Physiology Summary

First Year

By

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Lecturer Physiology

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Cardiac

CVS د/ محمد فايز

Function

- Homeostasis
- Temperature regulation
- Communication

Characters of vessels

- **Aorta** → Elastic fibers
- **Arteriole** → Smooth muscles (wall)
- **Capillaries** → Thin wall
- **Veins**

Capacitance

$$C = \frac{\Delta V}{\Delta P}$$

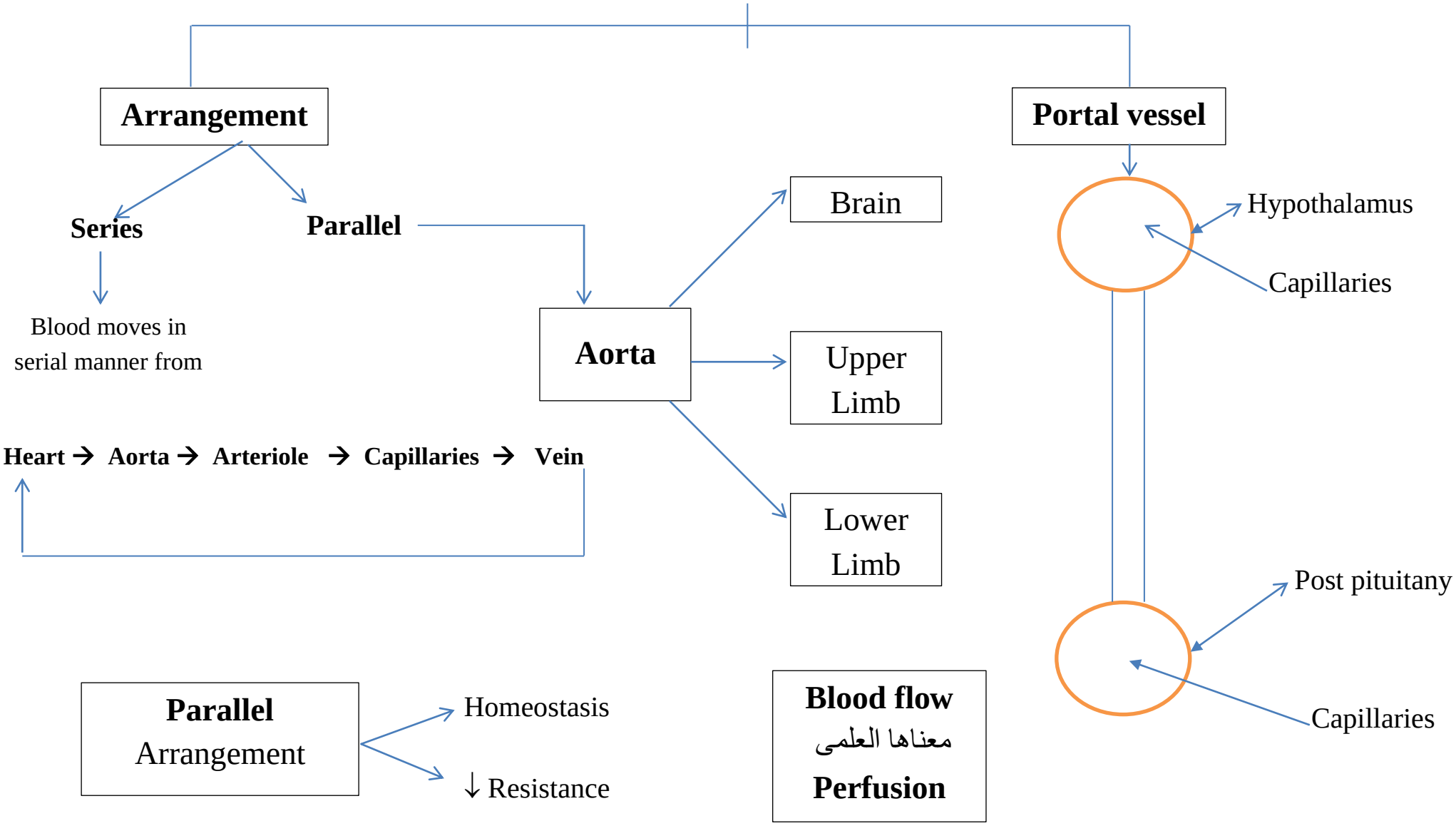
المعنى:
يمتلىء باكبر حجم دم
باقل ضغط ممكن

الدليل : 54% blood (vein)

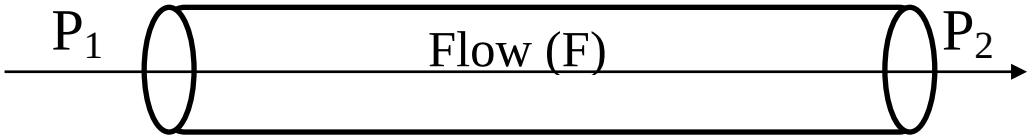
MCQ

- **Windkessel** Function
- **Resistance** → Stopcock (محبس)
- **Exchange**
- **Capacitance**

د. محمد فايز CVS



CVS د. محمد فايز



F = Δ P/R → Resistance

Intermittent
(LV → Aorta)

Types

Pulsatile → Capillaries

Steady → Veins

Velocity

Rapid → Aorta

Slow → Capill.

Velocity α 1/CSA

Vessels Aorta (**93** mmHg) → Arteriole (**32** mmHg) → Capillaries → veins (**10** mmHg) → Right atrium (**0** mmHg)

High Pressure
Ventricles (Systole)
+
A orta
rteries
rterioles
rterial side of capillaries

Low Pressure
Atria + العكس
Pulmonary circulation

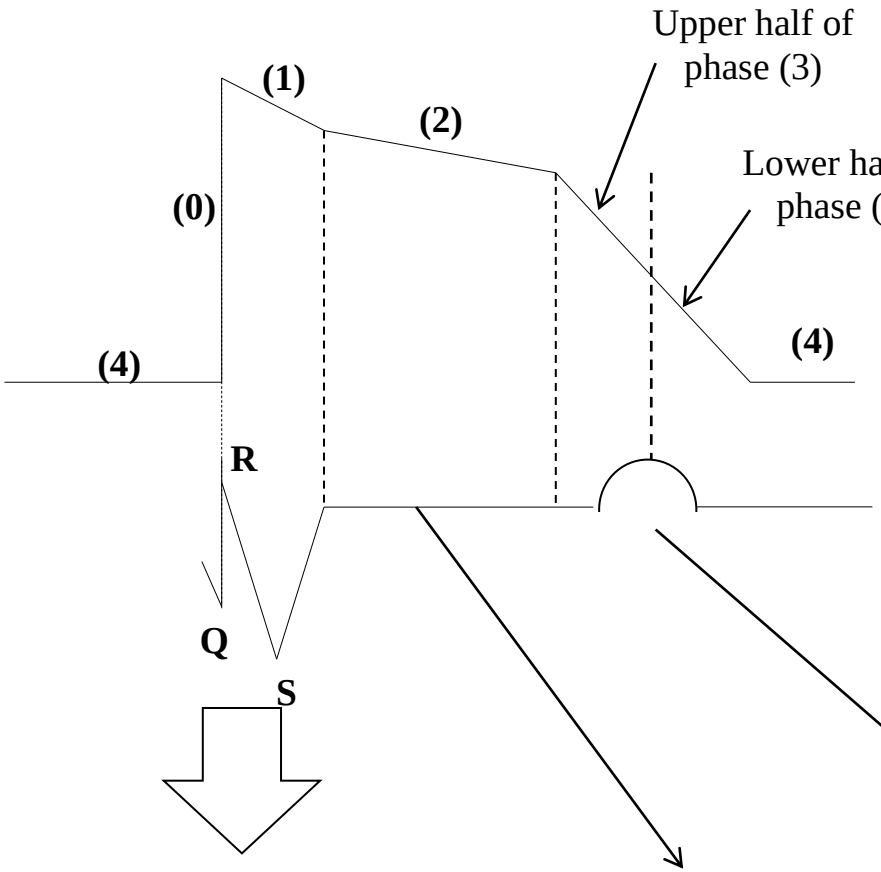
	Fast Response	Slow response
Phase 4	**Stable** ← RMP →	**Unstable** (Prepotential)
Cause	K^+ efflux > Na^+ influx	= Diastolic depolarization
		Early: Na^+ influx } > K^+ efflux
		Late: Ca^{++} influx (T-channels)
Value	-90 mV	- 55 mV
Phase 0	← Depolarisation →	
Cause	Na^+ influx (voltage gated Na^+ channels)	Ca^{++} influx (**L-type** Ca^{++} channels)
Firing level	- 70 mV (**Peak**: +30 mv)	- 45 mV (**Peak**: +10 mV)
Phase 1	Open voltage gated k^+channels	----------
Phase 2 (platau)	**Early**: ca^{++}Influx = K^+ efflux **Late:** Na^+ influx = K^+ efflux	----------
Phase 3	Open voltage gated k^+ channels	

Repolarisation

Refractory Periods دكتور/ محمد فايز

	Fast Response	Slow response
Refractoriness	Shorter	Longer → Start phase 4 (post repolar. Refractoriness)
ARP Time Significance	Phases (0, 1, 2, upper 1/2 (3)) Safety against tetanisation	Phase (0) → 2/3 phase (3) Voltage dependent refractoriness
ARP Time Significance	Lower 1/2 (phase 3) Partial recovery of fast Na^+ channels	Late 1/3 phase (3) → start phase (4) Pathological importance of AVN (2)
ERP Time Significance	ARP + 1st 10 mv of RRP → 60 mv As ARP + **cardioversion** (2000-3000 volt) in V.F **(الهدف الغائه حتى يرجع SAN لطبيعته)**	————
Supernormal period Time Significance		Short at end of phase (3), start phase (4) Propagated AP → ↑ excitability

Relation between Fast response, ECG د. محمد فايز



Vulnerable period

Lower 1/2 phase 3:

Ectopic ممكن ان يظهر فيها

(Some fibers depolar./others repolar)

QRS complex
Coincide with
phase 0 "**Depolar**."

S-T Segment
Coincide with
phase 2 "**Plateau**"

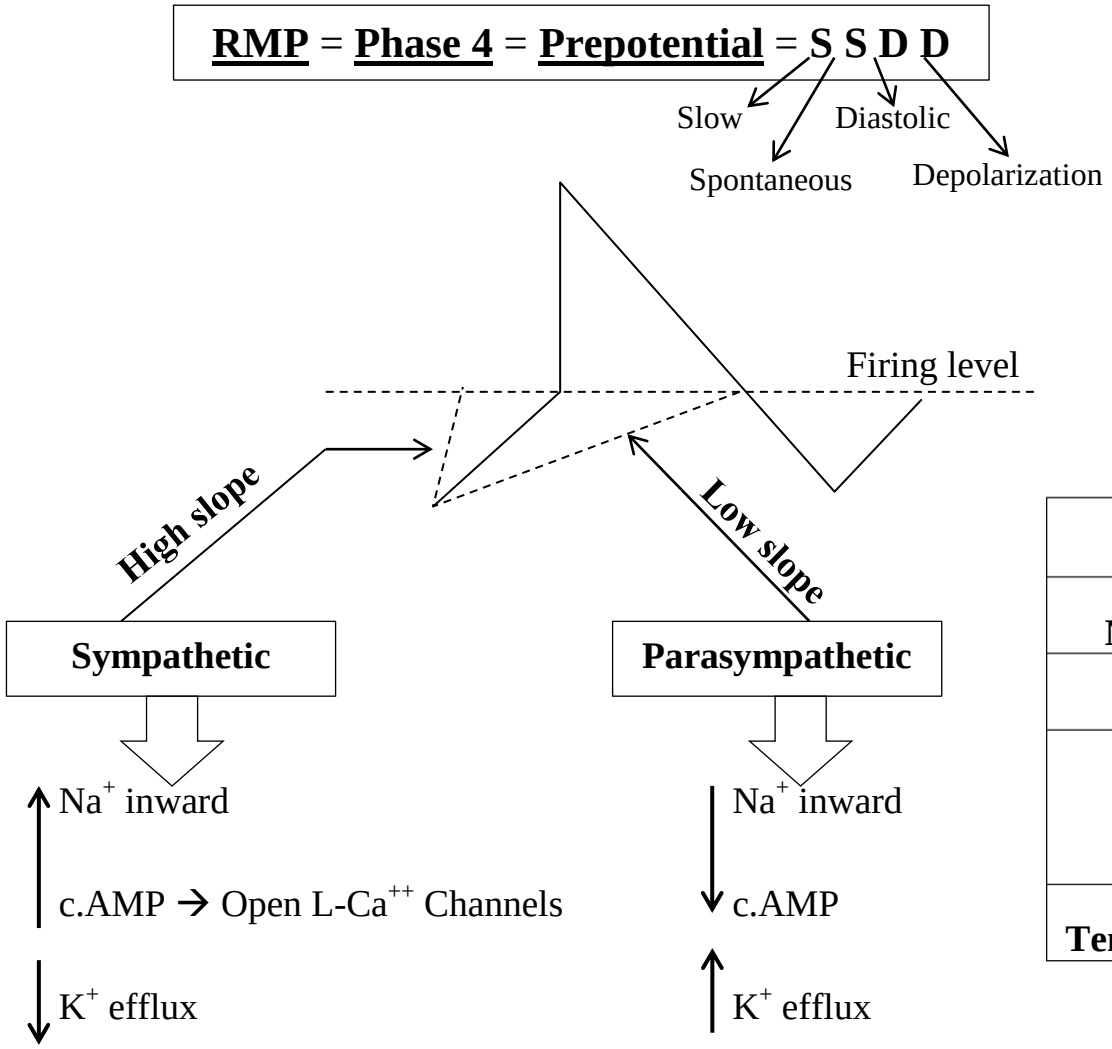
T-wave

Upper 1/2 Lower 1/2

Coincide with

Upper 1/2 phase (3) Lower 1/2 phase (3)

SAN د. محمد فايز



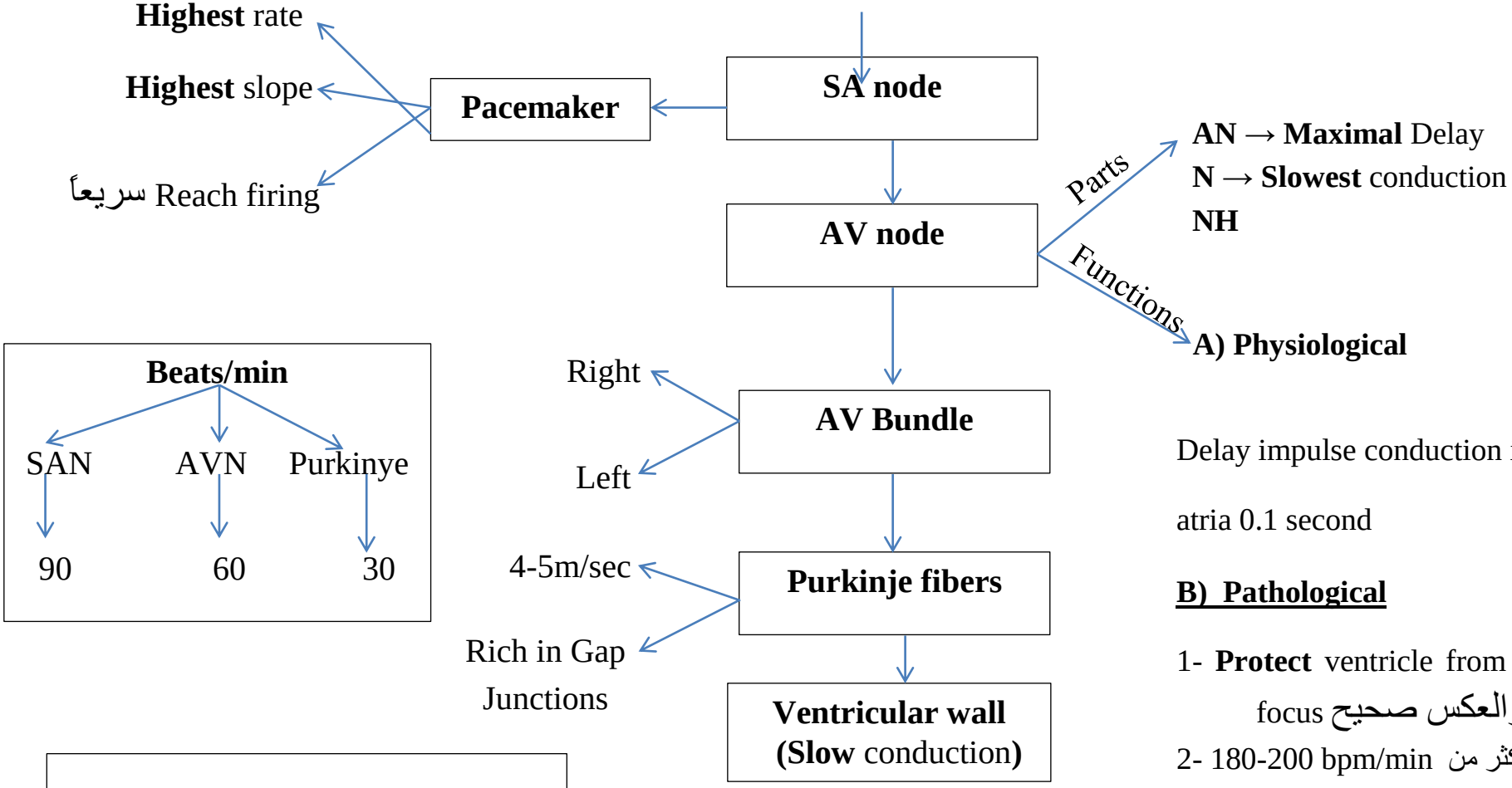
SAN is Normal Pacemaker

Highest Slope → Discharge → Repolarized

Low Amplitude

	↑ SAN firing	↓ SAN firing
Nervous	Sympathetic	Parasymp.
Ions	Hypocalcemia	Hyperkalemia calcemia
Drug	Adrenaline Atropine	Muscarine β-Blockers
Temperature	Warming	Cooling

Conductivity



Delay impulse conduction in

atria 0.1 second

B) Pathological

1- **Protect** ventricle from atrial ectopic focus والعكس صحيح

2- 180-200 bpm/min لا يسمح بمرور أكثر من

(Voltage, time refractoriness)

