damaged tissues

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- Metabolic Theory- **High tissue metabolism** ↓ Collection of local vasodilators eg. CO2, K+, adenosine & in temp.

↓

**Vasodilatation** & metabolites are washed away

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- **Local vasoconstrictors-**
- 1. Serotonin, ↓temp
- 2. Substances released from endothelium –Prostaglandins, thromboxane $A_2$, Endothelium derived relaxing factor
- 3. Endothelins

# Role of endothelium

Endothelial cells secret

Vasoactive substances like:

- Prostaglandins
- Nitric oxide
- Endothelins
- Thromboxanes &
- Many growth factors

# Thromboxane $A_2$

- Formed by platelets & promotes platelet aggregation & vasoconstriction
- Another substance prostacycline is secreted by endothelial cells and promotes vasodilatation

# ❑Venous pressure

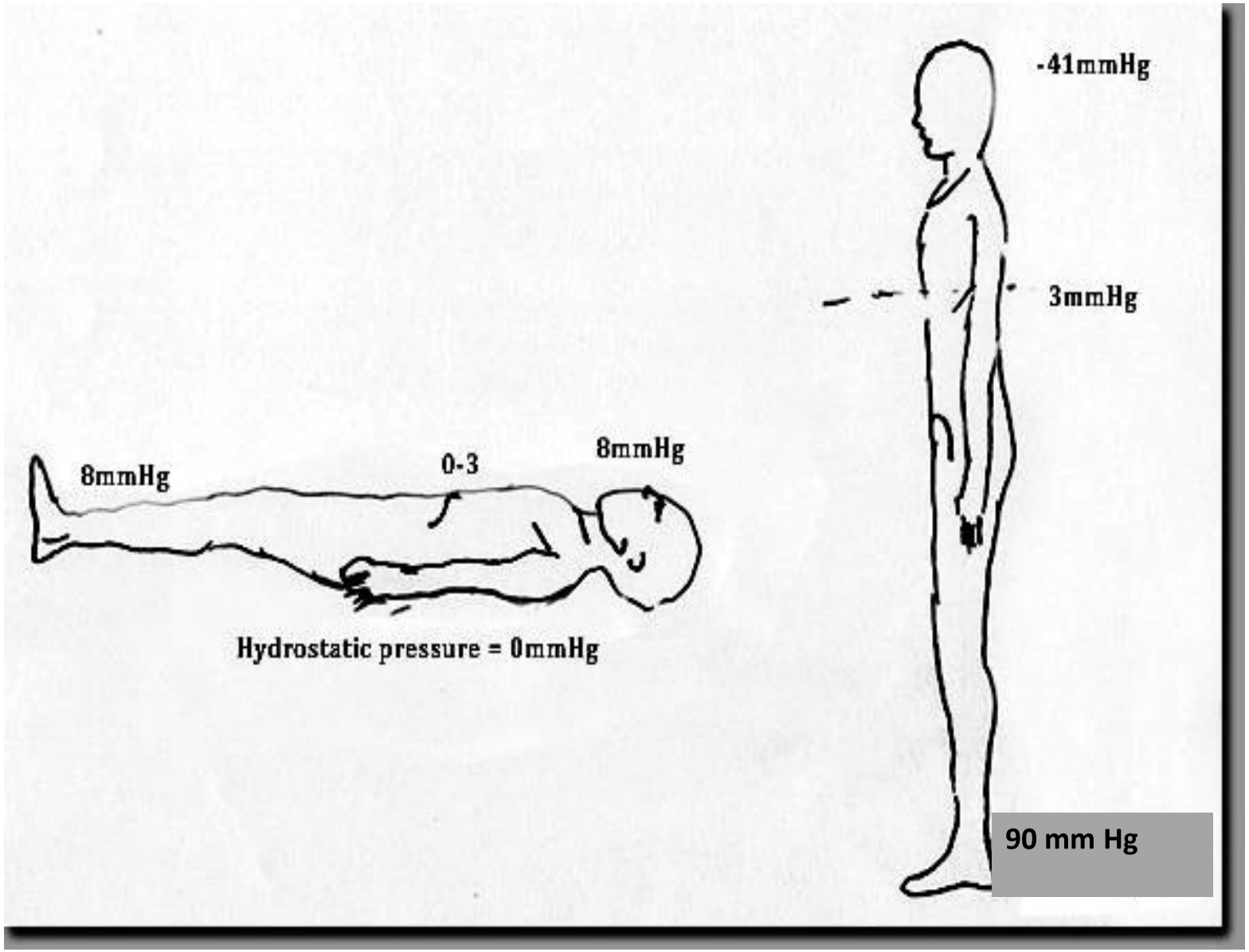
- **Central Venous Pressure:** is pressure in rt. atrium because all the systemic veins open here
- Clinically it is assessed by observing the degree of distension of neck veins
- Venous pressure in feet is always about +90
-

