MICROCIRCULATION AND THE VENOUS SYSTEM

DR\DINA MONIR

ILOS

- DESCRIBE CAPILLARY BLOOD FLOW AND PRESSURE AND FACTORS AFFECTING/REGULATING THEM,
- CAPILLARY FRAGILITY, PULSATION.
- 2. ILLUSTRATE HOW THE WATER MOVES ACROSS THE CAPILLARY WALL.
- 3. DEMONSTRATE THE MECHANISMS OF PULMONARY AND SYSTEMIC OEDEMA.
- 4. DEFINE VENOUS PRESSURE, FACTORS THAT INFLUENCE IT.

INTRODUCTION

- CAPILLARIES CONTAIN **5%** OF BLOOD
- THEY ARE **EXCHANGE VESSELS**
- THEY ARE **10 BILLION** CAPILLARIES
- DIAMETER 10 UM
- LENGTH 700 UM
- SURFACE AREA 100 M_2
- CAPILLARY WALL IS FORMED OF: SINGLE LAYER OF ENDOTHELIUM RESTING ON A THIN BASEMENT MEMBRANE WITH PORES (FENESTRATIONS 80-90 A) IN DIAMETER.


100 SQUARE METRE
NEW BUILD TOUR

WHY CAPILLARY BLOOD FLOW IS INTERMITTENT?

- DUE TO VASOMOTOR INTERMITTENT CONTRACTION OF ARTERIOLES AND PRECAPILLARY SPHINCTER 6 -12 TIMES/MIN.

NB: IF ALL CAPILLARIES ARE OPENED AT ONE TIME (AS IN BURNS), CVS CAPACITY WILL EXCEED BLOOD VOLUME CAUSING MARKED DECREASE IN ABP IN CIRCULATORY SHOCK.

VELOCITY OF BLOOD FLOW IN CAPILLARIES

- VELOCITY = 0.5 MM/SEC

$$V = \frac{F}{R}$$

- THE CAPILLARIES HAVE THE LARGEST CROSS-SECTIONAL AREA → THE LEAST VELOCITY

CAPILLARY FRAGILITY

LAPLACE LAW:

- T= TENSION
- P= PRESSURE
- R= RADIUS

$$P = T \times R$$

ACCORDING TO LAPLACE LAW: - CAPILLARIES CAN WITHSTAND A PRESSURE OF 100 MMHG

- (ALTHOUGH THEIR WALLS ARE VERY THIN)→ AS THEY HAVE VERY SMALL DIAMETER →

SO TENSION IS NOT MARKEDLY INCREASED

CAUSES OF CAPILLARY FRAGILITY

1. **CAPILLARY WALL DEFECT**: VITAMIN C DEFICIENCY, SENILITY, ALLERGY, TOXINS.

1. **BLOOD DISEASES**: THROMBOCYTOPENIC PURPURA.

- **CLINICAL APPLICATION**

SCURVY (↓ VIT. C) BLEEDING AS RESULTS IN BLEEDING GUMS

CAPILLARY REACTIONS TO MECHANICAL INFLUENCES:

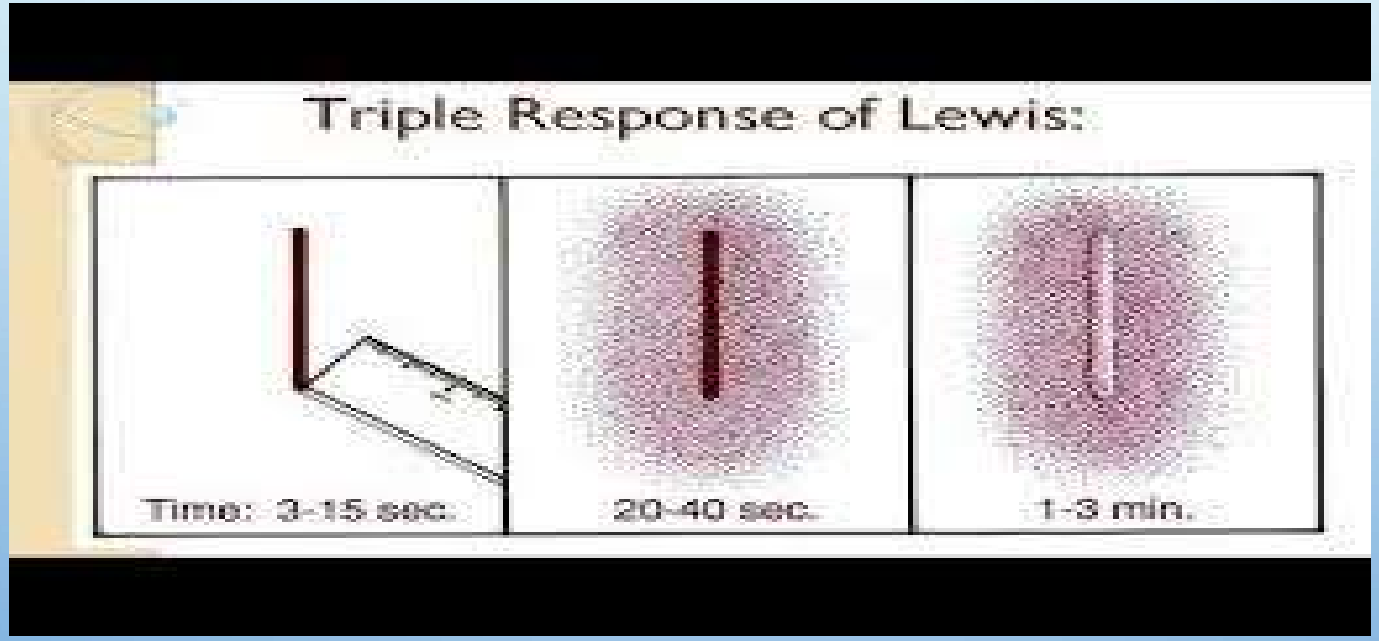
A- **WHITE LINE**: GENTLE STROKING OF SKIN BY A BLUNT OBJECT → WHITE LINE FOR FEW MINUTES, CAUSED BY VC OF CAPILLARIES

