Physiology Summary

* for the whole Make up lectures except excitable membrane

I wish you all good luck ☆

pray for me to pass anatomy exam :-

R.S

- Steps of respiration: ventilation → perfusion → exchange → transport → internal respiration
- Respiratory passages:
  conducting zone (Dead space)
  Respiratory zone (exchange zone)
- Alveoli cells → type 1 (squamous pneumocets)
  → type 2 (granular pneumocets) surfactant
  → type 3 alveolar macrophages + dust cell
- protective function of R.S
  ① protective reflexes → sneezing: nose
  → cough: trachea, larynx, bronchi
  ② lymphoid tissue in pharynx
  ③ >10 μ — nasal hair
  dust — <10 μ — mucous blanket (synthesized by goblet cell)
  ↓cell — muco-ciliary mechanism
  dust <2 μ + bacteria dust cell
- Respiratory cycle: Active inspiration — passive expiration (long) — pause
- Respiratory rate 12–16 respiratory cycle
- Tidal volume: 500 mL [0.5 L] of air inspired + expired per each cycle

	Active inspiration	Passive expiration
	respiratory centers active	respiratory centers stope
Cause	contraction inspiration ms contract diaphragm (moves down) contract external intercostal ms	relaxation inspiration ms relax diaphragm relax external intercostal ms
Intra-alveolar – pulmonary pressure	volume of lung ↑ Intra pulmonary pressure ↓ −1 mm Hg	volume of lung ↓ Intra pulmonary pressure ↑ +1 mm Hg

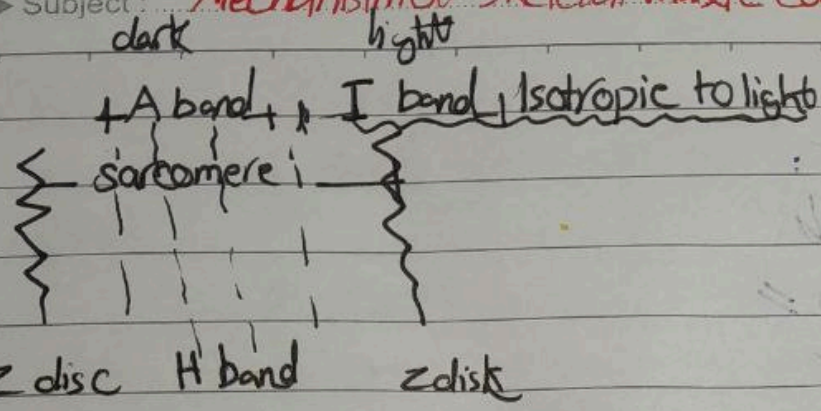
* respiratory pressure during pause phase = 0
* negative pressure in intra pulmonary pressure is caused by
tendency of lung to recoil + tendency of chest wall to expand → الصدر
1/3 ← 2/3 → elasticity of
stretched fibers surface tension muscles tendons tissues

state / respiratory P	Intra-alveolar pressure / Intra-pulmonary pressure		Intra-pleural pressure / Intra thoracic pressure	
Normal	inspiration −1 mm Hg	expiration +1 mm Hg	inspiration −6 mm Hg	expiration −3 mm Hg
forced	−30 mm Hg	+40 mm Hg	−12 mm Hg	
forced with closed glottis لسان المزمار	−80 mm Hg muller maneuver	+100 mm Hg Valsava maneuver	−30 mm Hg muller maneuver	+40 mm Hg valsava maneuver

* function of inspiration positive pressure (IPP)
1- venous / lymph return
2- continous lung expansion
3- measure lung elastisity
- Surfactant cortisol + thyroxin (24 weeks – 35 weeks)
function: ↓ surface tension (20–3 mm Hg) → ~~edema~~
amount of it in (large alveoli ↓ ~~rupture~~) (small alveoli ↑ ~~collapse~~)
↓ inspiration muscle effort, ↑ expiration duration ~~collapse~~
* Haylehine membrane disease (infintalie respiratory disease syndrome)
↓↓ surfactant formation in premature babies (cortisol ↓, throxine ↑, insuline ↑)
Diagnosis lecthine / Sphyngomyline < 1 . 35 weeks (2), 24 weeks (1) بالوضع الطبيعي