3- Post repolarization refractoriness

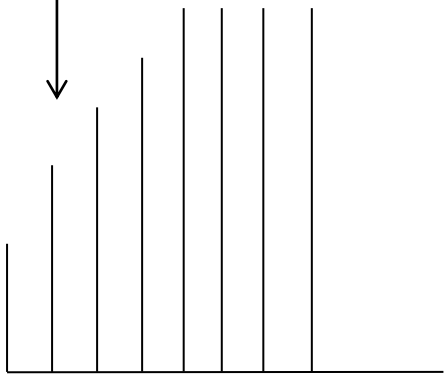
Interventricular septum

1st part to be depolarised in ventricle

Base of LV: Last depolarised

Factors Affecting Contractility د. محمد فايز

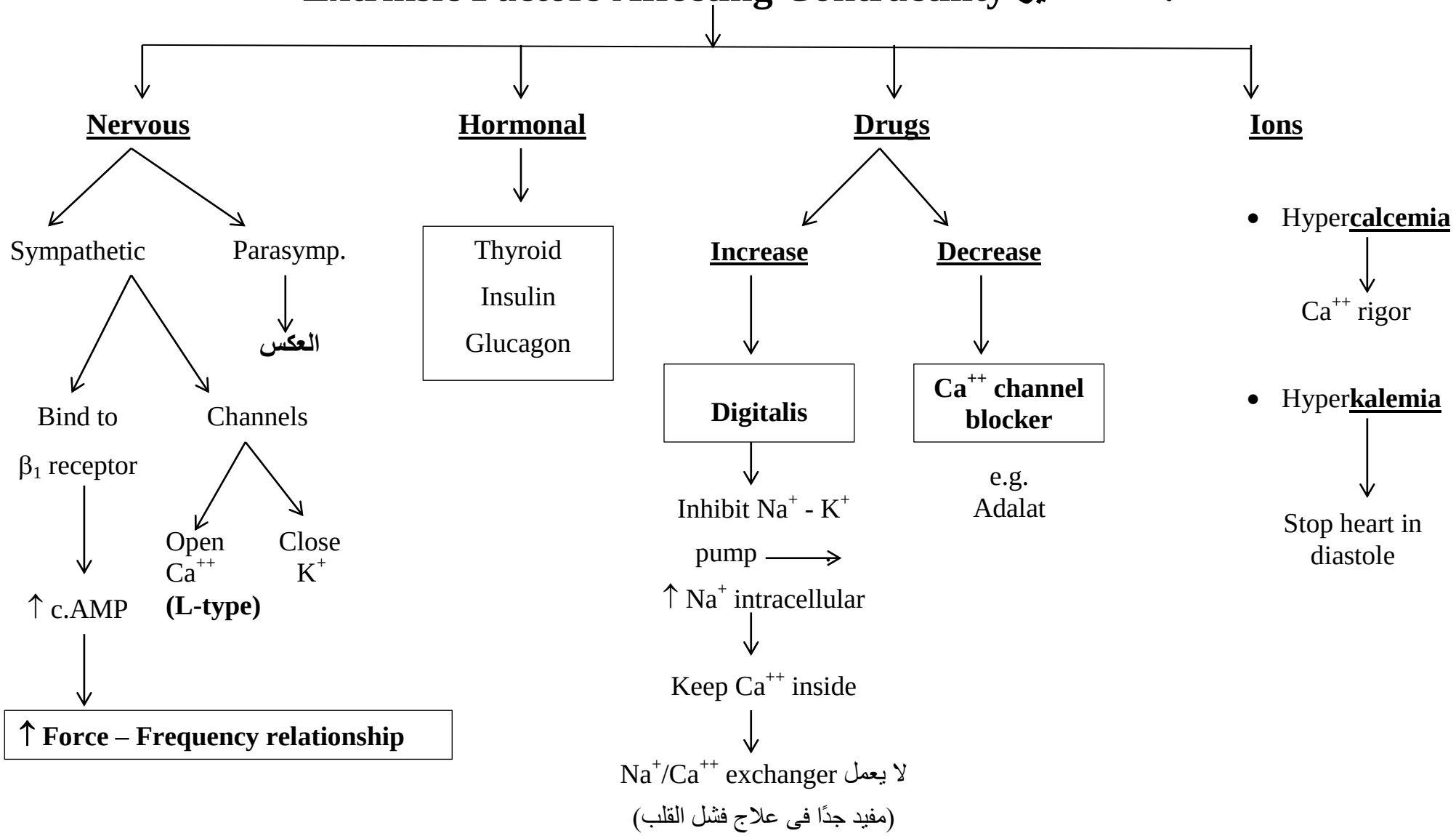
Intrinsic

- **Preload**
  - **Def**.: القلب يمتلىء بالدم قبل Contr.
  - **Starling law** أذكر
  - **Parallel** elastic element
    - Redundant (Stretch يقاوم)
    - **Muscle developed tension** = S —— D
- **Afterload**
  - **Def**.: Resistance Vs which blood is expelled
  - **Faced by**
    - Iso metric
    - Iso tonic
  - ⟵ Cont. ⟶
- **Force frequency**
  - **Stair-case phenomena**
  - 
- **Inotropic state**
  - Healthy myocardium
  - Intracellular Ca^{++}

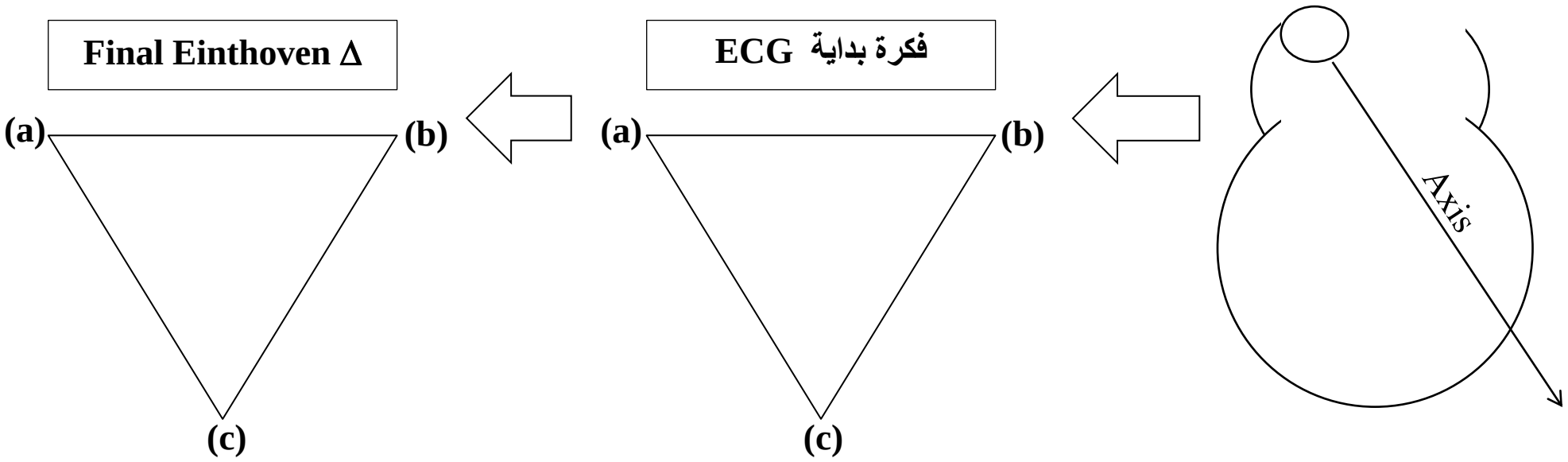
Extrinsic

أنظر الصفحة القادمة

Extrinsic Factors Affecting Contractility د. محمد فايز



Einthoven Triangle د. محمد فايز



أساس فكرة رسم القلب:

Axis of heart is directed downward, to left

∴ Einthoven Δ

معظم الوصلات ناحية الشمال

يمكن تسجيل كهرباء الناتجة من القلب

لانه يقع على ابعاد متساوية من ٣ نقاط:

a) Rt shoulder.

b) Lt shoulder.

c) Symphysis pubis.

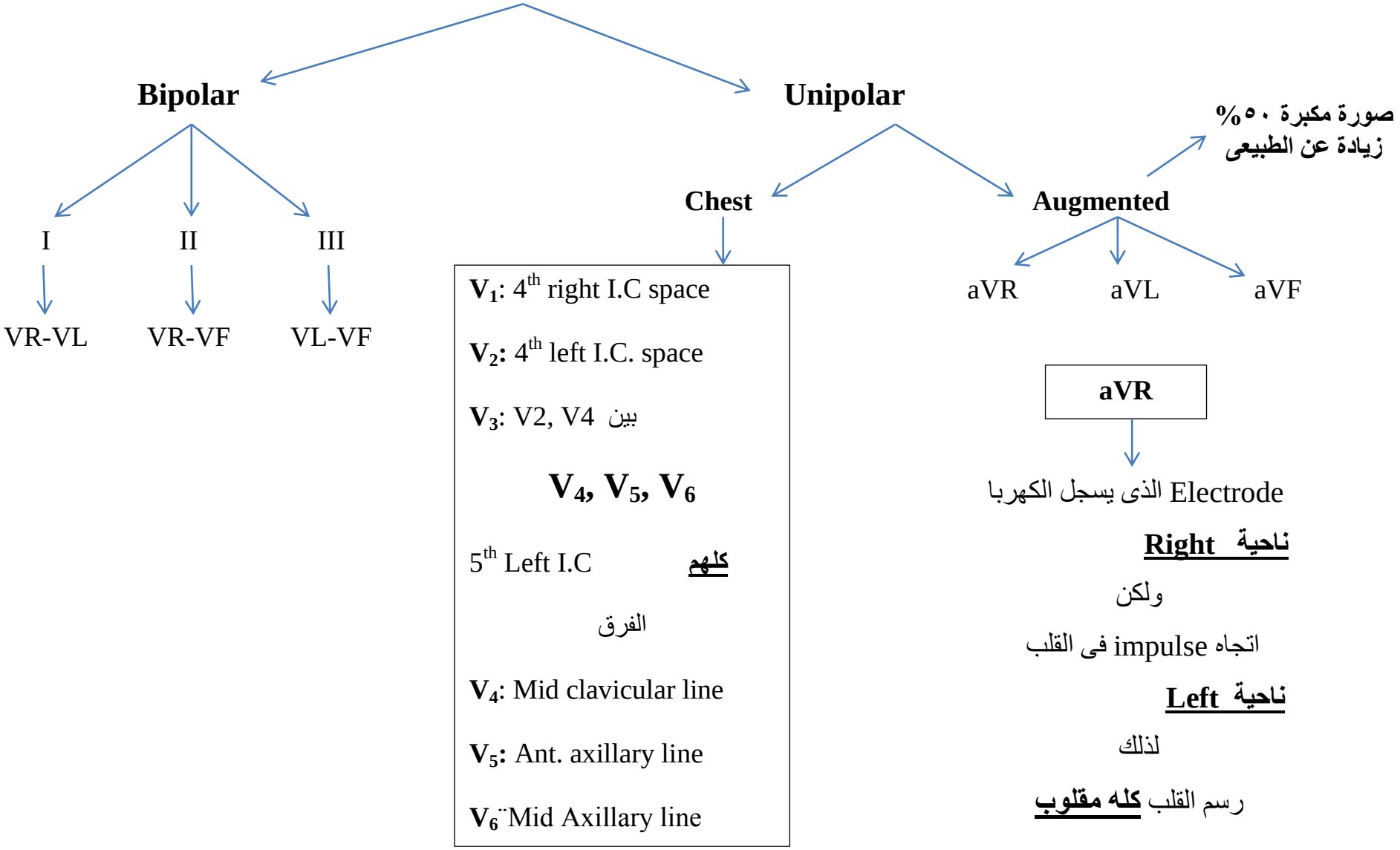
تم تغيير ٣ نقاط

∴ Final Einthoven Δ:

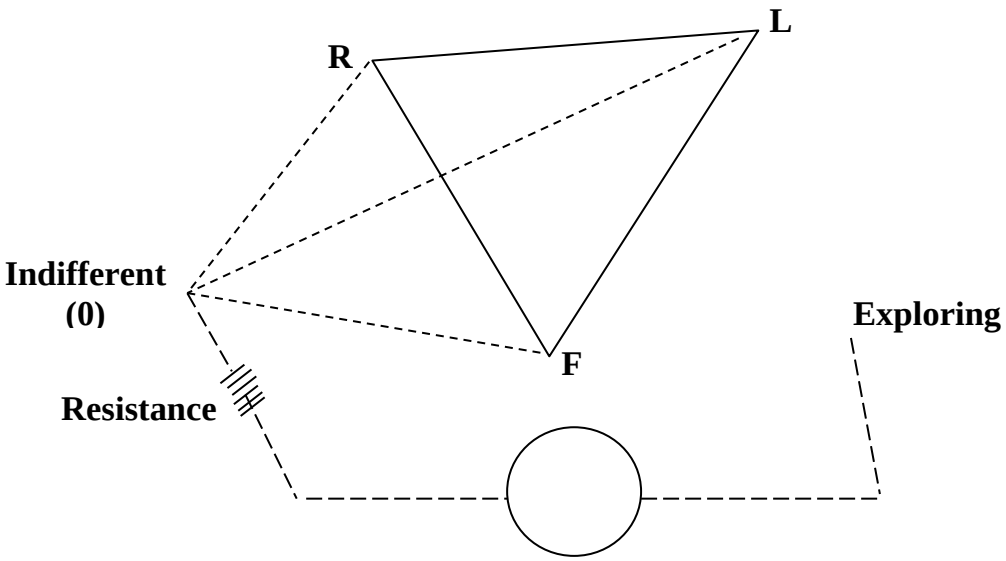
a) Rt arm.
b) Lt arm
c) Lt foot

معظم الوصلات شمال

ECG Leads د. محمد فايز



Unipolar Lead



Unipolar lead معناها أسجل الكهربا عند نقطة واحدة (**Exploring**) أما النقطة الأخرى (**In different**) **تساوى صفر**

Indifferent = (R – L) + (L – F) + (F – R) = 0

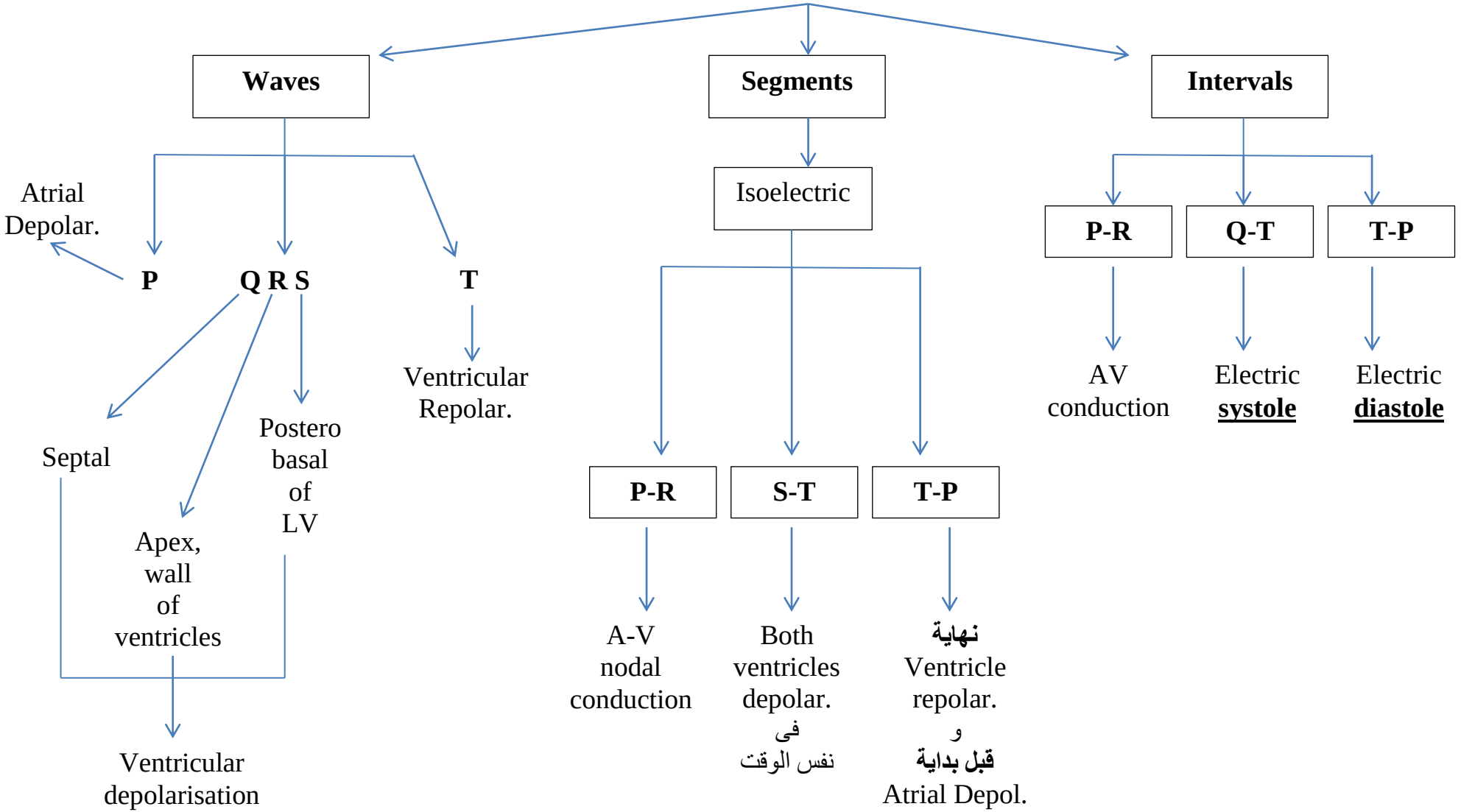
لا توجد نقطة فى جسمك، الكهربا عندها تساوى صفر

It can be applied only by **high resistance (5000 ohm)** to be indifferent

Exploring electrode ⟨ Limb **(unipolar Limb Lead)**

Chest **(unipolar chest Lead)**

Normal ECG د. محمد فايز



ECG Notes د. محمد فايز

Not recorded

- SA node → **Small** Muscle mass
- Atrial repolarization → **Masked by** Ventricular depolarization (جاء فى نفس الوقت)

Waves

- **+ve** → Direction to left with
- **-ve** → Direction to right against

→ **Axis**

T wave in same direction as QRS Complex

- **1st** part depolarized is **Last** part repolarised
- اختـلاف فـى Charges

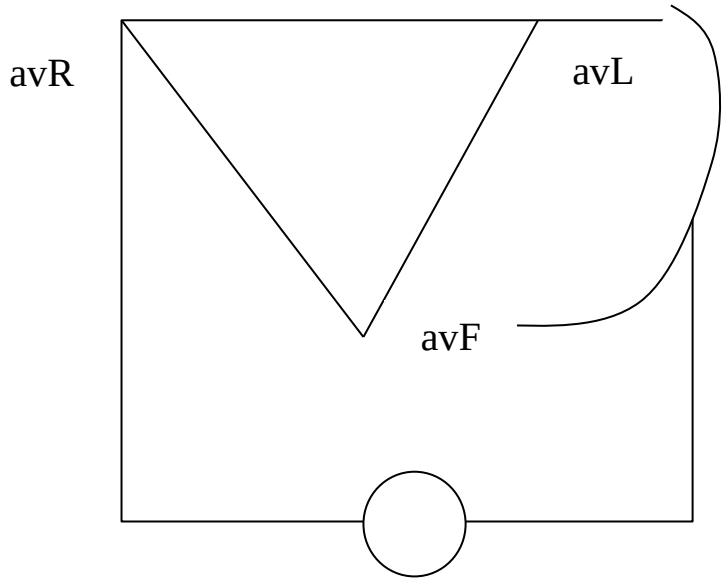
هـام

Duration حفظ

of

- Waves
- Segments
- Intervals

Proof: Augmented Unipolar Leads = 50% ↑ Unipolar Lead د. محمد فايز



$$avR = VR - \left(\frac{VL + VF)}{2} \right)$$

$\therefore$ 2 avR = 2 VR – (VL + VF)

Δ VR + VL + VF = Zero (**Kirchhoff's 2nd law**)

$\therefore$ (VL + VF) = - VR

$\therefore$ 2 avR = 2 VR – (-VR) = 3 VR

$\therefore$ **avR = 3/2 VR**

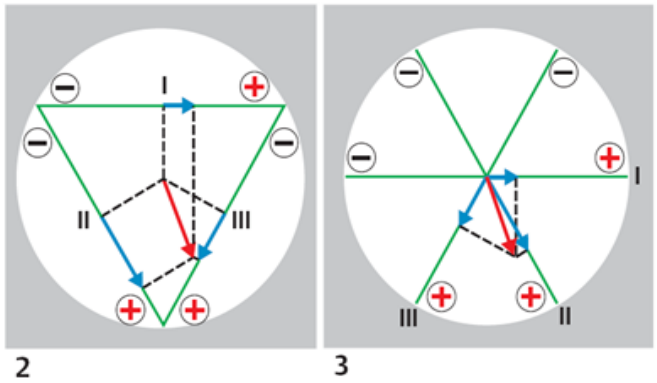
	Atrium		Ventricle	
	Endocardium	**Epicardium**	**Endocardium**	**Epicardium**
Depolarised	1st	Last	1st	Last
Repolarised	1st	Last	Last	1st