B- **TRIPLE RESPONSE**: FIRM STROKING OF SKIN →

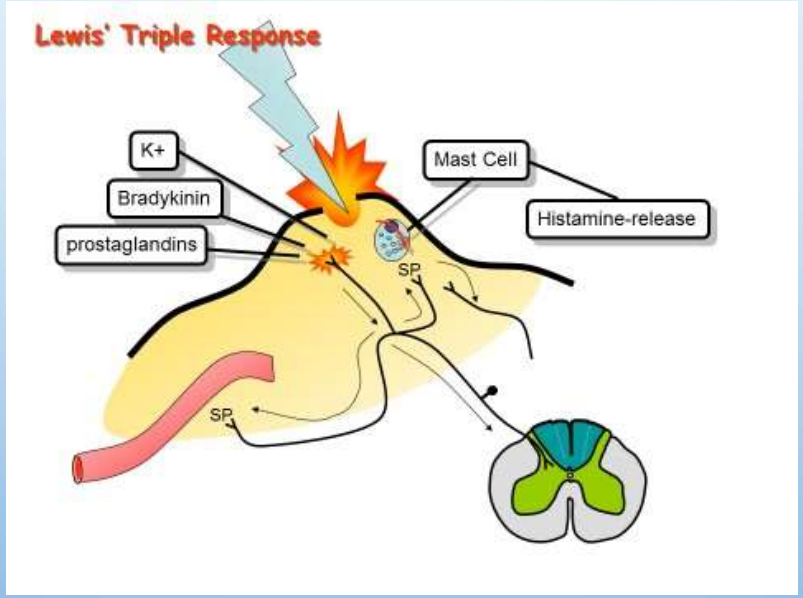
1. **RED LINE**: AT SITE OF INJURY DUE TO CAPILLARY VD BY HISTAMINE
2. **SPREADING FLARE**: AROUND RED LINE DUE TO ARTERIOLAR VD BY ANTIDROMIC RESPONSE
3. **LOCAL EDEMA (WHEAL):** DUE TO INCREASE CAPILLARY PERMEABILITY DUE TO HISTAMINE RELEASE

- IT OCCURS DUE TO INFLAMMATION + INSECT BITES

THE TRIPLE RESPONSE



THE TRIPLE RESPONSE



EQUILIBRIUM WITH INTERSTITIAL FLUID

1- **DIFFUSION**: - QUANTITATIVELY, IT IS MORE IMPORTANT THAN FILTRATION

- IT IS PASSIVE

- OCCURS IN BOTH DIRECTIONS

- IT IS CONCERNED WITH H2O & DISSOLVED SUBSTANCES

2- **FILTRATION** - BULK FLOW ACROSS CAPILLARY WALL

- IT IS A PASSIVE PROCESS

- IT IS THE BULK TRANSPORT OF H2O, ELECTROLYTES & CRYSTALLOID

FACTORS AFFECTING DIFFUSION

- **FACTORS IN THE SUBSTANCE**:

1. CONCENTRATION GRADIENT
2. LIPID (MEMBRANE) AND WATER SOLUBILITY (PORES)
3. MOLECULAR WEIGHT

- **FACTORS IN CAPILLARY PERMEABILITY**
- LIVER CAPILLARIES, (LARGE FENESTRAE, INTERRUPTED BASEMENT MEMBRANE FILTRATION.
- MUSCLE, SKIN & HEART, LUNG CAPILLARY: NO FENESTRATION +BASEMENT MEMBRANE →LOW PERMEABILITY.
- KIDNEY& INTESTINE CAPILLARIES→ MODERATE PERMEABILITY

FACTORS AFFECTING FILTRATION

MEAN FORCES TENDING TO MOVE FLUID OUTWARDS:

1- **HYDROSTATIC CAPILLARY PRESSURE = 35 MMHG** AT ARTERIOLAR END, 15 MMHG AT VENULAR END.