Excitation contraction coupling

Subject : Mechanism of skeletal muscle contraction / 4 jul



* (sarcoplasmic reticulum) Ca^{2+}
* (T tubule) action potential

* Actin filaments
G globules

* tropomyosin : protein which is cover Actin (active binding site) in relaxation

Ca^{2+} C — Troponin — T , tropomyosin

3 subunits

* Mechanism of muscle contraction

depolarization .

1. release Ach
2. Ach ∝ cholinergic receptors → EPP (end plate potential)
3. ↑ EPP (T tubules)
4. ↑ Ca^{2+} release
5. sliding of actin filaments between myosin filaments → muscle contract

Ca^{2+} + troponin C → ATP → ADP + P_i

ATPase .

motor end plate potentials MEPs . detach

Results of contraction

sarcomere → shorter :

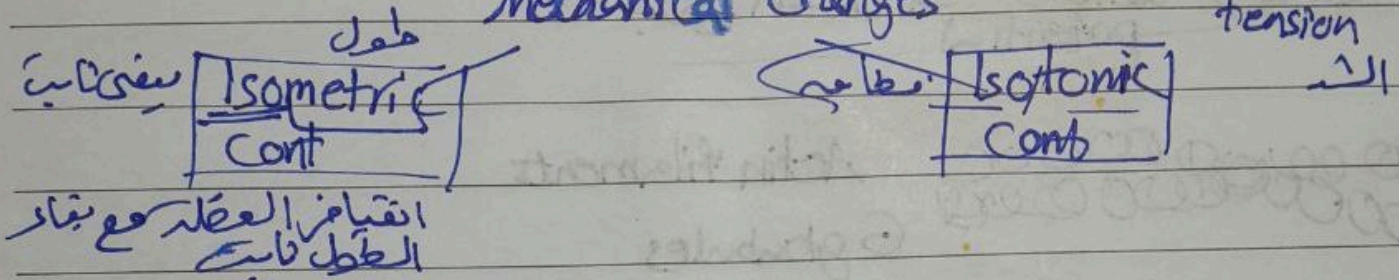
I band → width ↓

A band → constant =

H zone → narrower

M line → constant =

Results of Mechanical changes



انقباض العضلة مع بقاء الطول ثابت

Simple muscle twitch

latent period → contraction period → relaxation period

0.01 sec — 0.04 sec — 0.05 sec

Fatigue

gradual decrease in the muscle contraction + prolonged duration of all phases of simple muscle twitch especially relaxation due to repeated and strong stimulation of the muscle

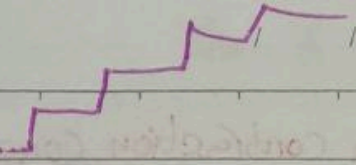
causes

- indirect: Ach
- direct: ① ATP ② metabolites

Contracture

Subject :