Premature atrial contractions **not** followed by compensatory pause

Premature ventricular contractions **followed by compensatory pause**

Cardiac Axis د. محمد فايز

Calculation



Value

Normal

-- 30° → + 100°

Deviation

Right

Value
> + 100°

Causes

Physiological

Tall, thin person

Pathological

Rt BBB

Lt Ectopic focus

Lt Ventricular extrasystole

Left

Left axis deviation

Value
> - 30°

Causes

Physiological

قصير ، طخين

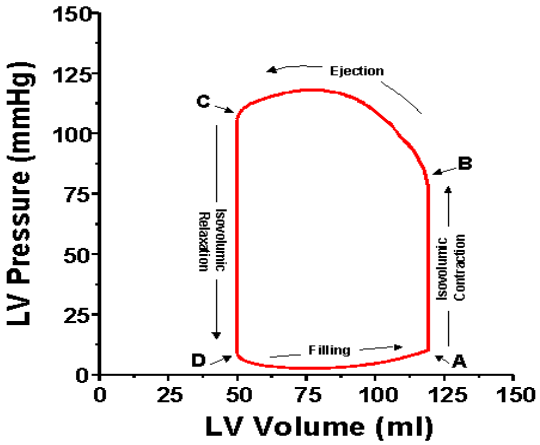
Pathological

(Rt عكس)

Cardiac Cycle د. محمد فايز

	Atrial systole	Isovol. contraction	Maximum ejection	Reduced ejection	Proto diastolic	Iso vol. relaxation	Rapid filling	Slow filling
Atrial Pr.	↑	↑	↓	Increased (VR)			↓	No change
Ventricle Vol.	↑	Constant	↓	↓	↓	Constant	↑	**S**low ↑
Ventricle Press.	↓	↑	↓	↓	↓	↓	↓	**S**light ↑
Aortic Press.	↓	↓	↑	↓	↑	بداية ثم يقل ↑	↓	↓
CBF	↓	↓	↑	↓	↓	↑	↓	↓
Valves	**Open (A-V) Close Semi lunar**	**All closed**	**Open**: Semilunar **Close**: A-V valves			**All closed**	**Open**: A-V **Closed**: Semilunar	
Heart sounds	`4th	1st	1st	-	-	2nd	3rd	-
ECG	P-wave before (0.02 sec)	* Q-wave before (0.02 sec) * QRS	ST segment T بداية	1st ½ T-wave		End T-wave TP segment بداية	T-P segment	

LV Pressure Volume Loop د. محمد فايز

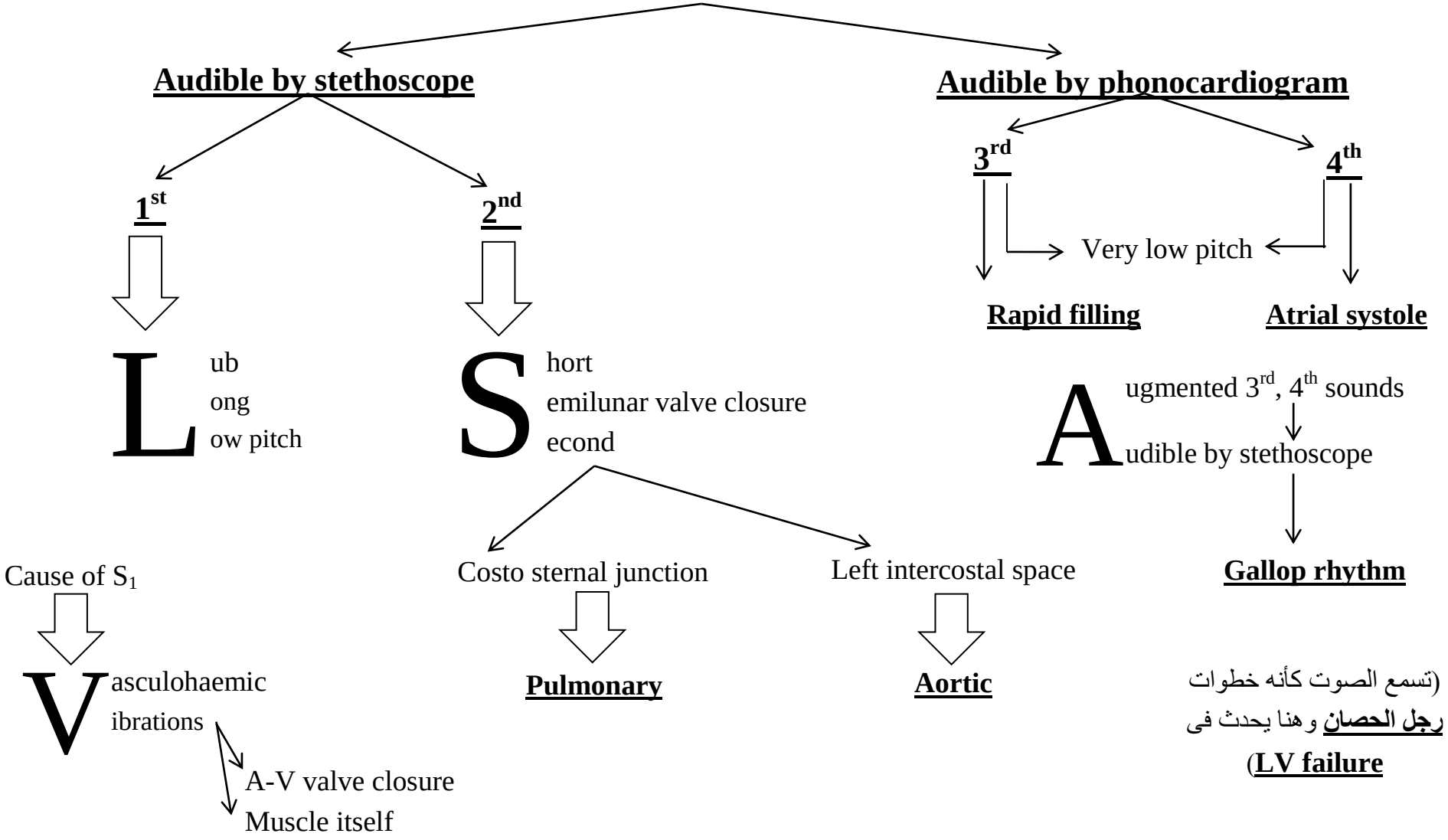


Significance

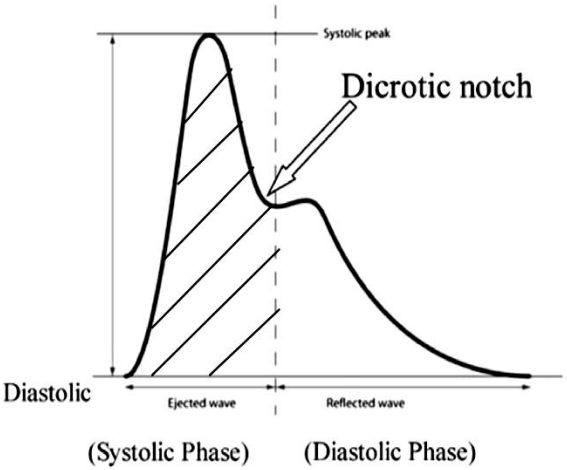
1. Area **under curve** = SW
   = **SV X MAP**
2. **BC** represents SV
3. **DA** represents ventr. Filling.
4. **RV pressure volume loop (as LV)**
   (Systolic RV pressure: 25 mmHg)
5. **Heart failure:**
   Contractility curve (**Shirt to Rt**)

Points				
	A	**B**	**C**	**D**
Valves	MVC	AVO	AVC	MVO
Lines				
	AB	**BC**	**CD**	**DA**
Phase of c. cycle	Isovolumetric contraction	**R**apid, **R**educed ejection	Isovolumetric relaxation	Rapid, slow filling
Ventricular volume	Constant	↓	Constant	↑
Ventricular pressure	↑	**Rapid ejection** ↑ (80-120 mmHg) **Reduced ejection** يقـل	↓↓↓	**Rapid filling** يقل حتى يصل صفر **Slow filling** ↑ up to (5-8 mmHg)

Heart Sounds د. محمد فايز



Arterial Pulse Wave د. محمد فايز



	Anacrotic limb	Catacrotic limb	Dicrotic notch (Incisura)	Dicrotic wave
Aortic pr.	↑	↓	↑	↑
Cause	Opening of Aortic valve	Less vibrations Set up in Aorta	Sudden AVC	Aortic Elasticity
Phase of c. cycle	Maximum ejection	* Reduced ejection * Ventricular diastole	نهايـــة Protodiastolic	Iso vol. relaxation

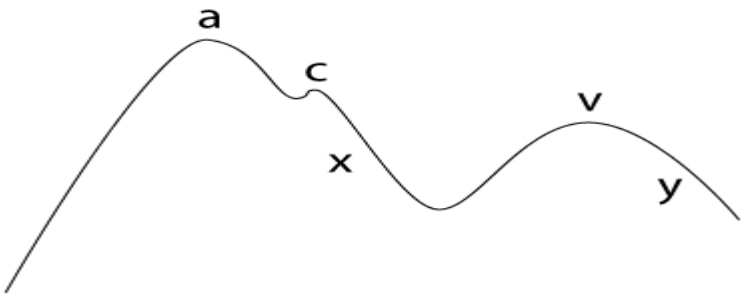
Factors affecting

- **Pulse wave**
  - Elasticity
    - if decreased (**Atherosclerosis**)
      - ↑ Conduction
- **Pulse volume**
  - Strong
    - **A**therosclerosis
    - **A**.I
  - Weak
    - نزيف

Notes

- **Rate**
  - **4-8** (Aorta)
  - **16** (Artery)
  - **لا يعتمد على** Blood flow
- **Periphery**
  - تبدأ Notch متأخرًا
  - يعلى Anacrotic متأخرًا

Jugular Venous Pulse (JVP) د. محمد فايز



	"a" wave	"c" wave	"x" descent	"v" wave	"y" descent
Wave	+	+	-	+	-
Cause	Atrial ms contract ↓ Atrial press. ↑ JVP	Bulge of **Tricuspid valve** Into Rt atrium	Down ward Displacement of AV ring	VR While **Tricuspid valve** is closed	Opening of **Tricuspid valve** and Rapid emptying
Phase	Atrial systole	Iso volumetric contraction	Rapid ejection	Isovolumetric relaxation	Rapid filling
Signif-icance	No → **AF** Large → **Tricuspid stenosis** Giant → **3rd AV block**	Giant ⟶ Tricuspid regurge	**Note** a-c interval **prolonged** ↓ **1st degree AV block**		

د. محمد فايز COP

COP = SV x HR

COP = (EDV – ESV) x HR

Definitions هامــة

* Volume of blood pumped by each ventricle

Beat → **SV**

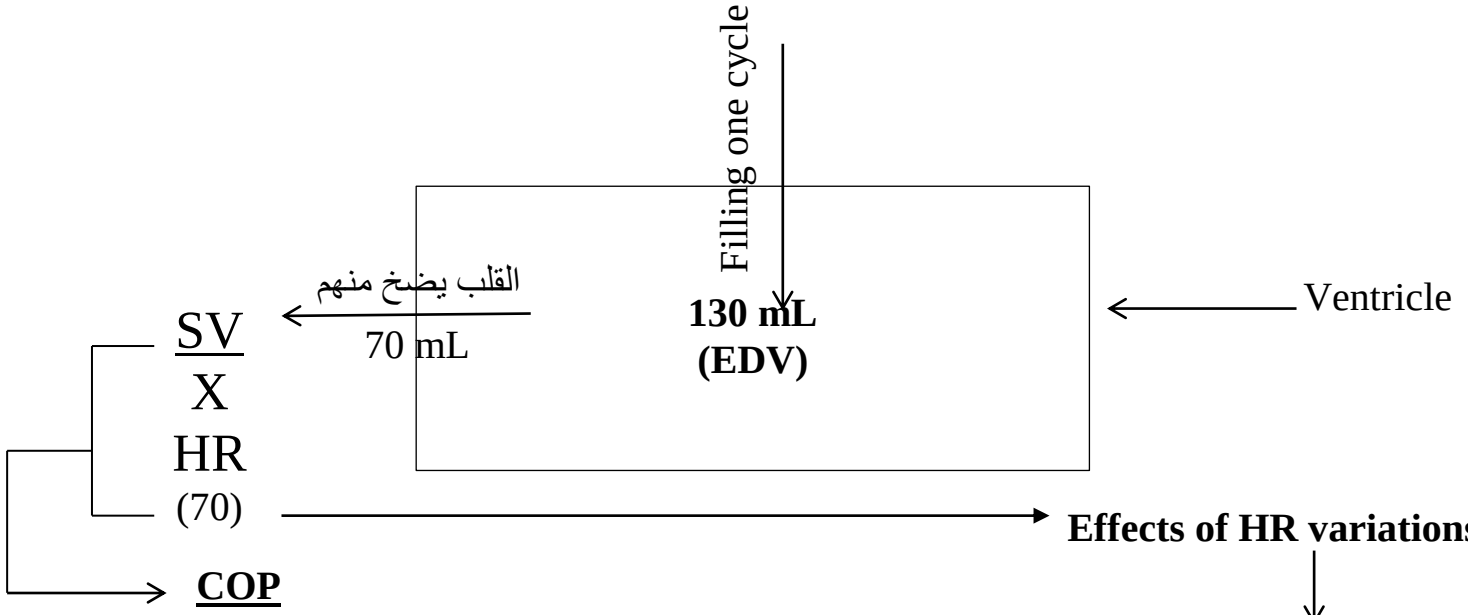
Minute → **COP**

* **Cardiac Index** = COP/SA = 3.2 L/m^2

* **Ejection Fraction (EF)** = SV/EDV x 100 (N=55%)

	EDV	ESV	HR
Rest	130 mL	60 mL	60-90 bpm
Exercise	240 mL	30 mL	180 bpm
Limited by	Starling Law	N° of catecholamine vesicles	Filling Time
↑	Physiological (↑ Venous Return = VR) 1. ↑ Blood volume 2. Venous tone. 3. Skeletal muscle contraction 4. Intrathoracic negativity 5. Atrial contraction 6. Ventricular compliance	**Physiological** ↓ Vagal Tone (-ve Inotropic) **Pathological** ↓ Heart Failure	أنظـر HR Regulation (Vascular)
↓	**Pathological** → Pericardial effusion, Heart failure, MI → Cardiac Tamponade	Physiological ↓ Exercise	

Cardiac Output (COP) د. محمد فايز



HR Range	SV (EDV-ESV)	COP	Notes
60-90 bpm (↑)	↓	لا يتغير	واحد زاد واحد قل
100-160 bpm (↑)	↑	↑	الاثنين زادوا
200 bpm ↑↑	↓↓↓↓	↓	فى الحالتين: النقص أكبر من الزيادة لذلك يقل COP
40 bpm ↓↓↓↓	↑↑	↓	

COP Measurments د. محمد فايز

Experimental Animal

- Anesthetized
  - Ultrasonic flow Meter on Aorta
- Heart-lung preparation
  - Ventricle volume تحسب
    SV = EDV - ESV
    COP = HR x SV

Non Invasive Human measurements

- **Radionuclide Imaging**
  - Injected IV
  - Visualise Heart (Systole, Diastole) → SV, EF (يتم حسابهم)
- **Echocardiography**
  - * Detect ultrasonic waves reflected on cardiac valves.
  - * Calculate EDV, ESV, SV, EF

Human

Fick's Method

Venous ← Tissue → **Arterial**

140 mL O_2/L — 190 mL O_2/L

Tissue use 50 ml O_2 /L
= Heart pumps 1 L → 50 mL O_2
Heart pumps ? L → 250 mL O_2/min

$$\therefore \text{? (COP)} = \frac{O_2 \text{ consump.}}{\text{A-V difference}}$$

= 5 L/min

Thermodilution

* نحقن المريض cold saline (هيفقد برودته لما يذهب للنسيج)، لذلك هنقيس Blood Temp.

Adv.: 1) Not Toxic
2) No 2nd Rise.

Dye Dilution

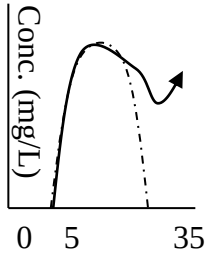
* تم حقن المريض بصبغة خضراء (Indocyanine)

Amount = 6 mg — Conc. = 2 mg/L

∴ Volume of blood pumped by Heart = (Amount / conc.) = 3 L → 30 sec.

*** كيف تم حساب الزمن؟**

- ظهرت dye لاول مرة فى Pulmonary artery بعد ٥ ثوانى ثم بدأ القلب يتخلص منها، قبل ما تصل إلى الصفر، زاد تركيزها مرة أخرى.
- كان المفروض القلب يتخلص من dye بعد ٣٥ ثانية



∴ ? L pumped by heart to remove dye → 60 sec

∴ COP = 6 L / min

* **Adv**.: Accurate, Non Invasive.
* **Disadv**.: Allergy.

Regulation of COP د. محمد فايز

Intrinsic ⟷ **Extrinsic**

	Heterometric	Homeometric
Phenomenon	Pre Load	After Load
Stimulus	↑ VR	
Time	يبدأ أولا **Transient** (2-5 minutes)	ثانيا **Prolonged** (Not Time Limit)
EDV	↑	Constant
ESV	↓	↓
SV	**Increased**	
Significance	**Physiological (**Exercise**)** **Pathological (**Heart Failure**)**	

Heterometric = Pre Load = ↑ EDV = Starling law

	↑	↓
1. Nervous	**Sympathetic** Ino chrono Tropic β receptor → ↑ c.AMP	Parasymp. العكـس
2. Hormonal	**Glucagon** ↑ c.AMP Tropic Ino (strong) chrono (weak)	
3. Drugs	**↑ c.AMP** β adrenergic agonist inhibit break down **Caffeine Theophylline** ↑ **Ca^{++}** intracellular: **Digitalis**	**↓ c.AMP** **β Blockers** **↓ Ca^{++}** **Ca^{++} channel blockers** (Adalat)
4. Ions		**H**ypoxia ypercapnia yperkalemia

Compare Between MCP, CVP د. محمد فايز

	Mean Circulatory Pressure (MCP)	Central Venous Pressure (CVP)	
Definition	**Average** pressure (Intravascular)	**Pressure in thoracic veins (متصلة بالقلب)**	
Normal	7 mmHg	0-5 mmHg	
	Static	Dynamic	
Determinants	**V**olume **V**enous capacity	**V**olume **R** **V**entricular Pump	
		Physiological	**Pathological**
Decreased	↓ Blood volume ↑ Venous capacity	Gravity Inspiration	Venodilatation Shock
		Physiological	**Pathological**
Increased	↑ Blood volume ↓ Venous capacity (Venoconstriction)	**E**xpiration (Forced) **S**ympathetic	**H**ypervolemia **H**eart Failure **E**xcess blood Transf. **E**mbolism (Pulmonary) **S**hock (Cardiogenic)

Vascular

Arterial Blood Pressure (ABP) د. محمد فايز

	Systolic (S)	Diastolic (D)	Pulse pressure (PP)	Mean arterial pressure (MAP)	**Proof: MAP = D + 1/3 PP**
Def.	Highest pressure on Arterial wall (Cardiac Cycle)	Lowest pressure on Arterial wall (Cardiac cycle)	S-D	Average pressure in Cardiac cycle	O. cycle S . D . D MAP = (S + D + D)/3 Δ PP = S – D ∴ S = PP + D ∴ MAP = (PP + D + D + D)/3 = D + 1/3 PP
Normal	100-140 mmHg	60-90 mmHg	30-50 mmHg	93 mmHg	
Average	120 mmHg	80 mmHg	40 mmHg`	90-95 mmHg	