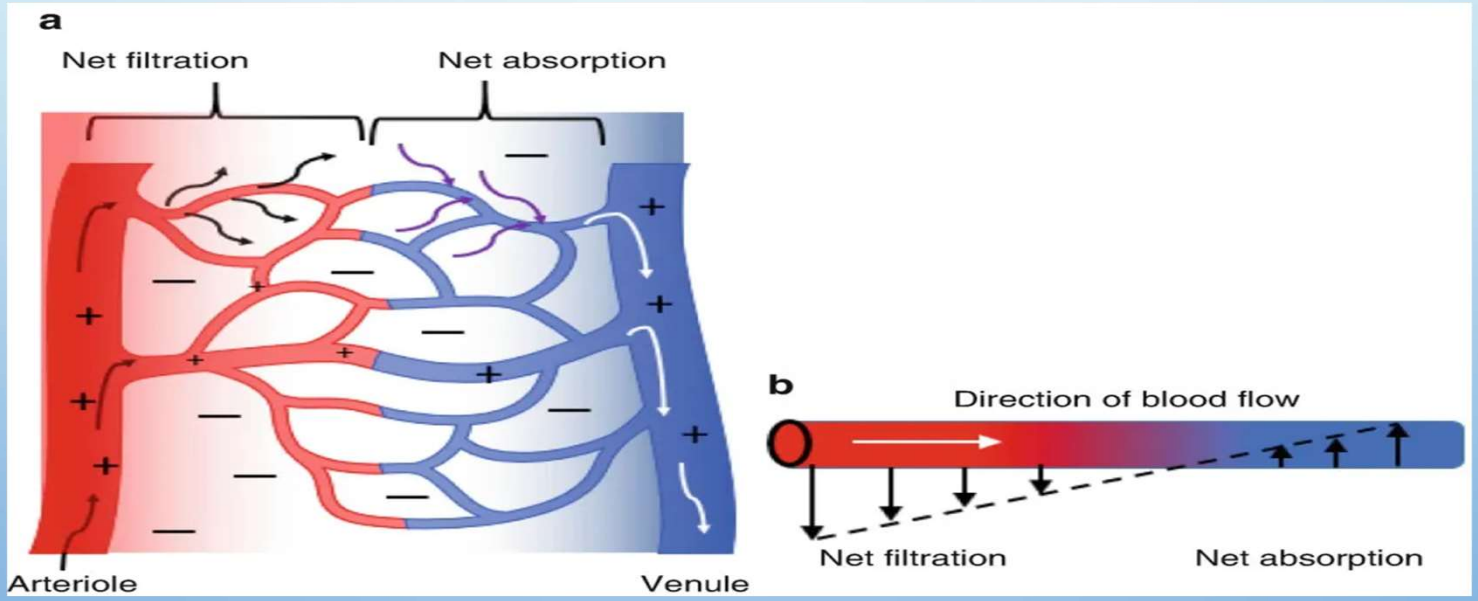
2- INTERSTITIAL FLUID COLLOIDAL OP = 3 MMHG (CAUSED BY 1% ALBUMIN)

MEAN FORCES TENDING TO MOVE FLUID INWARDS:

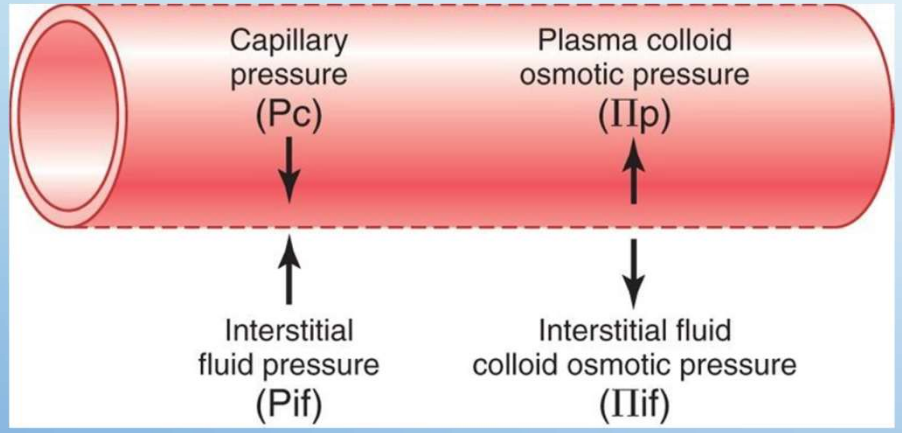
1- **COLLOIDAL OSMOTIC PRESSURE OF PLASMA PROTEIN = 25 MMHG** (MAINLY ALBUMIN)