Staircase (Treppe) phenomenon



Due to maximal stimuli in just after relaxation period of each muscle twitch

↑ Ca^{++}

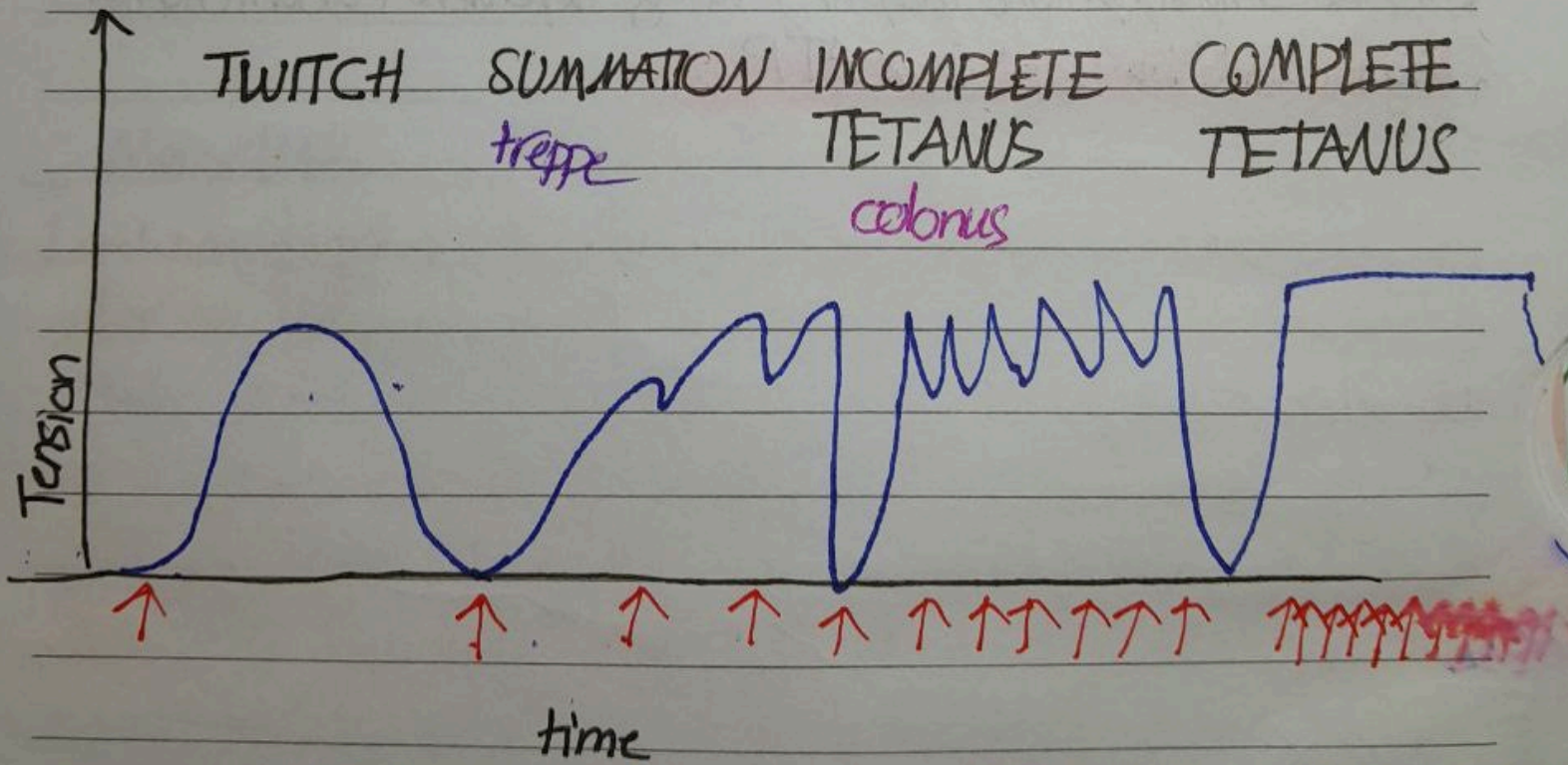
↑ tem

↓K^+ & ↑Na^+

Summation of muscle contraction

- 2 successive stimuli
- multiple successive stimuli

* absolute refractory period (latent period) مقيل تنبيب
relative refractory period (contraction) تنبيب اذا كان اكبر اقوى