Factors that affect

SV ← **S**ystolic

TPR ← **Diastolic** → **↑ PP**

Aortic valve

↑ SV

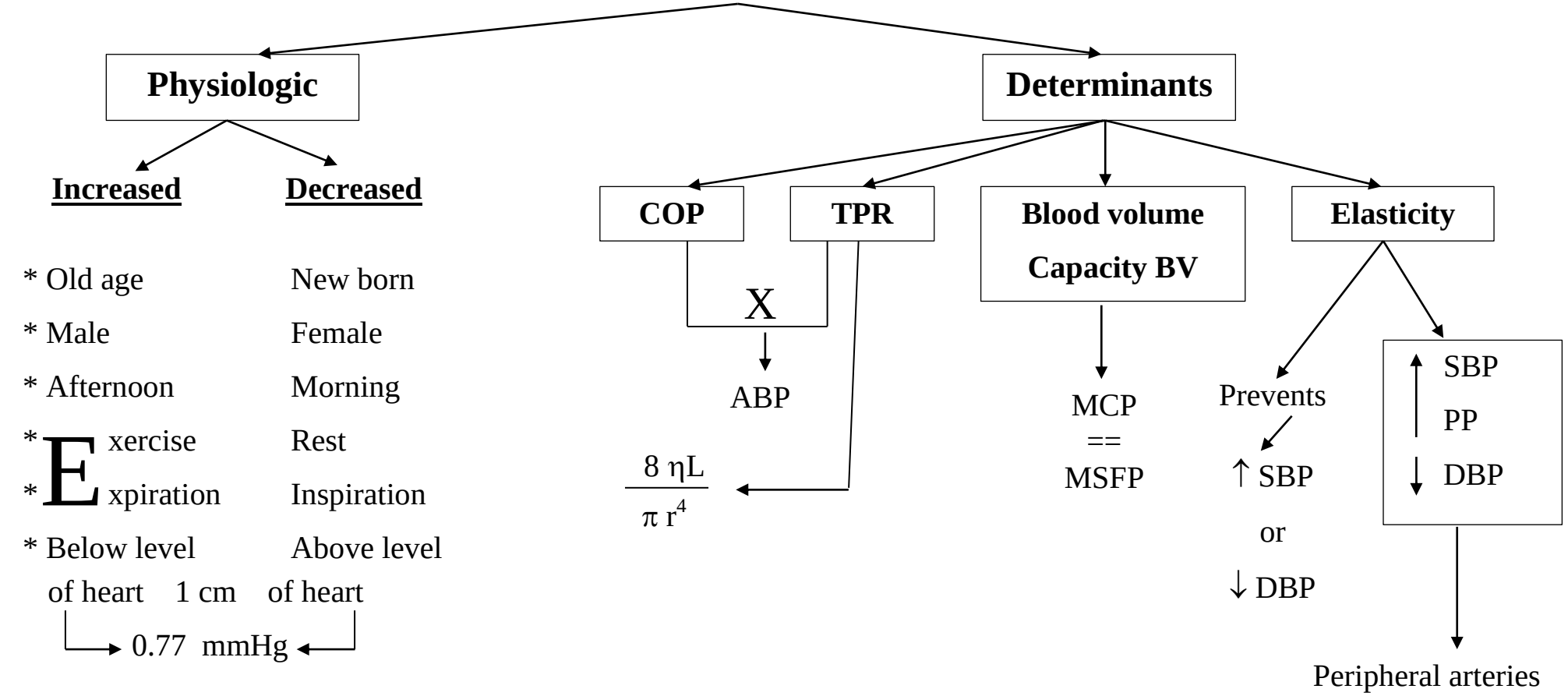
A therosclerosis

Elasticity of artery

MCQ هـام

$\frac{S + D}{2}$ لا يساوى MAP

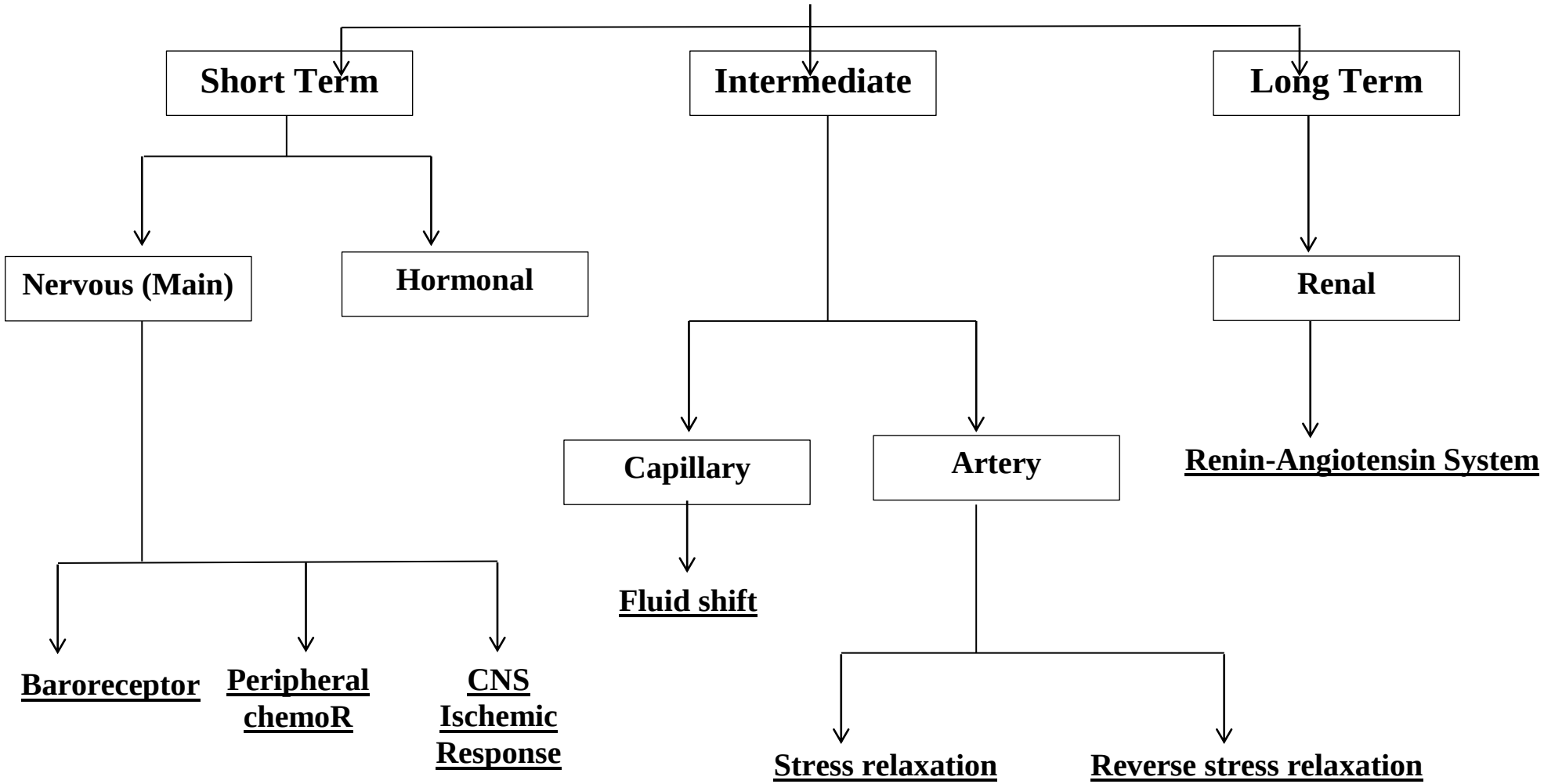
ABP د. محمد فايز



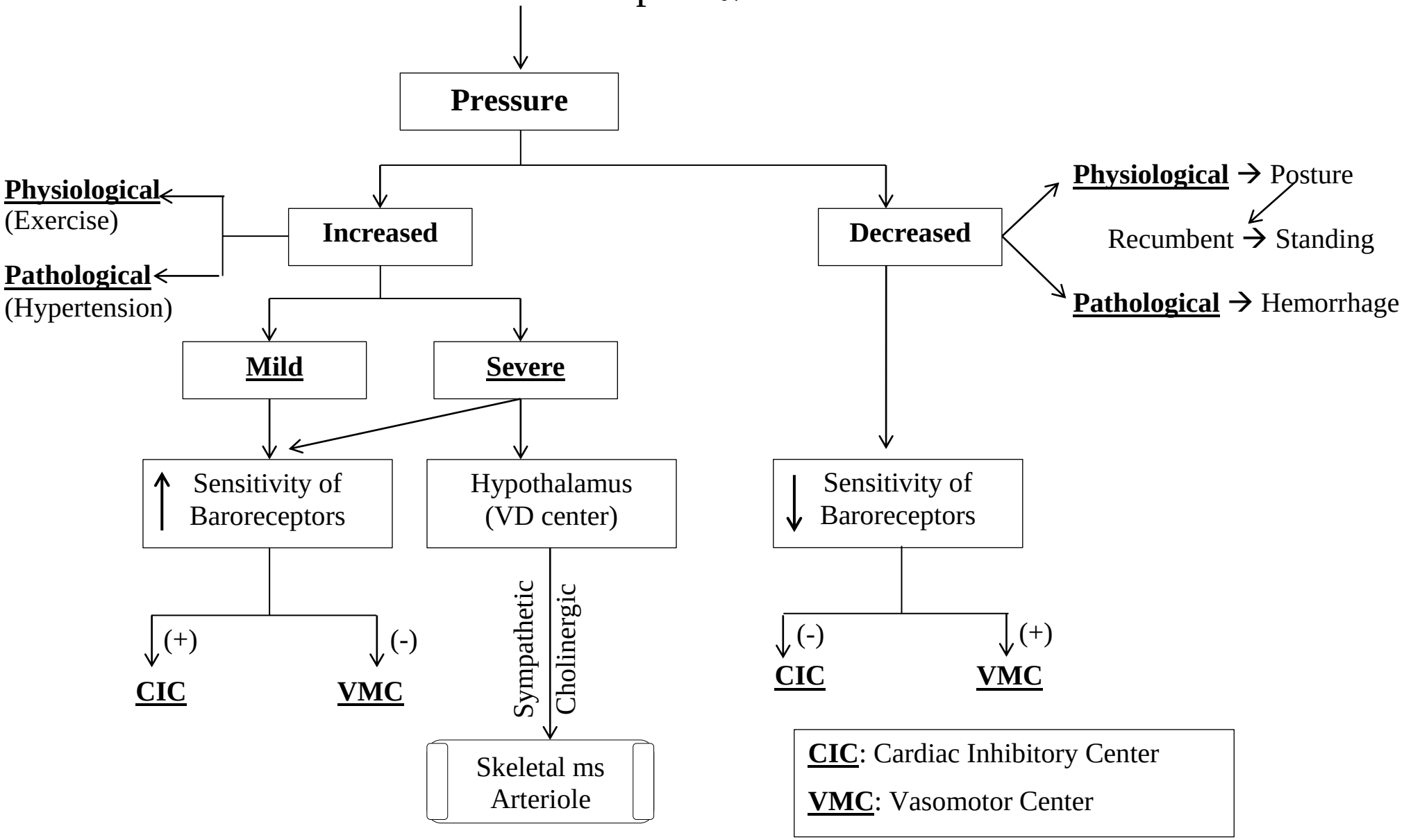
Inspiration: ↑ capacity of pulmonary vessels → ↓ VR (LA) → ↓ EDV → ↓ SV → ↓ COP → ↓ BP

Atherosclerosis: ↑ PP (**Water Hammer Pulse**)

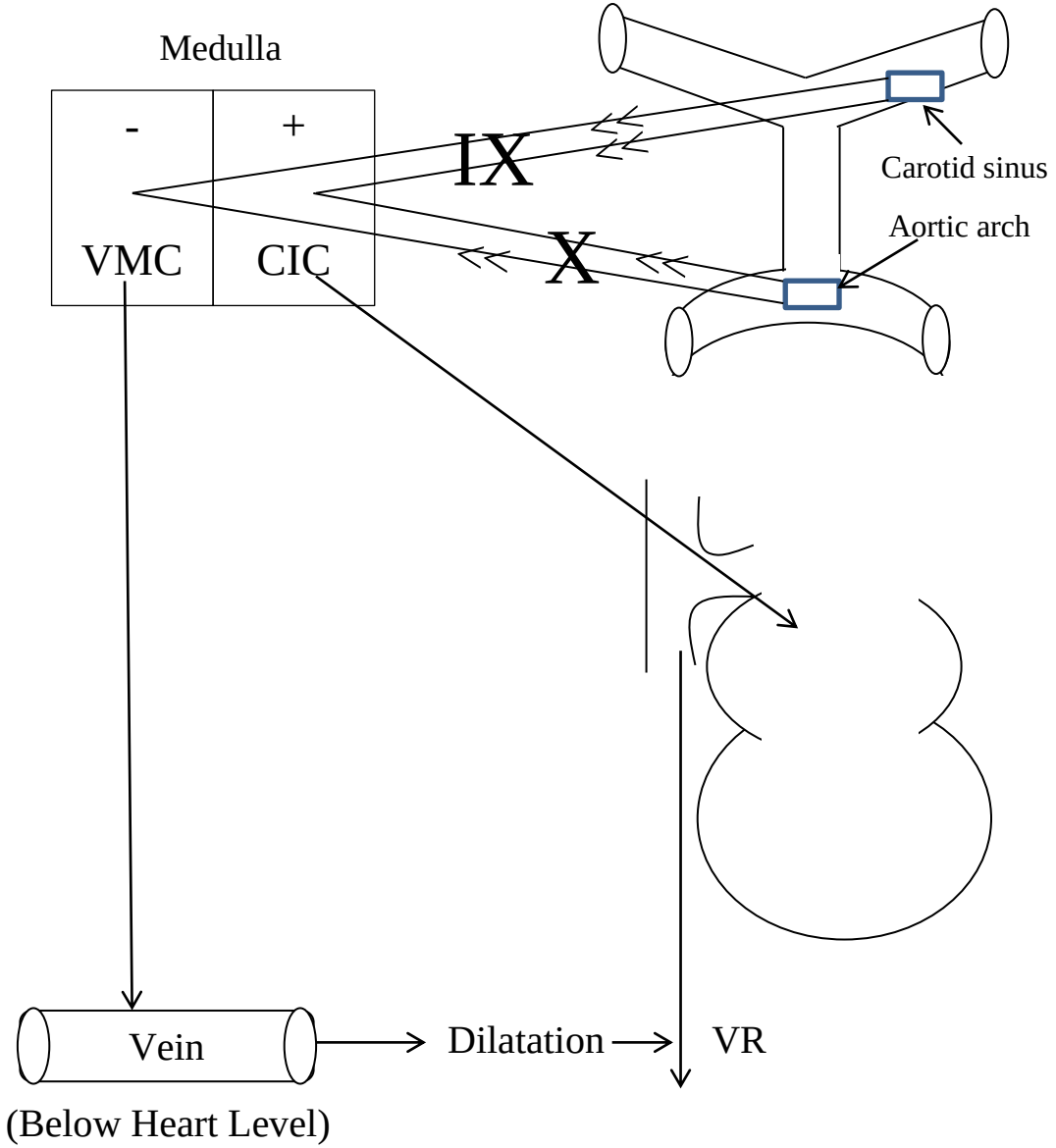
Regulation of Blood Pressure د. محمد فايز



Baroreceptor د. محمد فايز



Baroeceptor Reflex د. محمد فايز



Stimulus → **Pulse pressure** تغير فى

↓ Discharge (**Sustained** pressure)

Receptor → Aortic arch, carotid sinus

Type: **Mechano (Stretch)**

Start firing at 50 mmHg

Maximum firing at 200 mmHg

Afferent IX, X (Buffer nerves)

Center CIC, VMC

Efferent Vagus

Response

(+) → CIC → ↓ HR

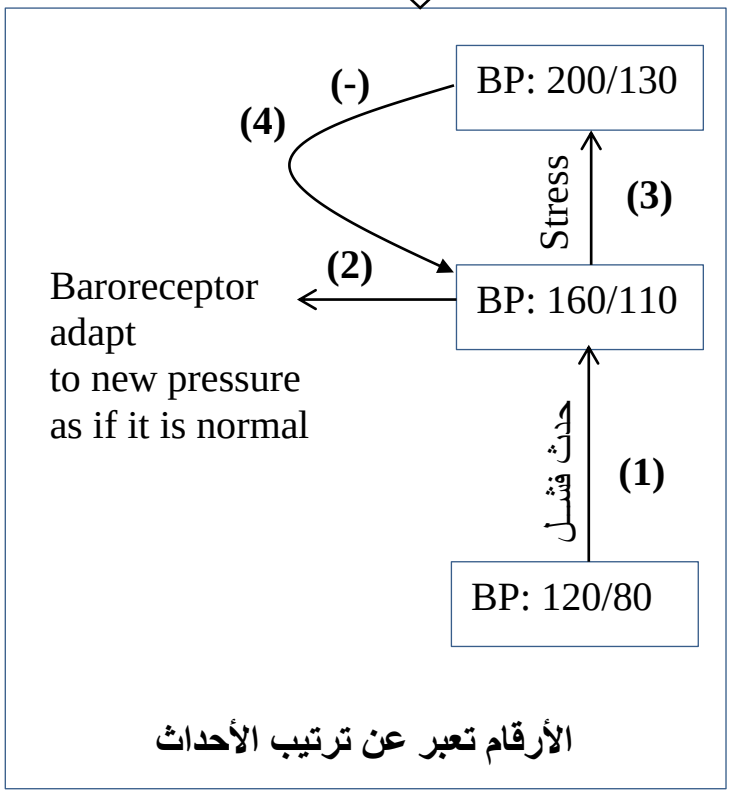
(-) → VMC → Artery, Vein → Dilatation

Decreased COP, BP ← ↓ SV ← ↓ EDV ← ↓ VR

Baroreceptor (تكملة) د. محمد فايز

Baroreceptor إذا فشل

Resetting



Carotid sinus Syndrome

Pressure on carotid artery

Shaving — Tight collar

(-) VMC

(+) Vasodilator center

Evaluation

Valsalva (Deep Expiration against closed glottis)

Phase	BP	Cause
1st (Close Glottis)	↑	**Straining** → ↑ Intrathoracic press. → ↑ Aortic pressure
2nd (Close Glottis)	↓	↑ Intrathoracic press. → Compress Veins → ↓ VR → ↓ COP
3rd (Open Glottis)	Normal	↑ Intrathoracic press., VR, COP يعودوا للطبيعى
4th (Open Glottis)	↑	↑ TPR → ↑ BP

Baroreceptor (2 تكملــة) د. محمد فايز

Other Types

	Atrial Receptors		Ventricular Receptors	
	High Pressure (A)	**Low Pressure (B) volume**	**High Pressure**	**Bezold-Jarisch reflex**
Stimulus	Atrial Systole	↑ VR Blood volume	Ventricular Systole	* MI * Injection of ↙ Veratridine Seretonin Nicotine
Response	VD ↓ HR ↓ Contractility	* Reflex VD ↓ ↑ capillary Pr. ↓ Capillary fluid shift (Intermediate) * ↓ ADH, ↑ ANP ↑ urine (Long Term)	VD ↓ HR ↓ Contractility	↓ HR ↓ BP

Effect on Respiratory Center

↓

Adrenaline Apnea — أنظـر صـ

↑ BP

↓

↓ HR

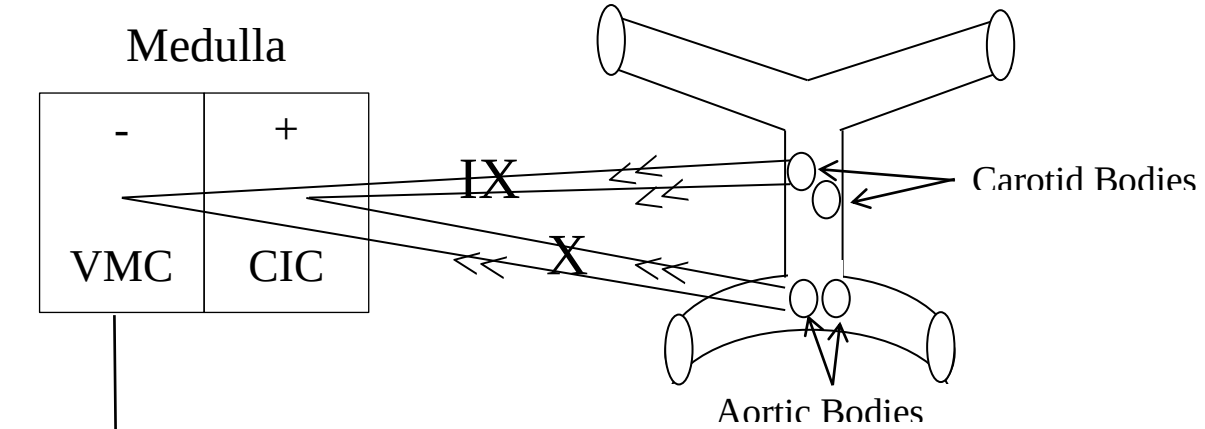
Marys' Law

Adrenaline Apnea → Low Pressure (Act as Atrial Type B)