2- HYDROSTATIC PRESSURE OF INTERSTITIAL FLUID = 3 MMHG

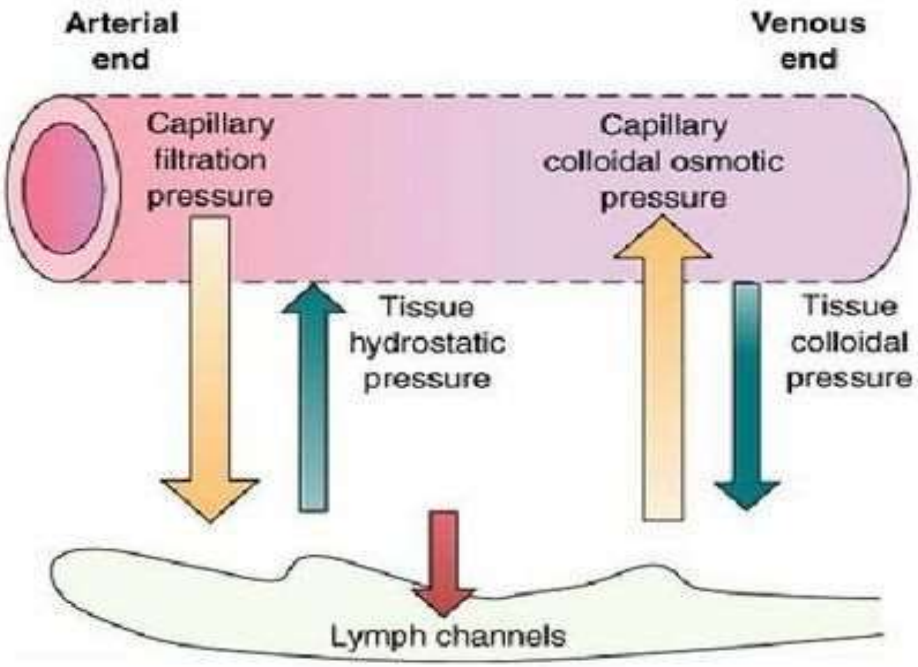
FILTRATION & REABSORPTION AT THE CAPILLARIES



EXCHANGE AT THE CAPILLARY



- AT ARTERIOLAR END → FILTRATION OF FLUID FROM BLOOD TO TISSUES (BULK FLOW)
- AT VENULAR END → REABSORPTION OF A NEARLY EQUAL AMOUNT OF FLUID INTO BLOOD
- THE REMAINING IS FLUID IS TAKEN BY LYMPHATICS)


Arterial end
Venous end
Capillary filtration pressure
Capillary colloidal osmotic pressure
Tissue hydrostatic pressure
Tissue colloidal pressure
Lymph channels

VARIATION IN BULK FLOW(FILTERATION)

1- **DECREASES COLLOIDAL OP OF PLASMA PROTEIN**: AS IN LIVER & KIDNEY DISEASES → INCREASES FILTRATION

2- **INCREASE CAPILLARY HYDROSTATIC PRESSURE AS IN VENOUS OBSTRUCTION** → INCREASE FILTRATION

3- **INCREASE CAPILLARY PERMEABILITY** AS IN ACTIVITY, INFLAMMATION → ALBUMIN GOES OUT → INCREASE FILTRATION

EDEMA

- **DEFINITION**: IS EXCESSIVE ACCUMULATION OF INTERSTITIAL FLUID IN ABNORMAL LARGE AMOUNT.
- CAUSE:

1- **INCREASE CAPILLARY HYDROSTATIC PRESSURE**:

- RIGHT SIDE HF OR CONGESTIVE HEART FAILURE.
- OBSTRUCTION OF VEIN BY THROMBUS OR TUMOR.
- INCOMPETENT VALVES OF VEIN AS IN VARICOSE VEIN.
- COMPRESSION ON VEINS (AS IN PREGNANCY).
- PORTAL HYPERTENSION.