# Injury to juglar vein

- Is life threatening because it can cause air embolism

# Hydrostatic pressure

- Pressure at the surface of water is equal to atm. press. & is zero mmHg.
- It changes by 1 mmHg for each 13.6 mm distance

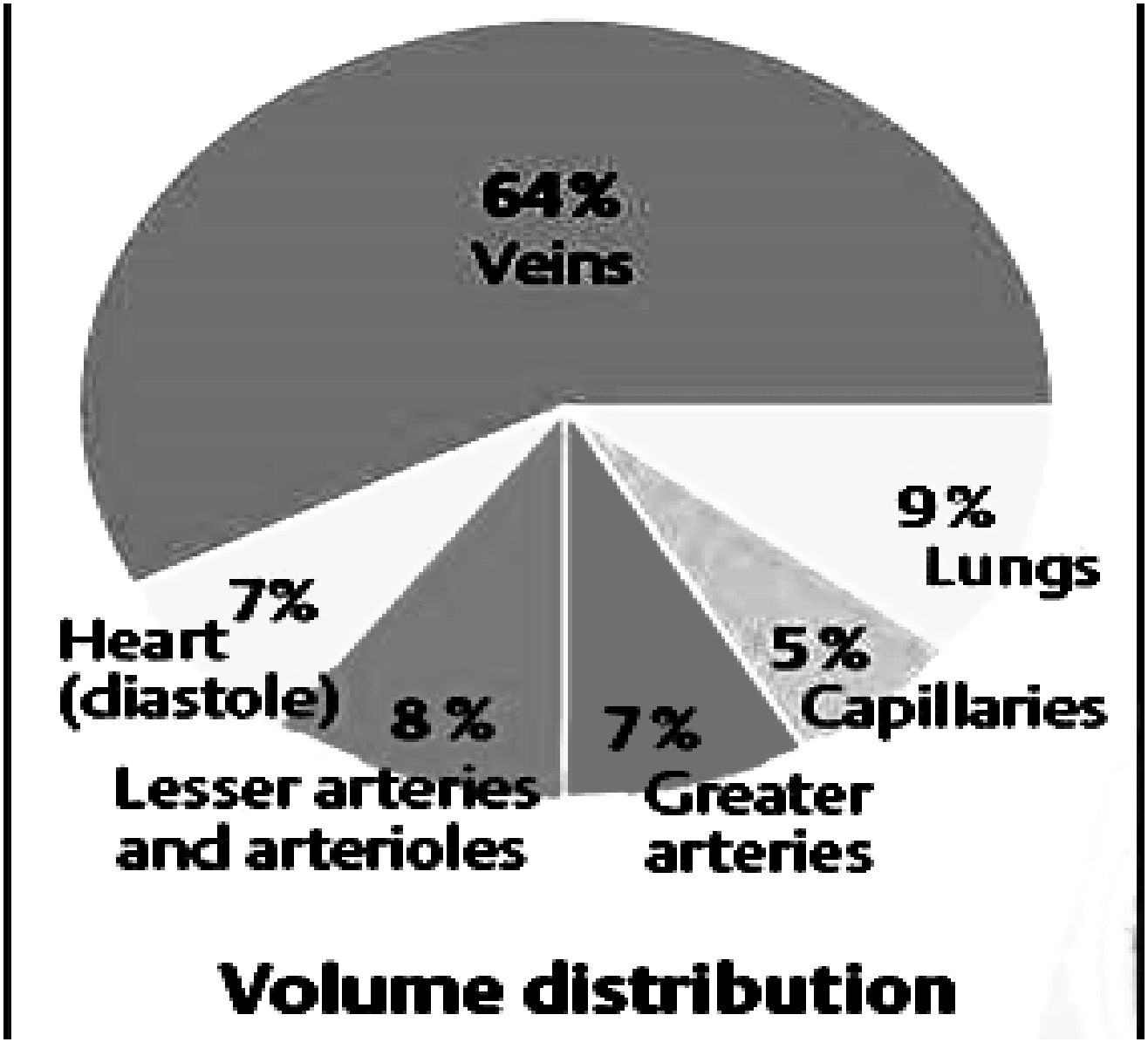


# Veins

- About 60 % of total blood is present in veins & they serve as **blood reservoir**