Adrenaline Apnea → High Pressure

Cardiac Stimulatory Center لا يوجـد

Peripheral Chemoreceptor Reflex د. محمد فايز



Stimulus MAP <80 mmHg

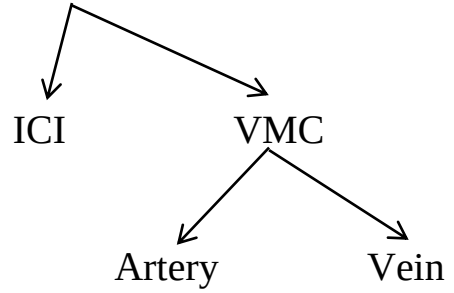
Receptor Bodies (Aortic, Carotid)

Afferent IX, X

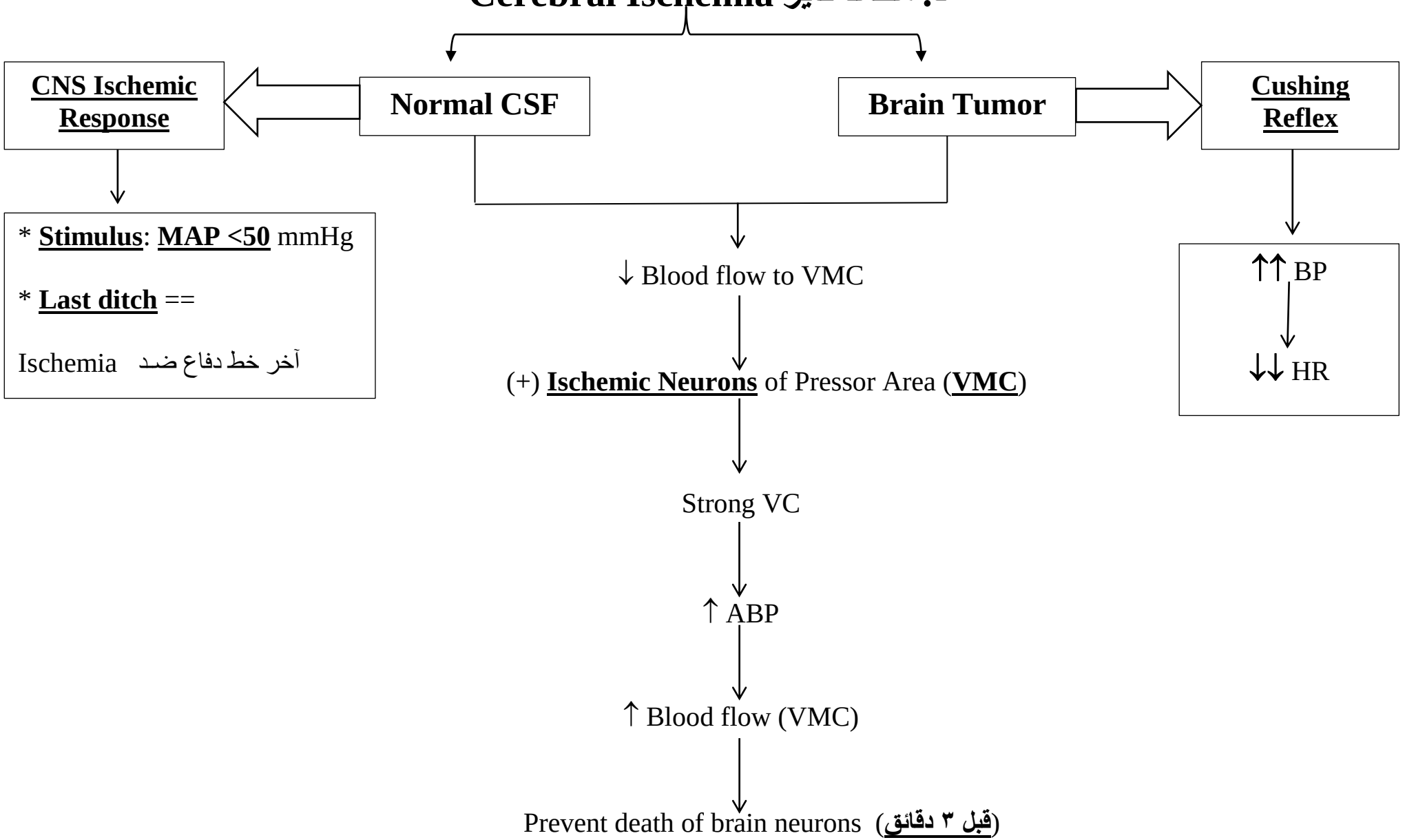
Center CIC, VMC

Efferent Sympathetic

Response



Cerebral Ischemia د. محمد فايز



Rapdily Acting Hormones د. محمد فايز

Catecholamines | **ADH** | **Renin-Angiotensin**

	Adrenaline	Noradrenaline
HR	↑	↓
SV	↑	↓
COP	↑	↓
TPR	Slight affected	↑
BP	Less affected	↑
SBP	↑	↑
DBP	Slight ↓	↑
PP	↑	Unchanged or slight ↓
MAP	Not or little affected	↑

Synthesis: Hypothalamus

tore: Posterior pituitary

Short Term (20 min)

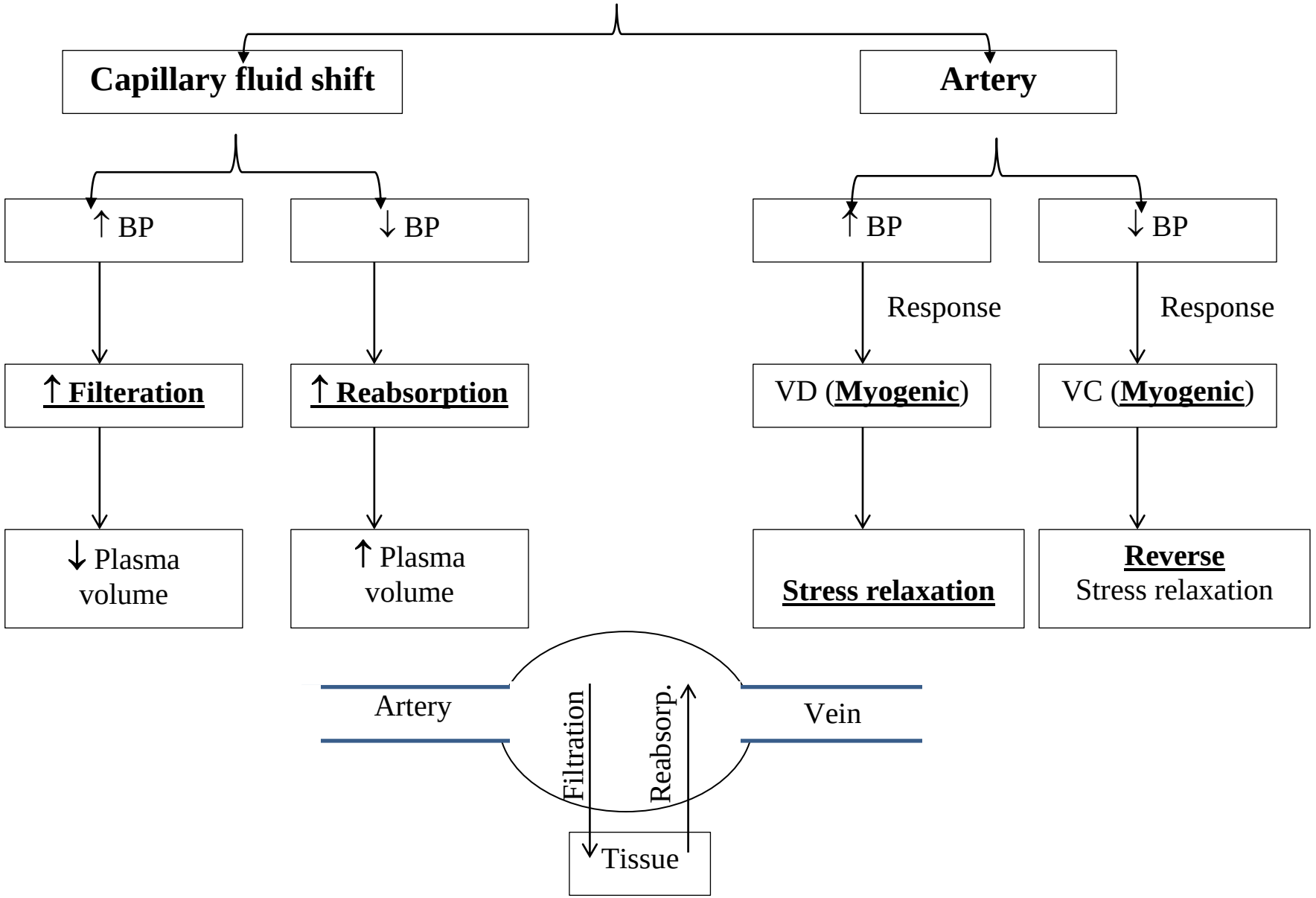
Long Term (Years)

Action

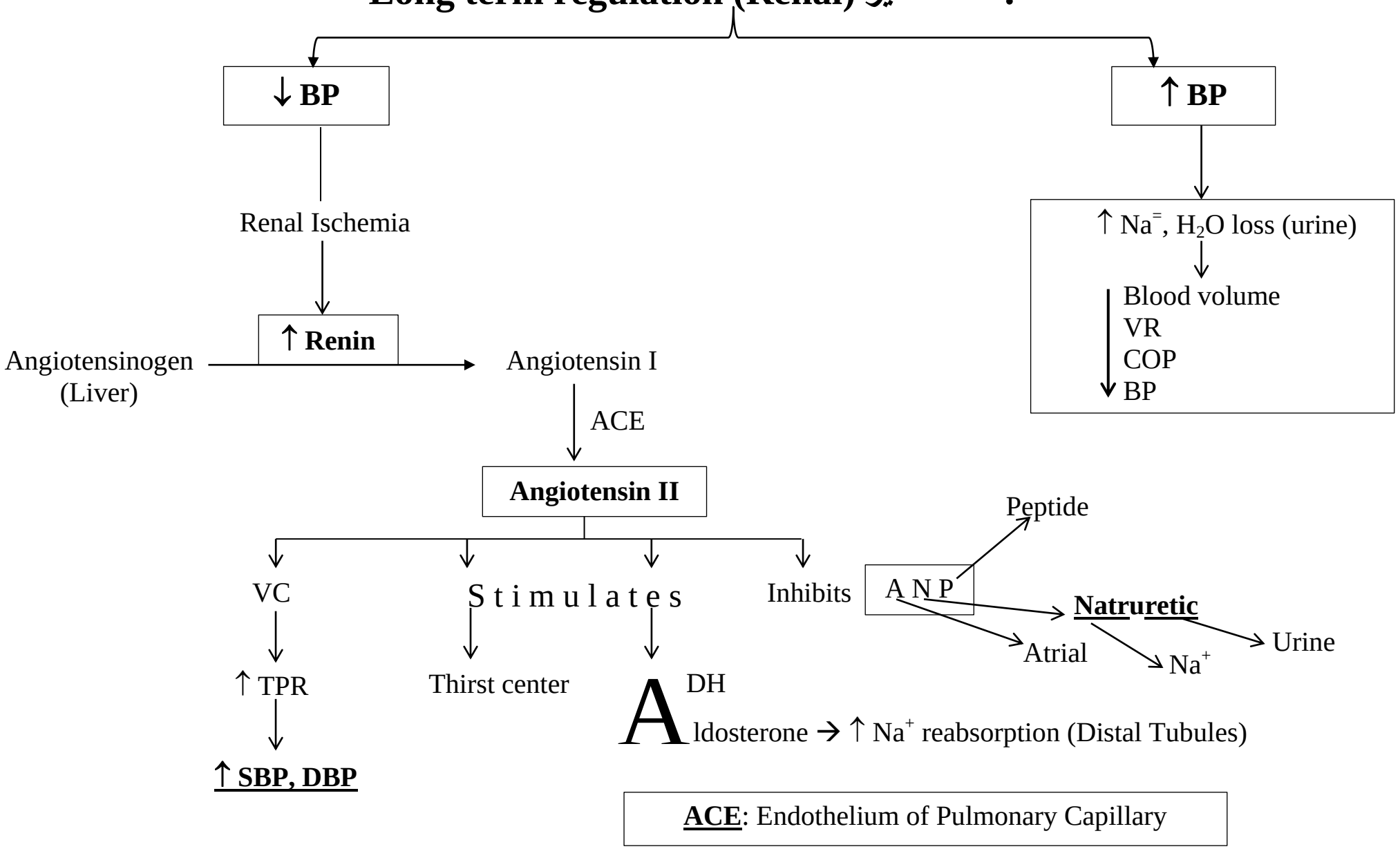
- **V_1** receptors → VC
- **V_2** receptors → H_2O reabsorption (Renal Collecting Duct)

↑ **BP**

Intermediate Regulation of ABP د. محمد فايز



Long term regulation (Renal) د. محمد فايز



Compare Regulation of BP د. محمد فايز

	Shot-Term	Long-Term
	Rapid	**Slow**
Act	Seconds or Minutes	Hours or Days
Control BP	**Moment to Moment**	Days/Weeks/Months
Regulate BP	Change **Capacity** of BV	Change **ECF Volume**
Mainly	**Nervous**	Renal, Hormonal
Effectiveness	↓ (Adaptation)	↑
Potency	**Moderate**	**Extreme**

Vasomotor Center (VMC) د. محمد فايز

Areas

- **Sensory** → Receives from Baro, chemo Receptors
- **Pressor** → VC
- **Depressor** → VD
- **Cardiac** → Tachycardia

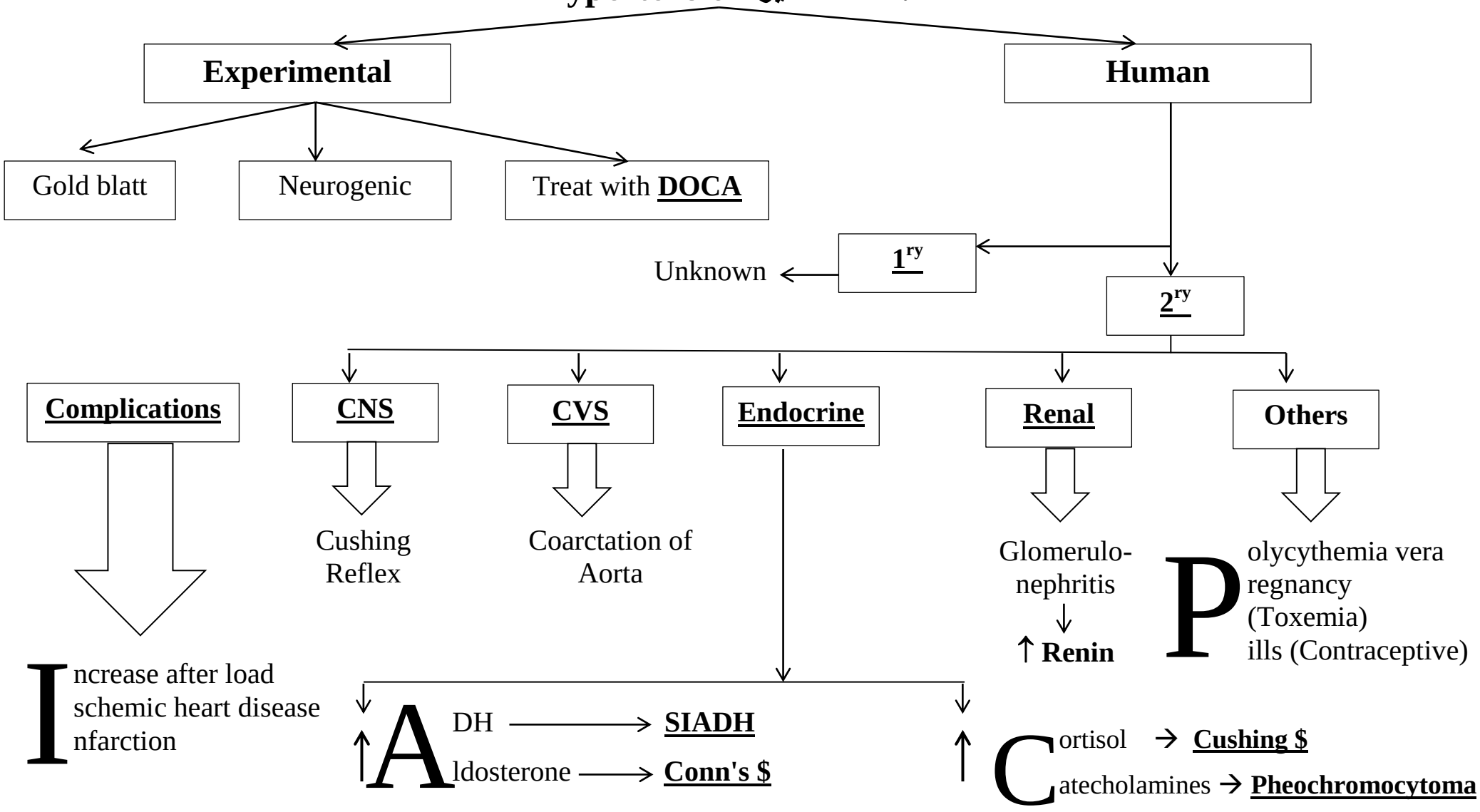
Factors affecting

Stimulatory	Inhibitory
Direct: ↑ CO_2, ↓ O_2 (Cushing R)	----
Chemo Receptors	Baro Receptors
Emotions (**Alarm**)	Emotions (**Grief**)
Pain (**Mild or Moderate**)	Pain (**Severe**)
----	**Lung inflation** (vagus)