CAUSES OF EDEMA

2- SALT & WATER RETENTION

1. INCREASE ALDOSTERONE

2. INCREASE ADH

3- DECREASE PLASMA OSMOTIC PRESSURE:

1. LIVER DISEASE: DECREASE HEPATIC SYNTHESIS OF PLASMA PROTEINS .

2. KIDNEY DISEASE: INCREASE LOSS OF PLASMA PROTEIN IN URINE

CAUSES OF EDEMA

4- INCREASE OF CAPILLARY PERMEABILITY:

1. ALLERGIC CONDITIONS
2. INFLAMMATORY CONDITIONS (KININS & HISTAMINE SUBSTANCE)

5- LYMPHATIC OBSTRUCTION:

1. FILARIASIS
2. TUMOR COMPRESS ON LYMPHATICS.

VENOUS CIRCULATION& VENOUS PRESSURE

- **CENTRAL VENOUS PRESSURE (CVP): IT IS THE PRESSURE IN RIGHT ATRIUM & VEINS OPENING INTO IT**
- **VALUE: 0 - 2 MMHG** (YET, IT FLUCTUATES WITH RESPIRATION AND CARDIAC CYCLE)
- -**MEASUREMENT**: **1-DIRECT(CATHETER)**
- **2. CLINICALLY:** BY OBSERVING THE DEGREE OF FILLING OF EXTERNAL JUGULAR VEIN WHILE THE PATIENT IS IN A SEMI SITTING POSITION
- NORMAL: IT DOES NOT FILL ABOVE LEVEL OF ANGLE OF LOUIS

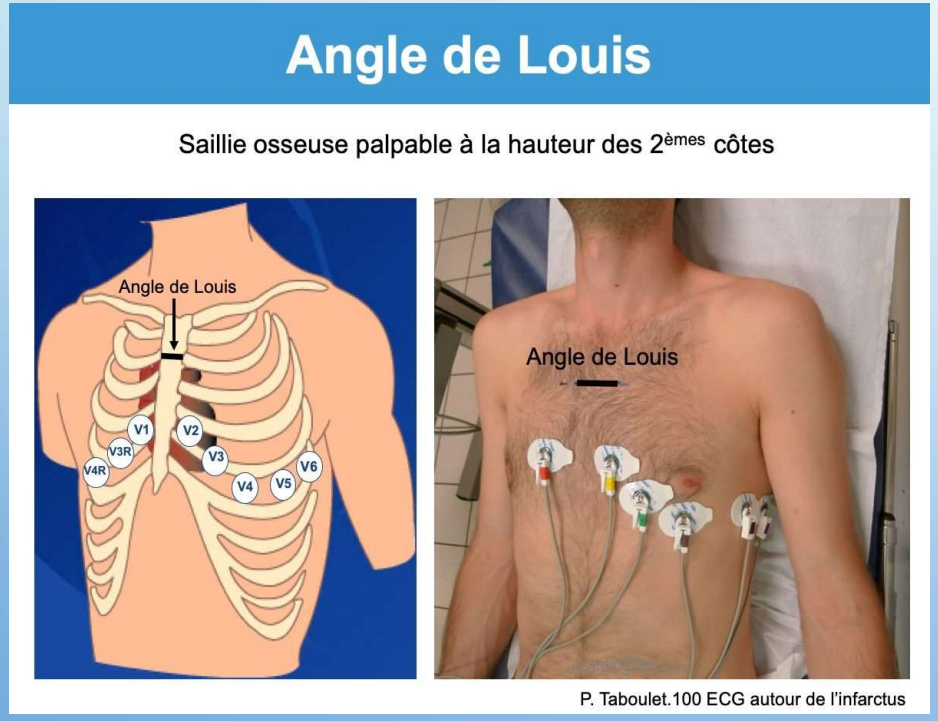
IMPORTANCE: 1. IT IS AN INDEX OF BLOOD VOLUME

2. IT IS THE CARDIAC FILLING PRESSURE.

VARIATION:

1. CVP DECREASES IN: HEMORRHAGE
- 2. CVP INCREASES IN: HEART FAILURE

WHAT IS THE ANGLR OF LOUIS



- THE ANGLE OF LOUIS, ALSO KNOWN AS THE STERNAL ANGLE, WHICH IS THE JOINT WHERE THE MANUBRIUM (UPPER PART) AND THE BODY OF THE STERNUM (BREASTBONE) MEET.

FUNCTIONS OF VEINS:

- CAPACITANCE VESSELS (BLOOD RESERVOIR)

$$C = \frac{\Delta v}{\Delta p}$$

- CAPACITY OF VENOUS SYSTEM = 200 ML/MMHG
- ARTERIAL SYSTEM CAPACITY = 1 ML/ MMHG
- DURING EXERCISE, A SMALL VOLUME OF BLOOD IS SHIFTED FROM VENOUS TO ARTERIAL
- SYSTEM RESULTING IN: 1) MARKED RISE IN ABP (SMALL CAPACITY)