**Specific blood reservoirs are**

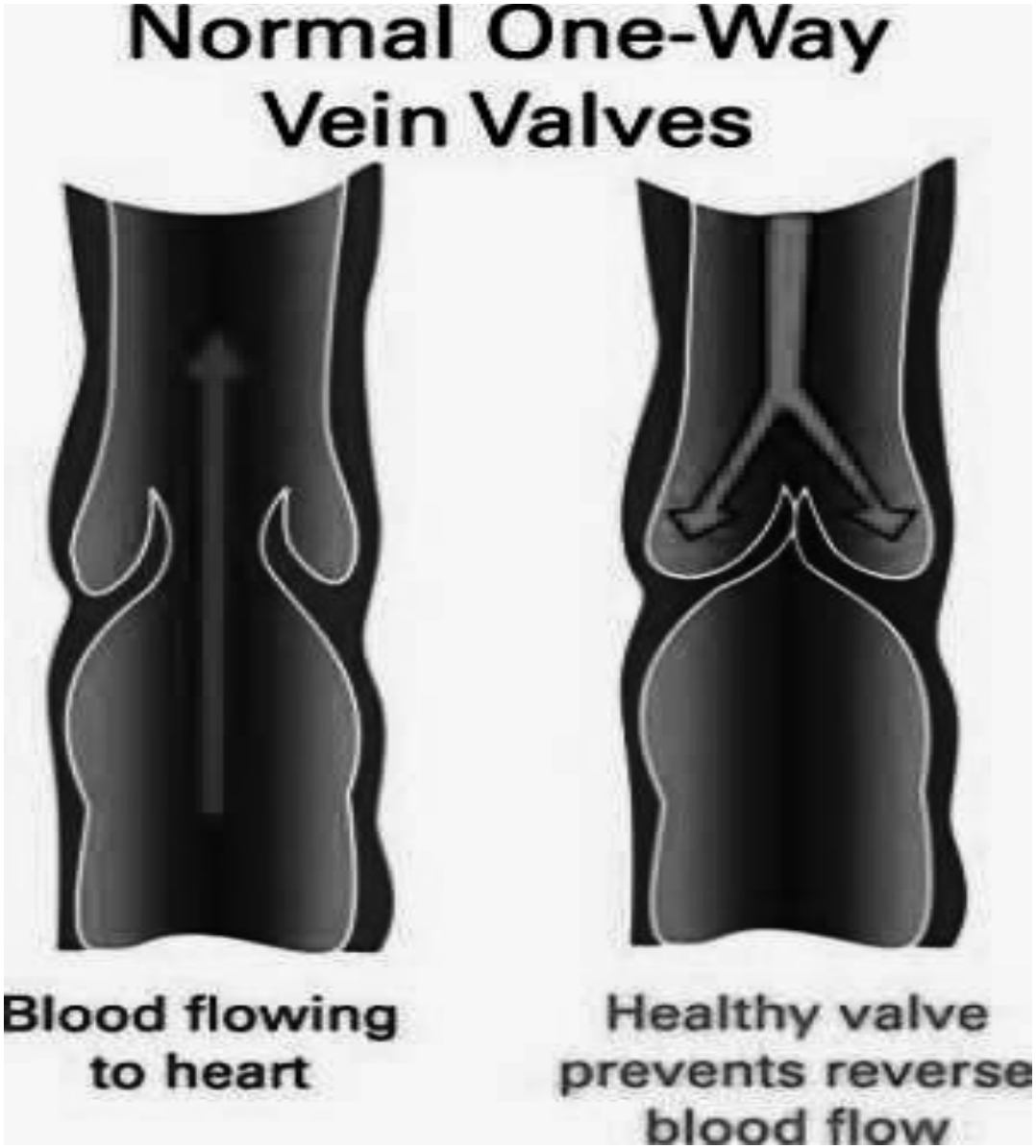
- Liver sinuses
- Large abdominal veins
- Venous plexus beneath the skin & spleen




**Volume distribution**

# Function of valves in the veins

- Venous pressure in feet is ~90 mmHg.
- Movement of legs & muscle contraction (muscle pump) squeeze the blood out of veins
- During walking pressure is less than 25
- Valves present in veins of limbs ensure that direction is only towards heart.



# ❑Varicose veins

- Valves become incompetent
- Large bulbous protrusions of the veins develop. These are varicose veins

Causes:

- Pregnancy