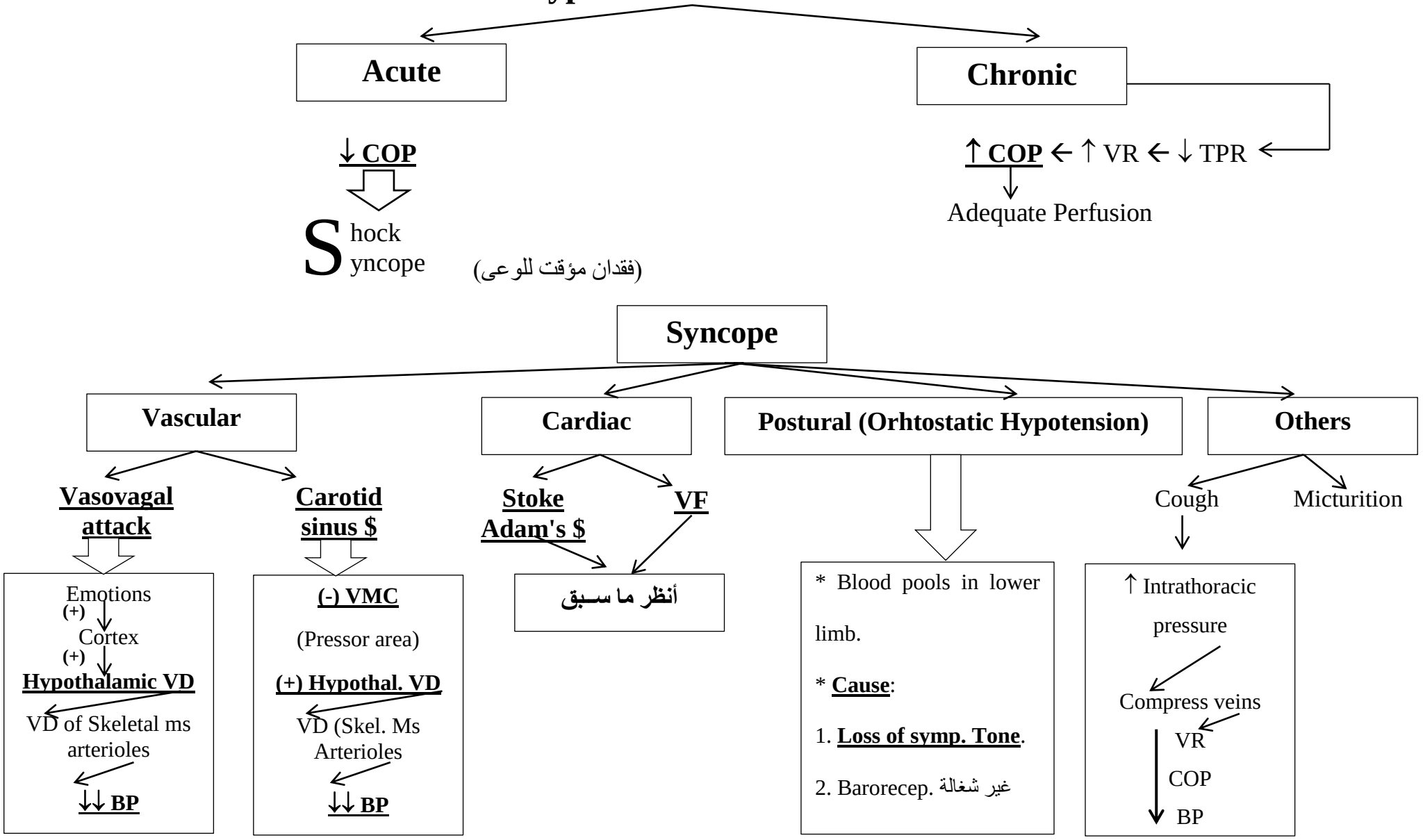
Hypothalamic Sympathetic VD

- **No Normal Tone**
- Activated
  - Physiological (Baroreceptors in Exercise)
  - Pathological **(Carotid Sinus $)**

Hypertension د. محمد فايز



Hypotension د. محمد فايز

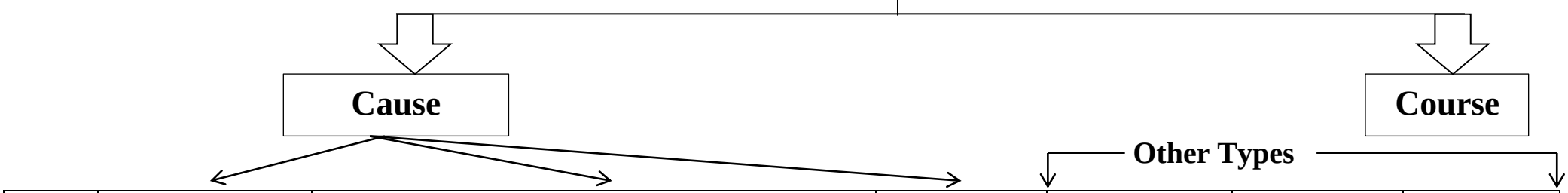


Hemorrhage د. محمد فايز

Compensatory Reactions

	Short Term (Immediate)	Long Term
Correct ↓ BP	1) **↓ Baroreceptor discharge** (+) **VMC** → Venoconstrict, VC Sympathetic → Heart, Adr. Med. VR, EDV, SV, COP ↑ **Arterioles** → ↑ TPR **Skin** → Pale, cold **Renal** → ↓↓ urine vol. ↑ HR → Rapid, weak pulse ↑ Catechol. → Restless Compensatory VC → ↑ DBP → ↓ Pulse Pressure 2) ↑ **Peripheral chemoreceptor reflex** (إشرح) 3) **Capillary fluid shift** (إشرح) 4) **Reverse stress relaxation** (إشرح) 5) ↑ **Angiotensin II, ADH** (إشرح)	إشـــرح **Renin-Angiotensin System**
Correct ↓ Volume	**Plasma volume** restored by **capillary fluid shift** **Plasma proteins** restored by **moving liver proteins to plasma** **RBCs restored** by **spleen contraction**	Plasma vol. restored (½-3 days) by Thirst, ADH Plasma ptn restored (3-4 dys) by hepatic synthesis RBCs restored (4-8 weeks) by ↑ erythropoietin

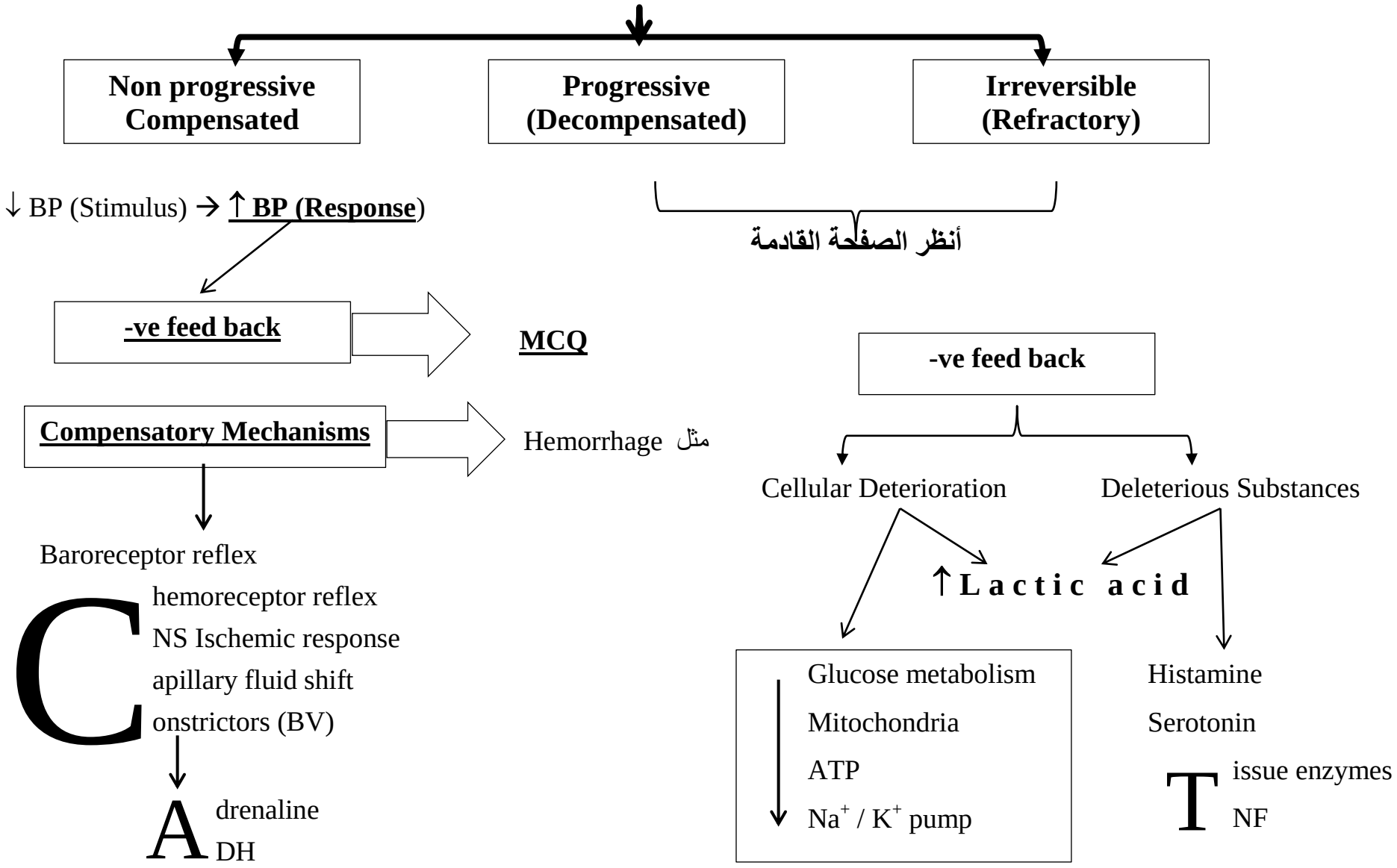
Shock (Tissue Underperfusion) د. محمد فايز



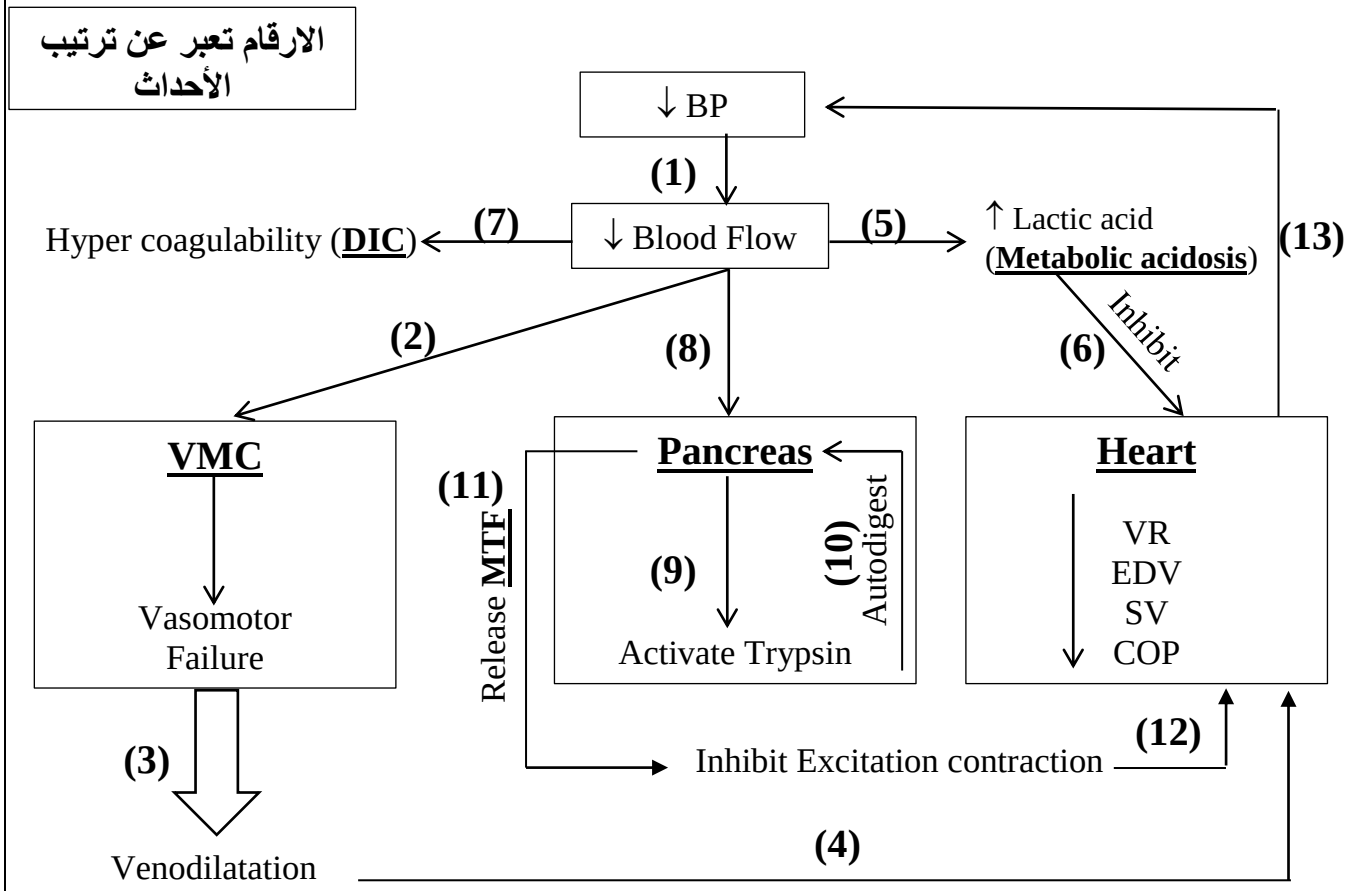
	Hemorrhagic (Cold Shock)	Low resistance	Cardiogenic	Septic	Traumatic	Surgical
Cause	**loss of fluid:** * Hemorrhage * Burn * Severe vomiting * Diarrhea	**Vasodilatation** Emotions → Sympathetic VD center → **Neurogenic** Loss of Vasomotor tone → Spinal cord Lesions → **Spinal** Antigen-Antibody → ↑ Histamine → **Anaphyl-actic** **S h o c k** **Low Resistance** ===== **Vasogenic** **Distributive** **Warm shock**	Heart failure	Severe bacteria infection → Endotoxins → 1) VD (General) 2) ↑ Capillary نفاذية 3) Cardiac depression * **Include features**: Hypovolemic Low resistance Cardiogenic	Crush injury يشمـــــل **Neurogenic Hypovolemic**	
CVP	**D e c r e a s e d**		Increased			

N.B.: ABP not a good measure for progression of shock.

Course of Shock د. محمد فايز

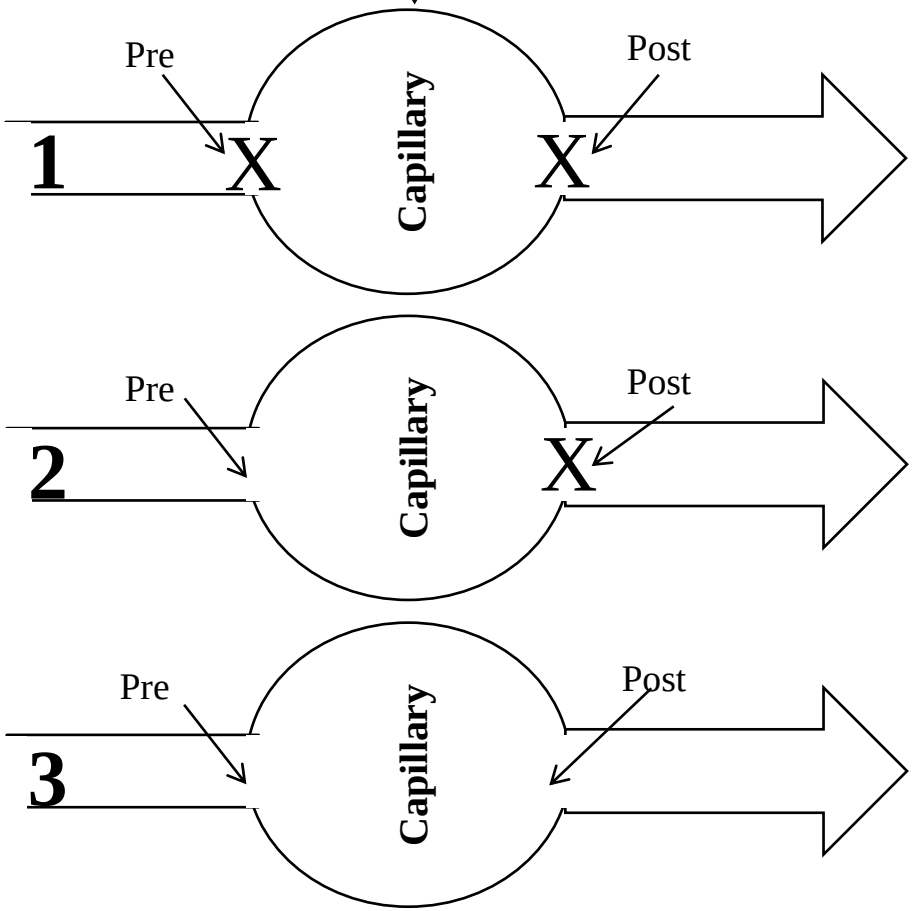


Course of Shock د. محمد فايز

	Progressive (Decompensated)	Irreversible (Refractory)
	Decreased (BP) as stimulus → Decreased BP (Response) **(+ve feed back = Viscious circle)**	
Decompensatory Mechanisms		أنظر الصفحة القادمة

Irreversible (Refractory Shock) د. محمد فايز

Decompensatory Mechanisms

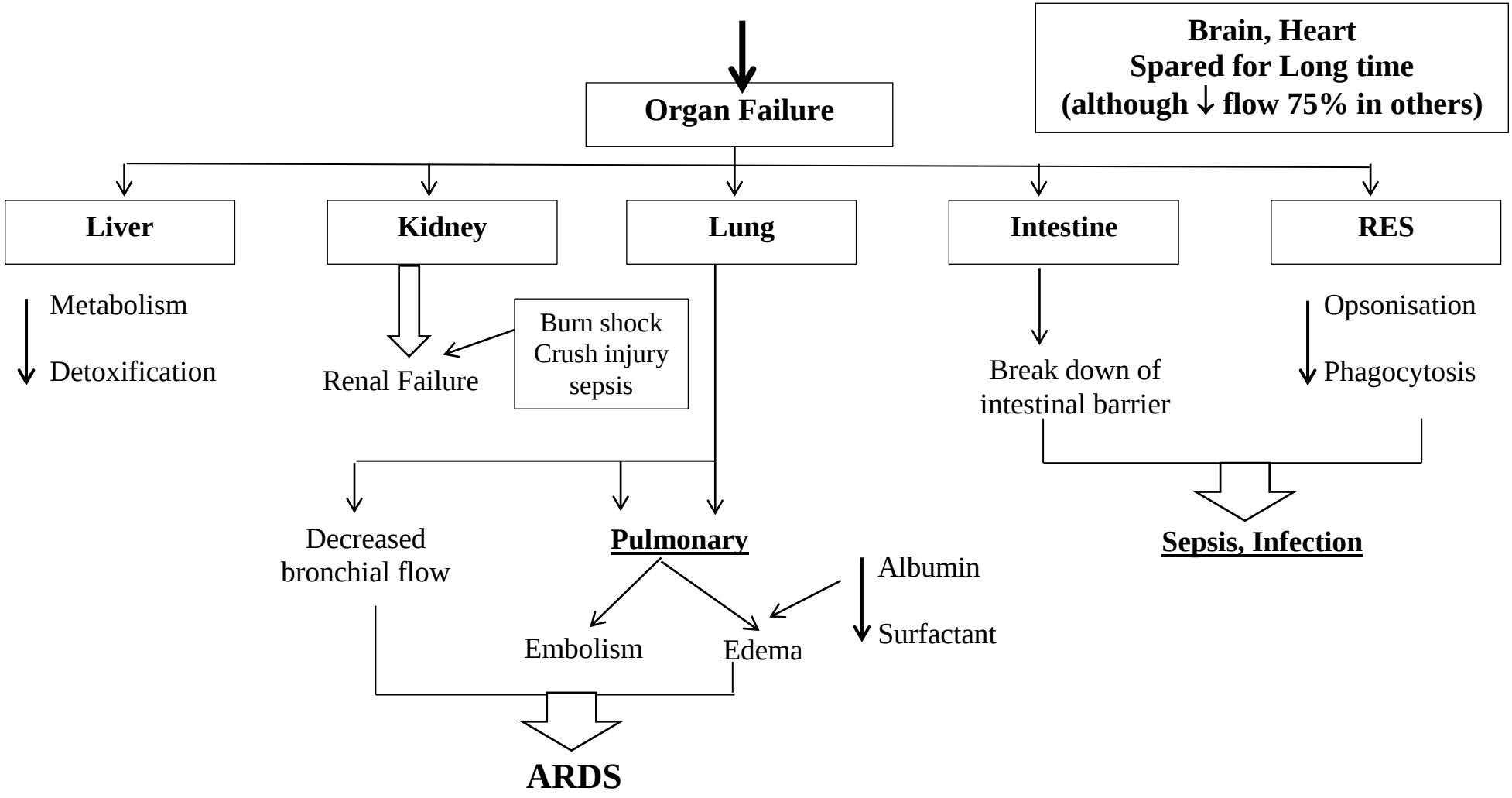


Sympathetic constricts (pre, post capillary sphincters) in (**Hypovolemic and Cardiogenic shock**)