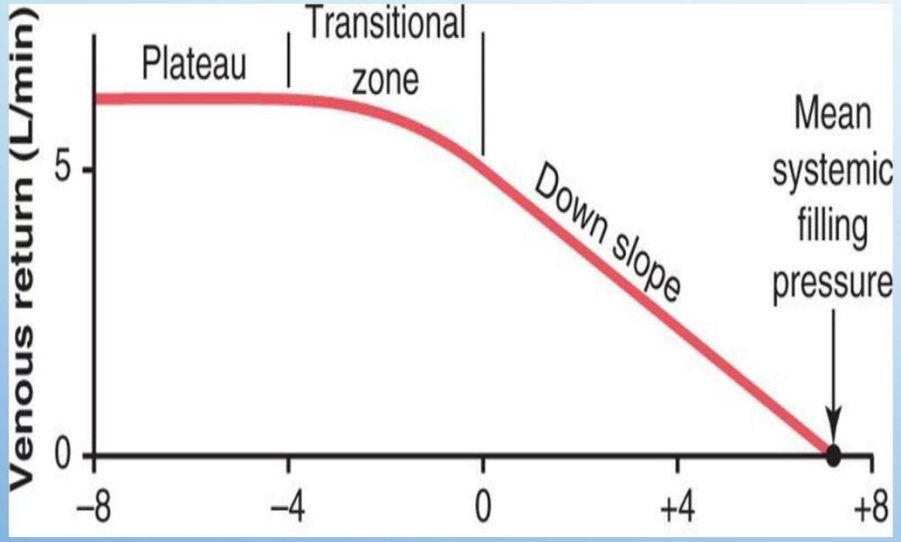
2) MINIMAL CHANGE IN CVP (LARGE CAPACITY) WHICH IS THE CARDIAC FILLING PRESSURE IE CARDIAC FILLING IS UNAFFECTED

VENOUS RETURN

- IT IS BLOOD FLOW THAT RETURN TO THE HEART IN MINUTE
- IT IS NORMALLY = CARDIAC OUTPUT = 5L/MIN.
- ***DIRECTLY PROPORTIONAL*** TO PRESSURE GRADIENT BETWEEN MSFP &RAP
- ***INVERSELY PROPORTIONAL*** TO RESISTANCE IN VENOUS SYSTEM

- **MSFP**: "**MEAN SYSTEMIC FILLING PRESSURE**" IT IS PRESSURE IN CIRCULATION IF PUMPING ACTION OF HEART STOP- EQUAL 6-8 MMHG.
- **RAP:** "**RIGHT ATRIAL PRESSURE**" IT IS PRESSURE IN RIGHT ATRIUM- IT EQUAL 0-2 MMHG.
- **RESISTANCE IN VENOUS SYSTEM CAN BE CALCULATED BY: R= Δ P/F = MSFP-RAP/CO = 7-0/5 = 1.4 MMHG /LITER/ MIN**

RAP (RIGHT ATRIAL PRESSURE)



- NORMAL VENOUS RETURN CURVE. THE PLATEAU IS CAUSED BY COLLAPSE OF THE LARGE VEINS→ ENTERING THE CHEST WHEN THE RAP (RIGHT ATRIAL PRESSURE) FALLS BELOW ATMOSPHERIC
- PRESSURE. NOTE ALSO THAT VENOUS RETURN BECOMES ZERO WHEN THE RAP RISES TO EQUAL THE MSFP (MEAN SYSTEMIC FILLING PRESSURE)

FACTORS HELP VENOUS RETURN AGAINST GRAVITY

1- CONTRACTION OF VOLUNTARY MUSCLE.

2- PRESSURE GRADIENT BETWEEN MSFP & RAP.

3- ARTERIAL PULSATION: PRESS ON NEARBY VEIN, HELPING VR.

4- DIAMETER OF ARTERIOLES: VASOCONSTRICTION INCREASE VR.

5- TONE OF VEINS: - VD OF VEINS DECREASE VR

- VC OF VEINS INCREASE VR

FACTORS HELP VENOUS RETURN AGAINST GRAVITY

6- TONE OF CAPILLARIES:- VD OF CAPILLARIES WILLS ↓ VR

- VC OF CAPILLARIES WILL ↑ VR

7- THORACIC PUMP: DURING INSPIRATION: IPP BECAME MORE NEGATIVE LEAD TO INCREASE VR

DURING EXPIRATION: IPP BECOME LESS NEGATIVE LEAD TO DECREASE VR

8- CARDIAC SUCTION:

I. SYSTOLIC SUCTION: OCCUR DURING: MAXIMUM EJECTION IN WHICH BLOOD EJECTED FROM VENTRICLE TO AORTA

II. DIASTOLIC SUCTION: OCCUR DURING: RAPID FILLING PHASE IN WHICH BLOOD PASS PASSIVELY FROM ATRIA TO VENTRICLE

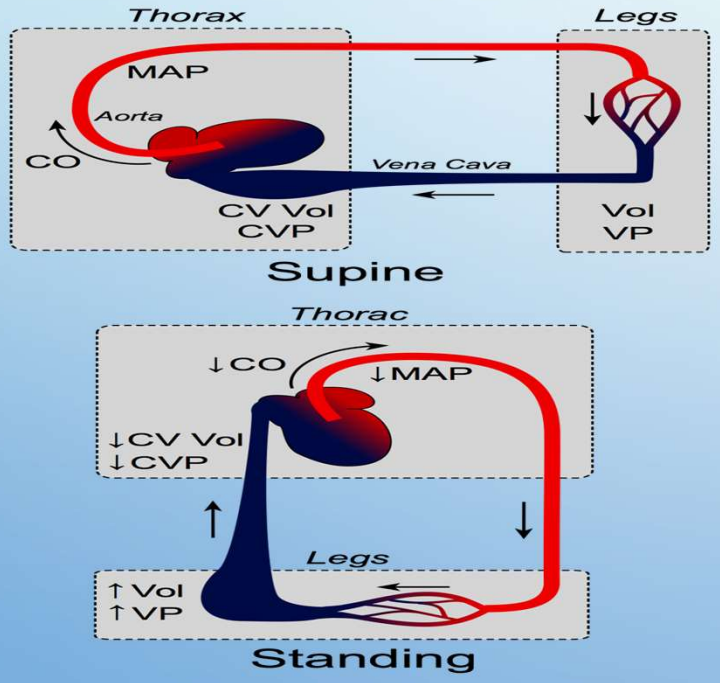
FACTORS HELP VENOUS RETURN AGAINST GRAVITY

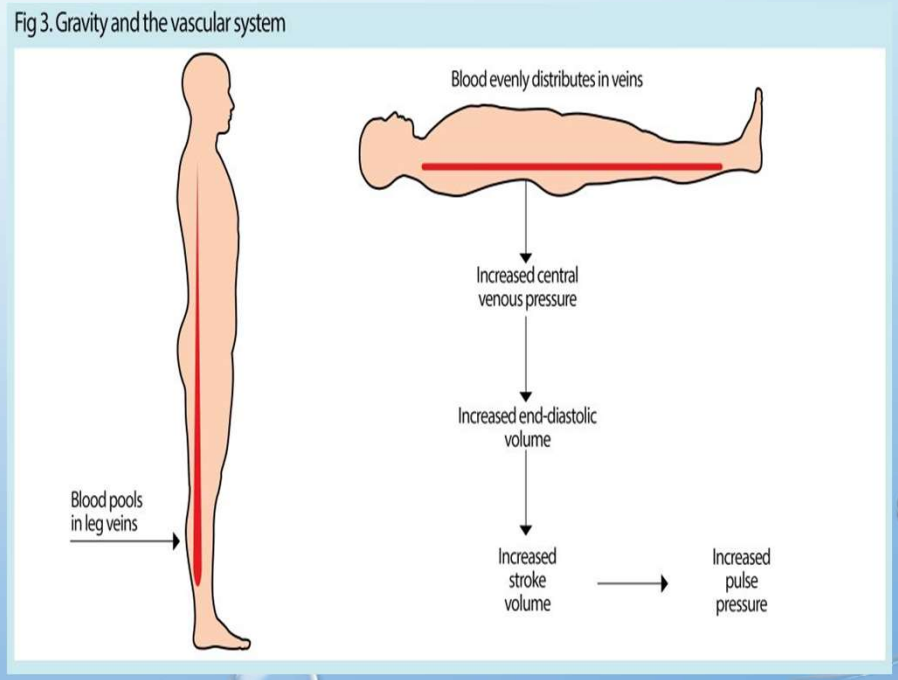
9- CONTRACTION OF SPLEEN: LEAD TO INCREASE BLOOD VOLUME & VR

10- GRAVITY: - ABOVE THE LEVEL OF HEART: GRAVITY HELPS VR

- BELOW THE LEVEL OF HEART: GRAVITY INTERFERES WITH VR

EFFECTS OF GRAVITY ON VENOUS RETURN





EFFECT OF GRAVITY ON VENOUS SYSTEM:

1- ORTHOSTATIC HYPOTENSION:

DURING ORTHOSTATIC (CHANGING THE POSITION FROM LYING DOWN TO ERECT)→ IF SIGNIFICANT AMOUNT OF BLOOD SHIFTS FROM THORACIC VEINS TO LOWER LIMB VEINS →

- DECREASE CVP, DECREASE CARDIAC FILLING → DECREASE CO → DECREASE ABP (IE ORTHOSTATIC HYPOTENSION)→ PERSON FEELS DIZZY
- **IN NORMAL PERSON:** COMPENSATORY MECHANISMS OCCUR RAPIDLY TO CORRECT THIS
- HYPOTENSION