Severe local hypoxia → Accumulation of VD substances → Dilate precapillary sphincter (**Post capillary closed**)

Post capillary sphincter **relax** → Packed RBCs and small fragmented **clots** reach circulation → Pulmonary **embolism**

Course of Shock د. محمد فايز



Factors Affecting HR د. محمد فايز

	Tachycardia	Bradycardia
Higher centers **cortex**	Pain (**Mild or Moderate**) Emotions (**Alarm reaction**)	Pain (**Severe**) Emotions (**Grief**)
Reflexes	1) chemo Receptors 2) Atrial **Type B** 3) Muscle, joint receptors	1) Baroreceptors 2) Ventricular, Atrial **Type A** 3) Cushing.
Respiration	Inspiration	Expiration
Hormonal	Adrenaline, NA, Thyroxine	--
Drugs	Thyroxine Sympathomimetics	β-Blockers Ca^{++} Blockers
Heat	↑ Temperature	Fever

هـام: تأثير **NA أقوى من Adrenaline** على BP ولكن **تـأثير Adrenaline أقوى على HR**

Notes on HR د. محمد فايز

Bain Bridge Reflex

Stimulus: ↑ VR

Receptor: **Type B 'Atrial', Pulmonary**

Afferent: X

Center: CIC

Efferent: X , Sympathetic

Response: ↓ CIC (+) SA node

Tachycardia

↓ O_2, ↑ CO_2, H^+

(-) (+)

SA node	CIC	ChemoRecep.
↓ HR	↓ HR then ↑ HR	↑ HR

Vagal Tone

Part of baroreceptor reflex

Prevented by 1) Vagotomy

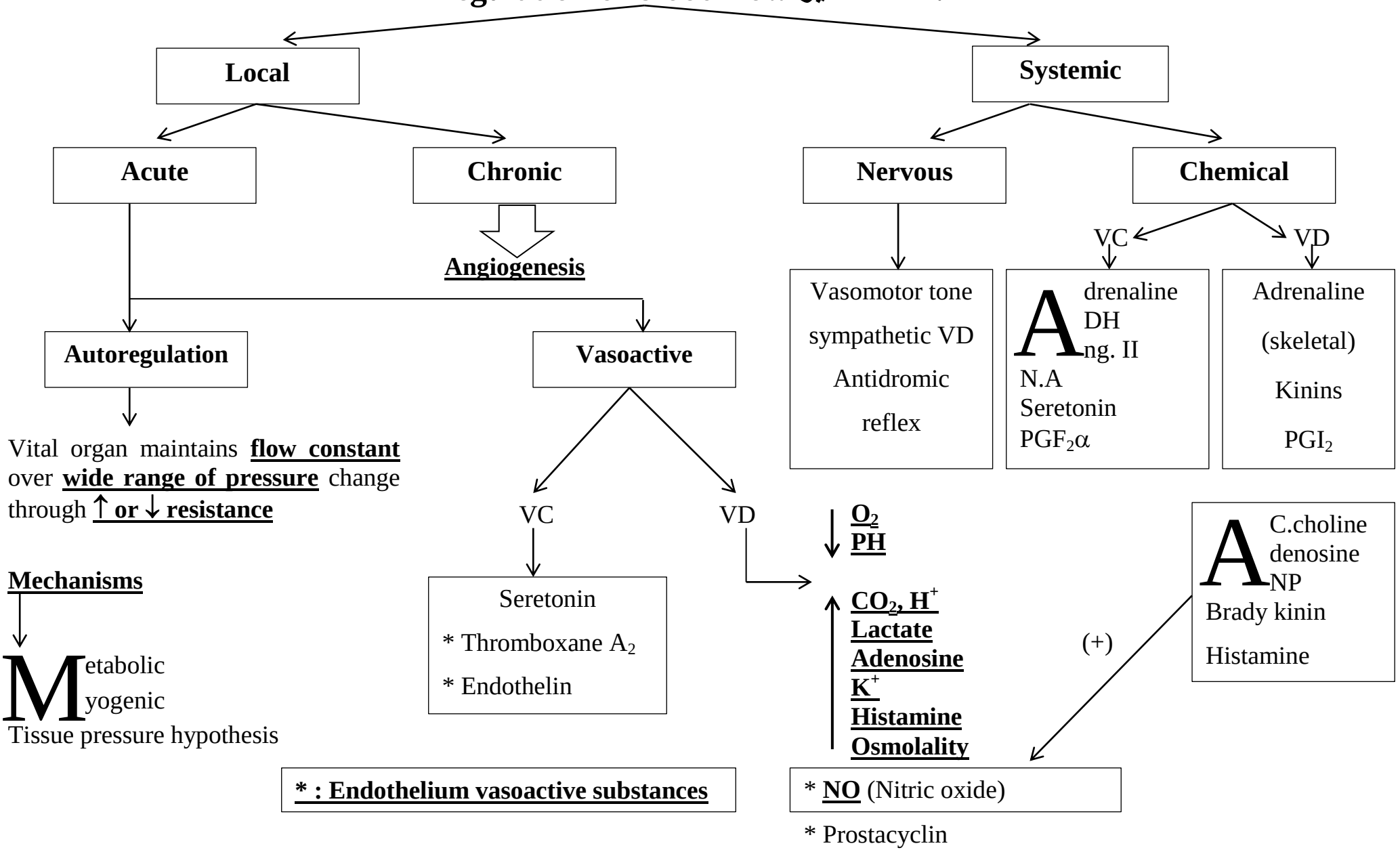
2) Atropine

New Born (**HR = 100 bpm**)

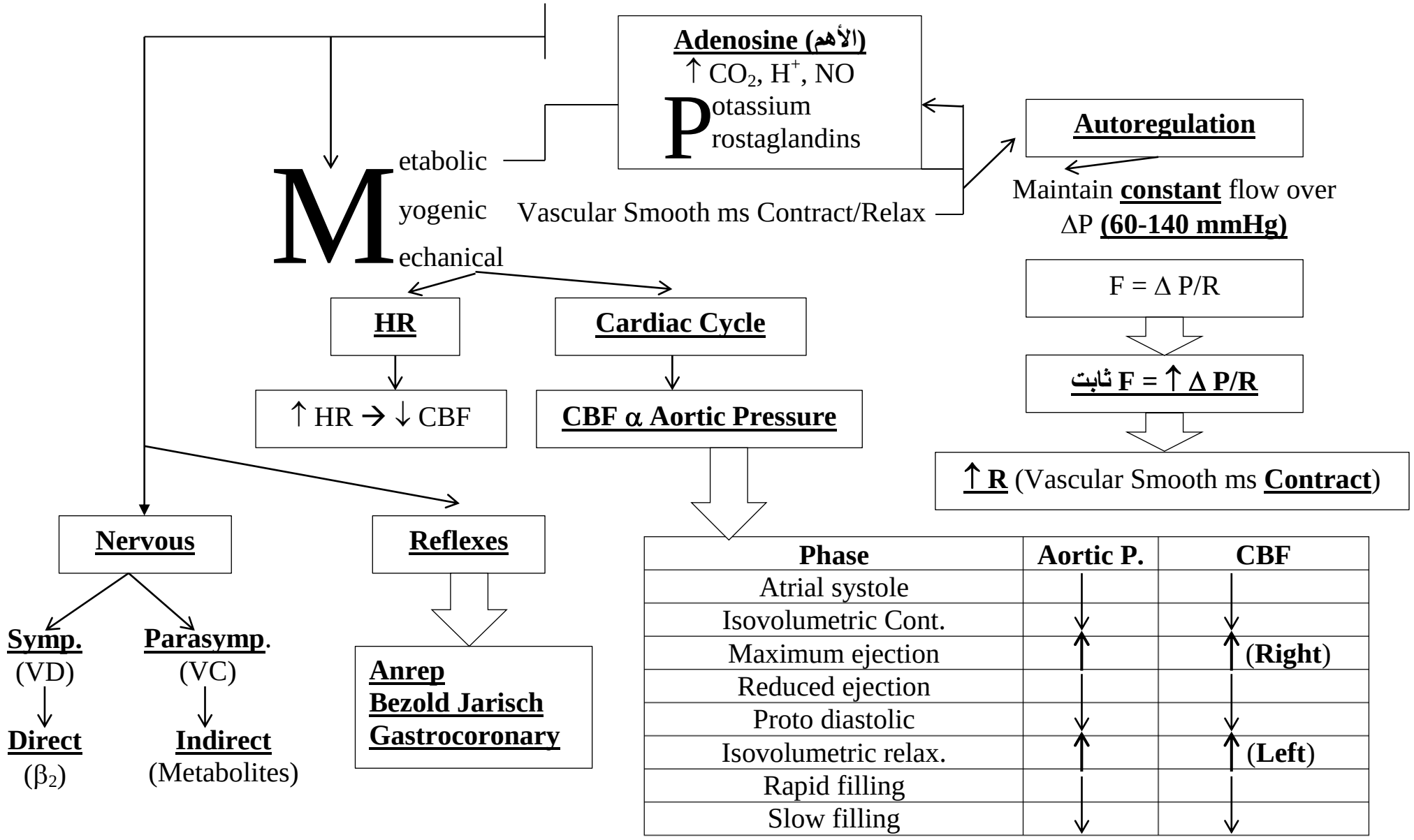
No inhibitory vagal tone

Pressure < threshold of baroreceptor

Regulation of blood flow د. محمد فايز



Regulation of Coronary Blood Flow (CBF) د. محمد فايز



Phase	Aortic P.	CBF
Atrial systole	↓	↓
Isovolumetric Cont.	↓	↓
Maximum ejection	↑	↑ (**Right**)
Reduced ejection	↓	↓
Proto diastolic	↓	↓
Isovolumetric relax.	↑	↑ (**Left**)
Rapid filling	↓	↓
Slow filling	↓	↓

Coronary Cicrulation (1) د. محمد فايز

Supply

Arterial

Venous — 2/3 → Coronary sinus, anterior cardiac veins

Venous — 1/3 → Heart chambers

Heart chambers → Arterio-sinusoidal, Arterio-luminal, Thebesian

Characteristics

* ↑↑ Blood flow.
* ↓↓ O_2 Reserve.
* Aerobic.
* No Anastomosis
* No O_2 debt.
* Systole supply all tissues except LV.

	Right	**Left**
	50%	20%
Supply	RV Inferior and post surfaces	LV Surfaces ant lat septum

Measurement of Coronary Blood Flow → 250 ml/min (Rest) → 1000 ml/min (Exercise)

- **Experimental**
- **Kety Method** → N_2O uptake / A-V difference
- **Radionucleotides**
- **Angiography**

Coronary Cicrulation (3) د. محمد فايز

Coronary artery insufficiency → Angina pectoris (يختفى الالم مع الراحة ويظهر مع المجهود)

علاج:
- Nitrite (VD → ↓ Preload)
- Slight exercise (O_2 lack → ↑ Adenosine → VD)

M.I.

ECG (أنظر ما سبق)

Causes of Death
1. Cardiogenic shock.
2. V.F.
3. Cardiac Tamponade.

Cardiac Work

Potential

MAP x COP

Kinetic
- Rest → 4-5%
- Exercise → 50%

Mechanical efficiency of Heart

$$\frac{\text{\% Work done}}{\text{Total energy expenditure}}$$

(↓ by sympathetic)

Cardiac O_2 Consumption → 60-80 ml/100 gm/minute

D e t e r m i n e d

Inotropic

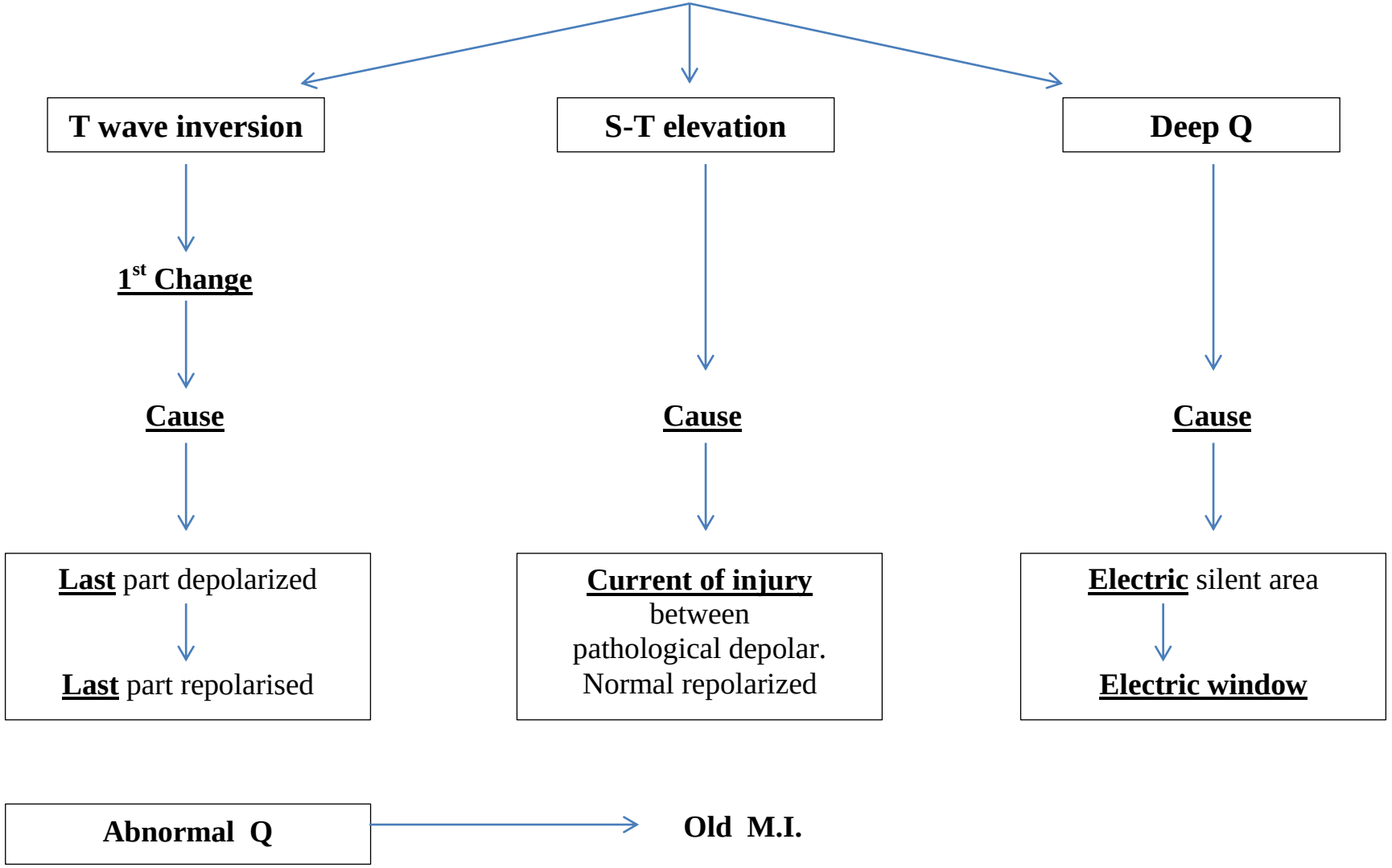
HR → Direct proportional

Tension = $\frac{P \times r}{W}$

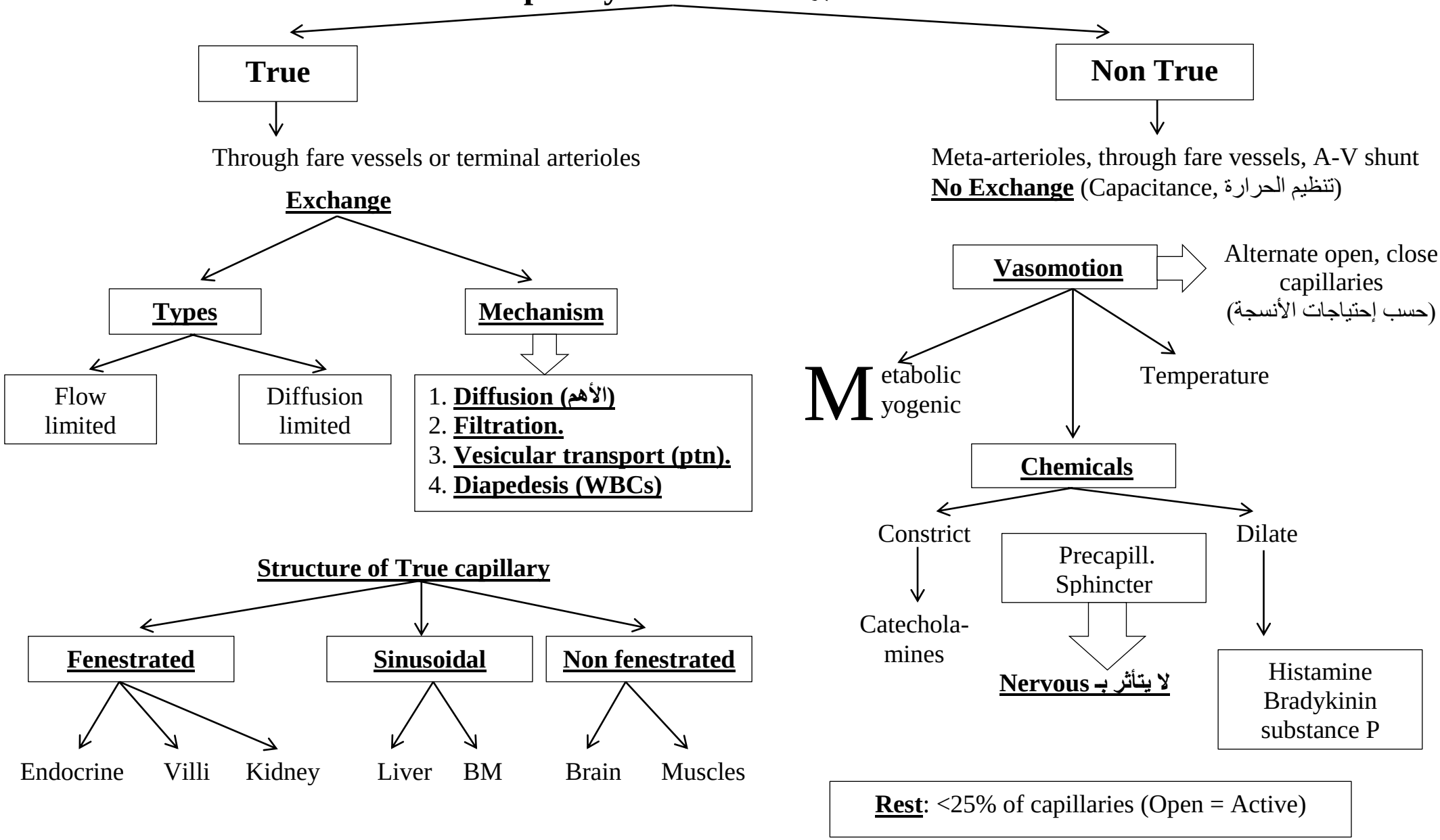
↑ r (radius) = Volume overload → ↑ Tension → ↑ O_2 Consump.

↑ w (wall thickness) = Hypertrophy → ↑ Tension → ↑ O_2 Consump.