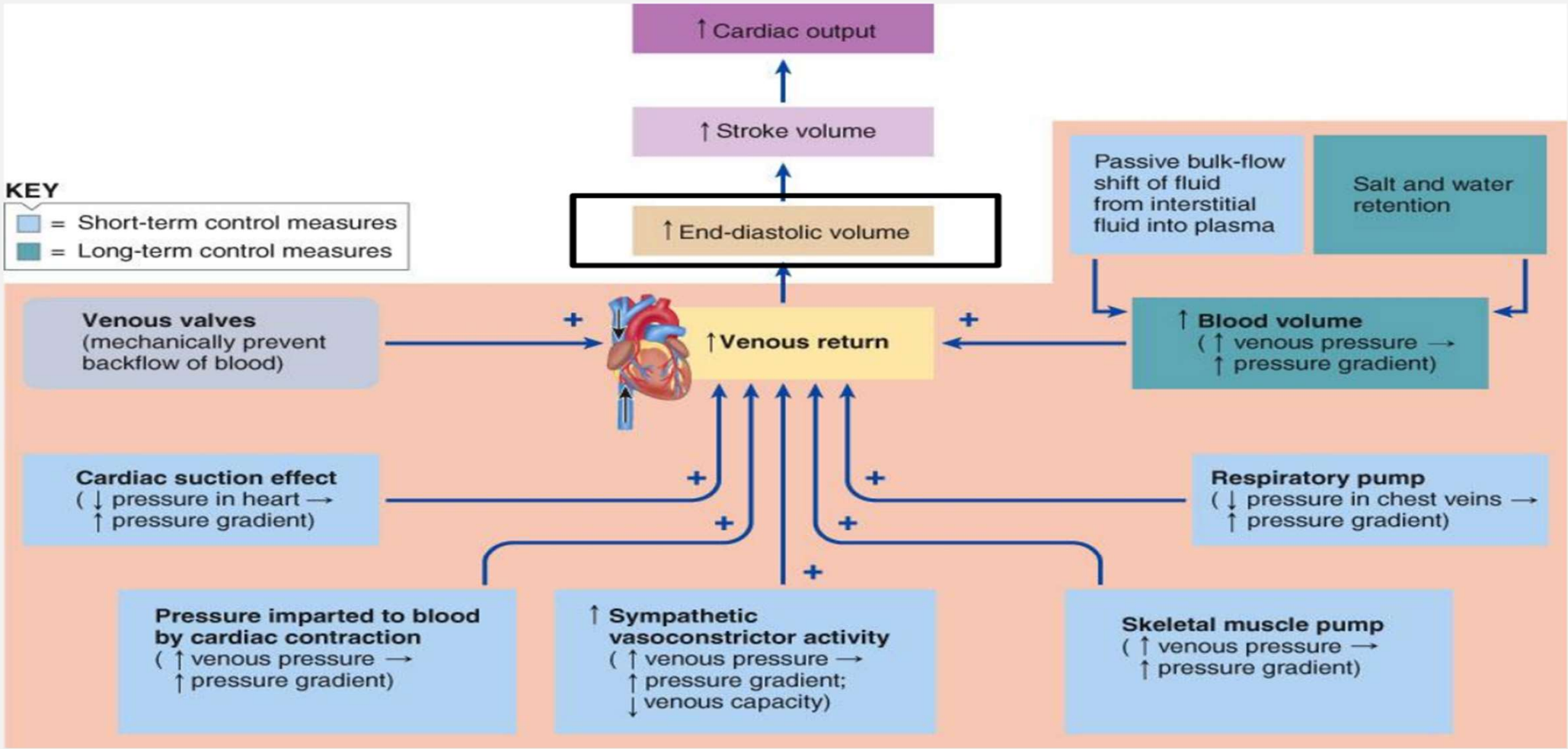
2- HYDROSTATIC INDIFFERENT POINT [HIP]:

- THERE MUST BE A TRANSITIONAL ZONE IN WHICH THE VENOUS PRESSURE REMAINS CONSTANT INDEPENDENT ON BODY POSTURE THIS ZONE IS CALLED HIP
- IT LIES 5 - 7 CM BELOW DIAPHRAGM
- **THE PRESSURE AT HIP = 10 - 11MMHG**
- -HIP POSITION IS RELATED TO CAPACITY OF THORACIC & LOWER LIMB VEINS.
- -IF THE BODY IS IMMERSED IN H2O (SWIMMING), THE LL (LOWER LIMBS) VEINS CAPACITY IS DECREASED → SHIFT OF HIP UPWARDS, LYING ALMOST AT THE LEVEL OF THE HEART → SO THE CVP DOES NOT VARY ON CHANGING POSITION → ORTHOSTATIC HYPOTENSION NOT OCCUR ON SWIMMING.

3- THE PRESSURE IN DIFFERENT VEINS:

- **THE PRESSURE IN LEG VEINS INCREASES FROM 20 TO 80 ON PROLONGED STANDING MMHG**, THUS ON THIS HIGH VENOUS PRESSURE RESULTS IN:
- VARICOSE VEINS (DILATATION & ELONGATION ON LOWER LIMB VEINS)
- EDEMA: DUE TO INCREASED. FILTRATION

Factors facilitating venous return to the right atrium




Thank
you