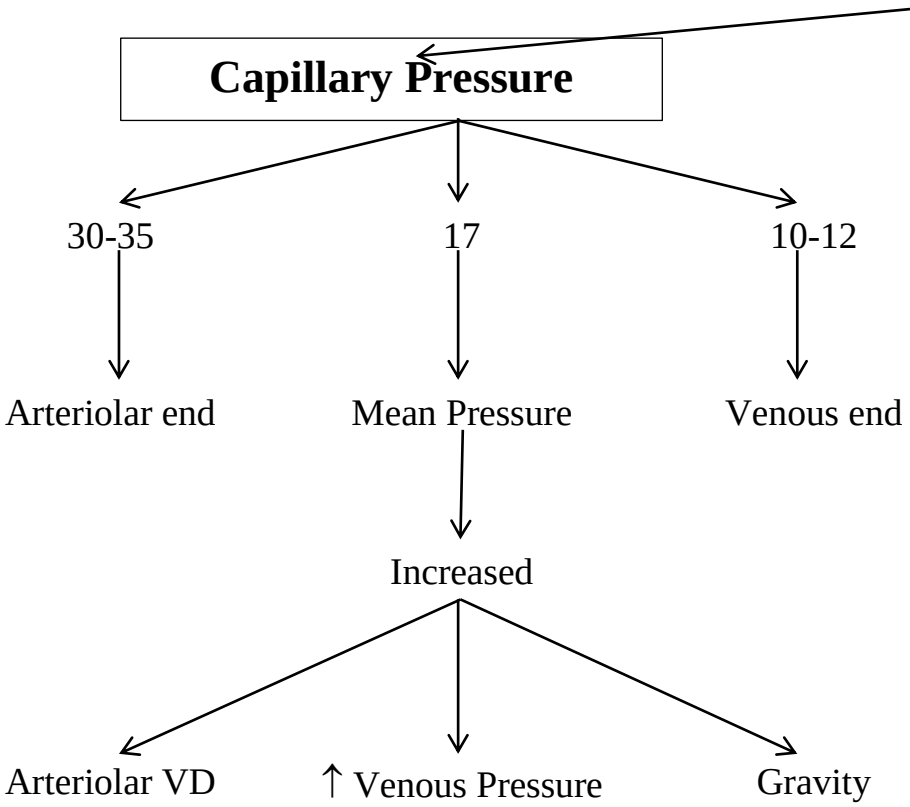
Coronary Insufficiency (M.I) د. محمد فايز



Capillary Circulation د. محمد فايز



Capillary Circulation (تكملة) د. محمد فايز



Capillary Permeability

Increased	Decreased
Liver, Kidney, Glands	Muscle, Brain
Venular end	Arteriolar end
Hypoproteinemia (Liver, Renal diseases) Inflammation (subs. P, Pyrogens)	Increased

Dilated capillaries (Fragile) → **Laplace law (T = P x r)**

Pulse Pressure (5 mmHg) → Decrease **(Arteriolar VC)**

Disappear (Post capillary resistance)

Capillary pressure **nearer to venous** than arterial press.

Precapillary resistance = **4-5** post capillary Resistance

Tissue Fluid Formation د. محمد فايز

Physiological (Starling Forces)

	Arteriolar end	Venous end
Filtration (mmHg)	Capillary pressure: 30	Capillary pressure: 10
	-ve ISF pressure: 3	-ve ISF pressure: 3
	ISF colloid pressure: 8	ISF colloid pressure: 8
Reabsorption	Colloid OP of plasma: 28	
Net effect	13 mmHg (Filtration)	7 mmHg (Reabsorption

Tissue fluid = ISF = Lymph

Edema → ↑↑↑ Tissue fluid

Causes

* ↑ Filtration
  - Arteriolar dilate
  - ↑ Venous Pr.
    - ↑ Capillary نفاذية
    - Pregnancy
    - H.F
* ↓ OP of plasma Ptn
  - Nutritional
  - Hepatic
  - Renal
* Obstruct lymph duct: Filaria → **Elephantiasis**

Types

- Pitting: صباعك يدوس على ميه فيغرس
- Non Pitting: صباعك يدوس على بروتين فلا يغرس → Inflammation

	Lymph	Plasma
Albumin	0.5-1 gm%	4.5 gm%
A/G ratio	↑	↓
Composition	**N e a r e r**	

Factors which ↑ lymph flow:

- Lymphatic pump (VR مثل)
- ↑ ISF pressure
- Activity of tissues

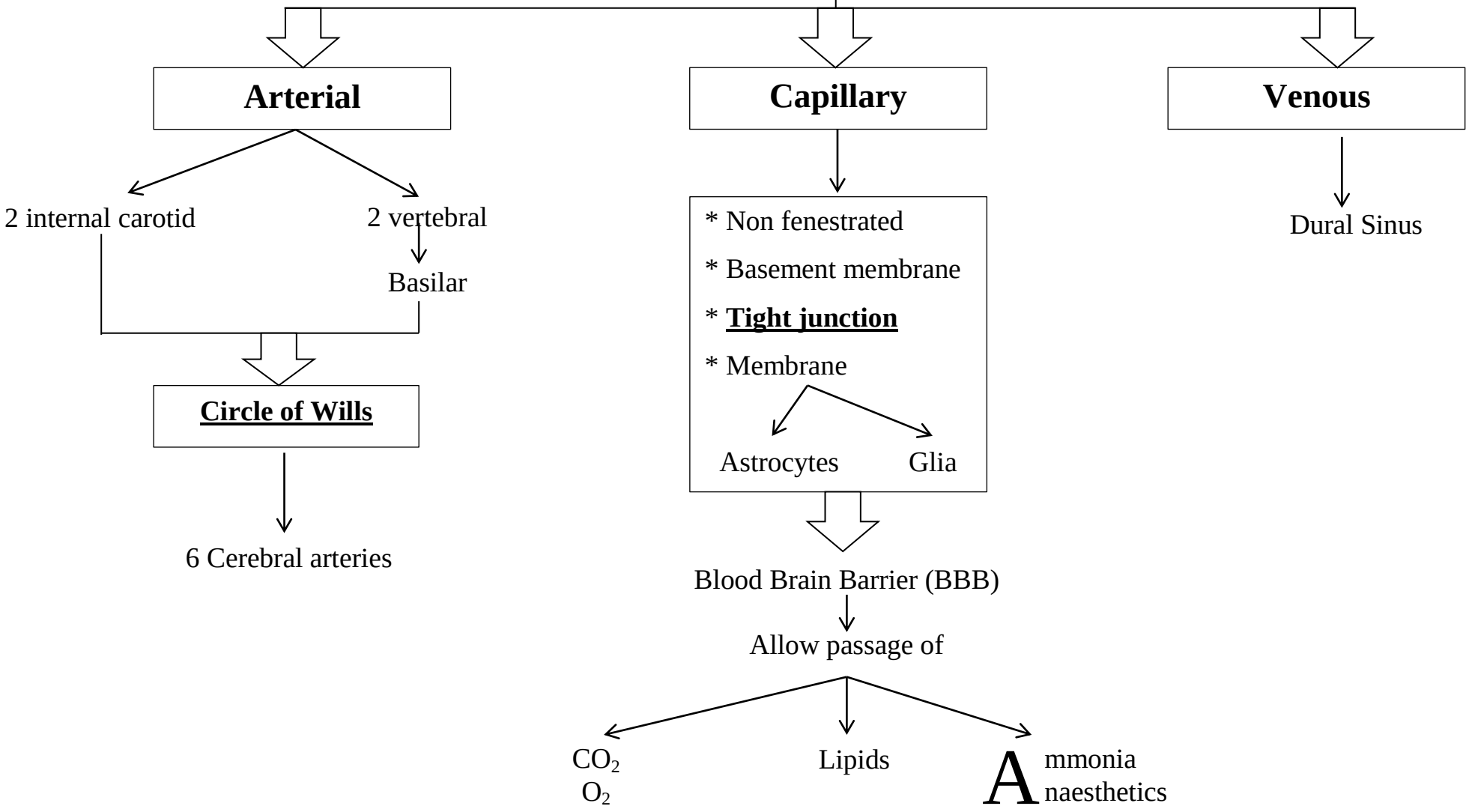
Lymphagogues (↑ Lymph)

- **1ry:** Bacterial Toxins, Peptones
- **2ry:** Hypertonic Solutions

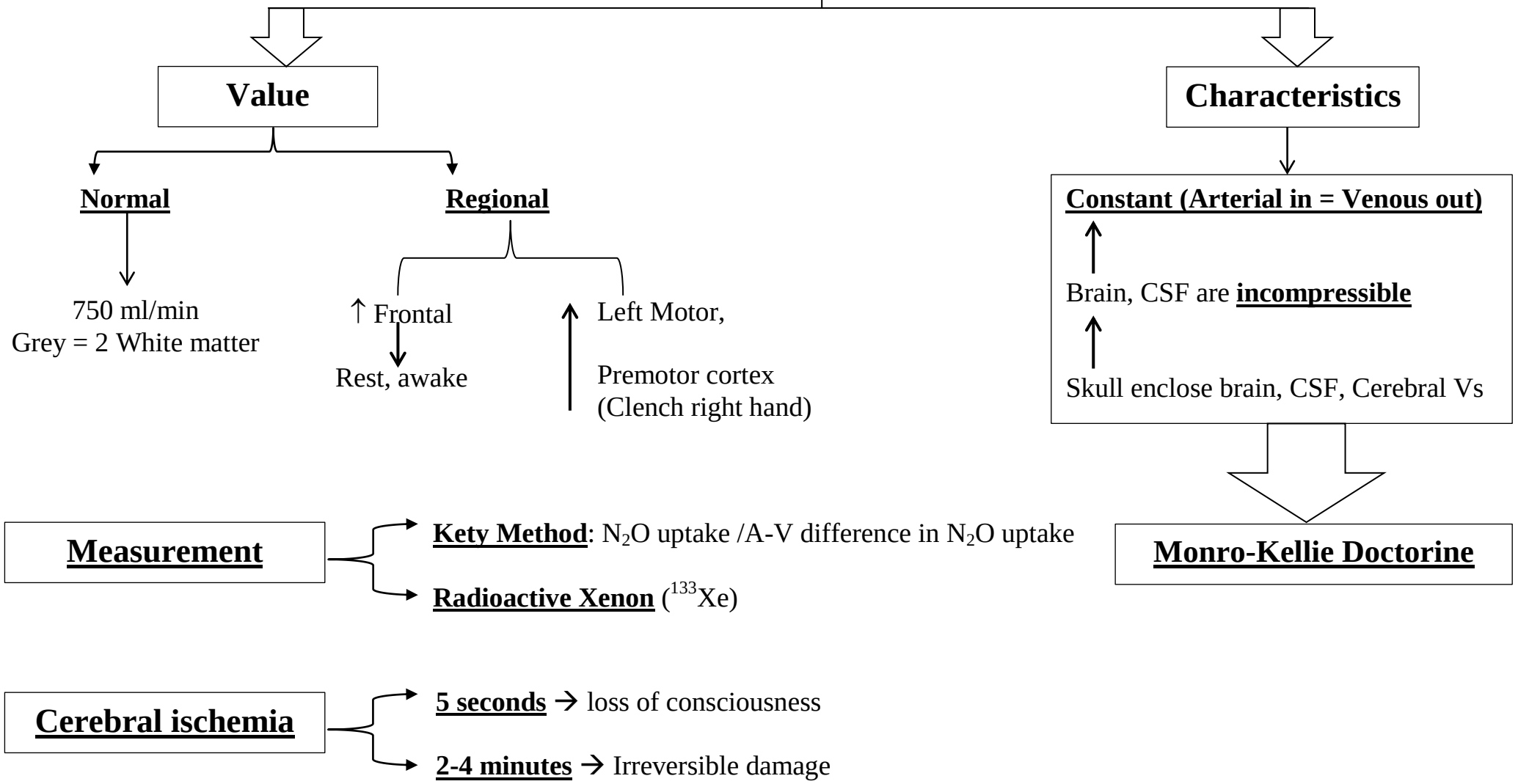
Edema Safety Factors

- Lymph flow
- ↓ ISF
  - Pressure
  - Protein
- 27 mmHg → Pulmonary
- 17 mmHg → Systemic

Cerebral Circulation د. محمد فايز



Cerebral Blood Flow (CeBF) د. محمد فايز



Factors Affecting Cerebral Blood Flow (CeBF) د. محمد فايز

Δ P

CPP = **ABP** (Head) – **VP** (Internal Jugular Vein)

- **Increase**
  - ↑ ABP
  - ↓ VP
- **Constant**
  - **Upward Acceleration**
    - ABP ↓
    - ==
    - VP
  - **Downward Acceleration**
    - ABP ↑
    - ==
    - VP
- **Decrease**
  - ↑ VP
  - ↓ ABP
    - Upright
    - ↓ Volume

هـام : راجـع

Cushing Reflex, CNS Ischemic

Intra Arterial, Venous, Cranial Pressures **are all balanced**

Upright: ↓ MAP (Brain) by **25** mmHg → ↓ CeBF **20%**

Resistance

- **Autoregulation**
  - Physiological
    - Maintain CeBF (60-160 mmHg) Myogenic Metabolic
  - Pathological
    - ↑ ABP (100-200 mmHg)
- **Blood Gases**
  - PO_2
    - Physiological
      - No effect
    - Pathological
      - 30 mmHg (↑ CeBF double)
  - PCO_2 (VD)
- **Viscosity**
  - D Substances
    - CO_2
    - NO
    - Adenosine
  - α Resistance
- **Vasomotor nerves (Symp.)**
  - **Minimal or No control** on Resistance
  - **Hypertension**: Protects BBB

Pulmonary Circulation د. محمد فايز

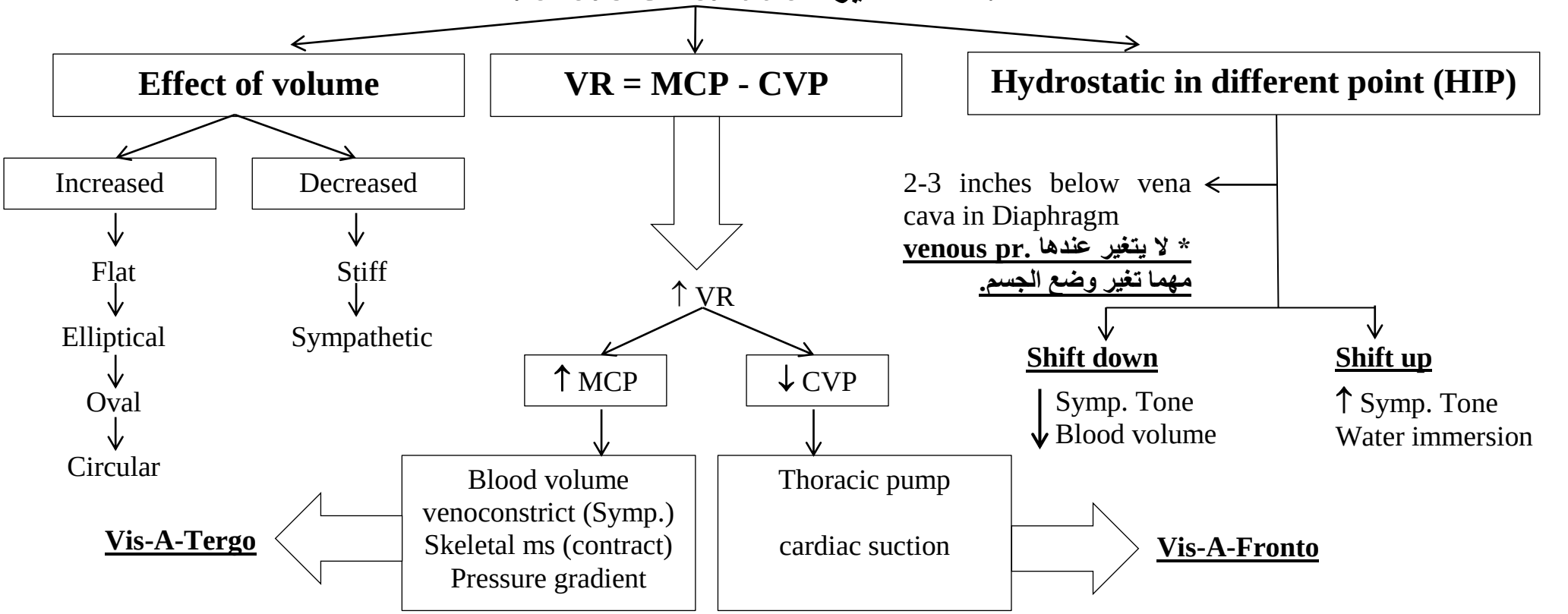
	Pulmonary	Systemic
Flow	More Pulsatile	Less Pulsatile
Pressure:		
SBP	24-25 mmHg	100-140 mmHg
DBP	8-9 mmHg	60-90 mmHg
PP	15 mmHg	30-50 mmHg
PP/SBP	15/25 = 60 %	40/120 = 33%
MAP	15 mmHg	93 mmHg
Hypertension	Mitral stenosis	1^{ry}, 2^{ry}
MCP	10 mmHg	17 mmHg
Precapillary R.	= Post capillary R	4-5 Post Capil. R
Edema	↑ Capillary Pressure / ↑ Permeability	
Edema Safety Factors	27 mmHg	17 mmHg
Gravity	**Apex** : 5 mmHg **Mid zone** : 15 mmHg **Base** : 25 mmHg	Each **1 cm below** heart level → ↑ **Pressure 0.77** mmHg
Resistance	Less Blood Reservoir → 600-700 (Erect) / 1000-1200 (Supine)	High
Respiration	* **Inspiration**: ↓ VR * **Expiration**: ↑ VR * ↓ O_2 → **VC** of pulmonary artery → ↑ RV press. → RV Hypertrophy (**Corpulmonale**)	* **Inspiration**: ↑ VR * **Expiration**: ↓ VR * ↓ O_2 → **VD**

Transient Time

Time which **RBC take** to Traverse pulmonary Cap.

0.75 sec. (Rest) **0.3 sec**. (Exerc.)

Venous Circulation د. محمد فايز



Venous Pressure
- 0-2 (vena cava)
- 6-8 (large veins)
- 8-10 (small veins)
- 10-12 (venules)

Rapid venous velocity (↓ CSA)

Reference Point
4th sternocostal jolint (middle of Rt atrium = **CVP = Zero).**

Flow of veins (2 Descent)
- X → **Rapid ejection**
- Y → **Rapid filling**

Cardiac activity α VR
- Both ↑ → Symp.
- Both ↓ → HF

Splanchnic Circulation د. محمد فايز

Hepatic

Intestinal

	Hepatic artery	Portal vein
Flow	350 ml/min	1000 ml/min
PO_2	↑	↓
O_2 Required	30-40%	60-70%
Exercise	More O_2 taken	Less O_2 taken

Regulation of Both

Mainly Neural (Sympathetic)

VC (α receptor)

No VD fibers to Liver

Portal Venous Pressure

8-10 mmHg (Normal)

15-20 mmHg

Portal Hypertension

Cause ← Bilharziasis

Complications

- Splenomgealy
- Ascites
  - ↓ Albumin
  - ↑ Venous Pr.

Splanchnic Circulation → Portal Circulation → 30% of COP, Blood reservoir, low pressure

Other Circulations د. محمد فايز

Cutaneous (Skin)

No → Autoregulation

VD fibers

سـؤال: إزاى يحصل VD ؟

Sympathetic (+) → Sweat glands → Kininogen Kallikrein **Kinin** (VD)

Triple Response

	Red line	**Flare**	**Wheal**
Duration	5-10 seconds	30-45 seconds	3 minutes
Cause	Capillary dilate	Local Axon R.	↑ Capillary Permeability

Skeletal

Rest
6-8 ml/min/100 gm

Exercise
60-80 ml/min/100 gm

Increased Skeletal Ms Flow (Exercise)

Before	During	After
Hypothalamic Symp. VD center ↓ VD of skeletal ms Arterioles	↑ B.P ↓ (+) Baroreceptor ↓ Vasomotor Tone ↓ VD of skeletal ms Arterioles	Reactive Hyperemia (**Vasomotion**)

Circulatory Response to Exercise د. محمد فايز

	Isometric	Isotonic
Blood flow	↓	↑
HR	↑	
SV	Little change	
SBP	↑	
DBP	↑	↓
PP	No or little change	↑
MAP	↑	The same