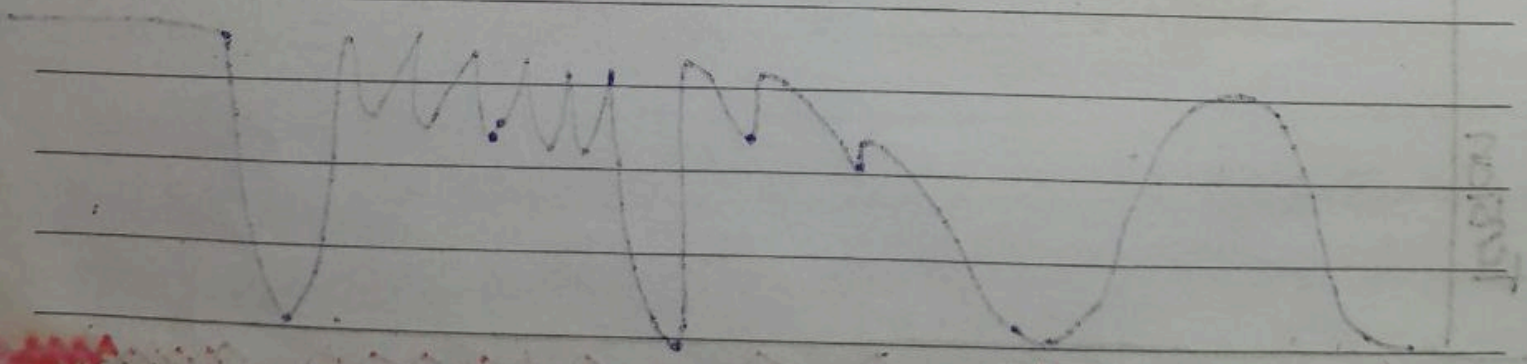


* Warmness will decrease all phases of SMT simple muscle twitch

* cooling + fatigue + anti-cholinesterase
colonus → complete tetanus

Subject : / /

* Excitation contraction coupling
  - series of events that link action potential (excitation) of muscle cell membrane (sacrolemma) to muscular contraction
  - process by which depolarization of muscle fiber intiate contraction
* (muscle contraction) (sliding of actin filiments between myosine filiments)
* action potential stages 1_ depolarization 2_ repolarization 3_ hyperpolarization 4_ refractory peirod
* refractory peiroid happens : absoutte refractory peirod _ theirshold level , relative refractory peiroid occurs after th
* Triad (tt tubules () 2 cisternae)
* DHPR (dihydropyridine receptor) Voltage dependent calcium channel
* Rigor caused by Depletion ATP



▸ Subject : Neuromuscular junction Thu / 6 / Jul

Neuromuscular junction = motor end plate

contain

- motor nerve
  - Ach vesicles
  - exocytosis
  - mitochondria
  - dense bars
- synaptic cleft
  - Ach
  - cholinesterase enzyme
- muscle fiber
  - invagination = synaptic gutter = subneural cleft
  - Ach
  - ligand gated channel
  - Nicotonic cholinergic Receptors

cholinergic

- Muscarinic
- Nicotonic
  - skeletal muscle

_ Motor UNit

1 ant horn cell (neuron) + axon + muscle fibers

all or non law

_ Motor POOL

all Ant horn cells + skeletal muscle

~~all or non law~~

* Miniature end plate potential

Ach vesicles ← subthreshold depolarization in motor end plate

Synaptic delay 0.5 msec

Neuromuscular transmittion

Stimulators

Ach

Anti-cholinstrase

Carbachol كاربا خول

prostigmine بروستيجمين

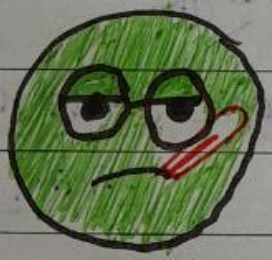
Blockers muscle relaxing

ACh competitive Curare

Succinylcholine سكسينيل خولين

depolarization block

absolute refractory period فترة صمّاء مطلقة



Myasthenia Gravis

hereditary disease cause muscle weakness

Causes

↓ Ach

↓ Ach receptors

↑ cholinesterase enzyme → Anti cholinstrase prostigmine

Curare

Ach & ∝ antibody → thymus removal

activation chance → Cortisone

Subject : Autonomic Nervous System 9 / 7 / 2023

Autonomic Nervous System

Sympathetic
thoraco-lumbar outflow

Para-sympathetic
cranio-sacral outflow

1) Cervical : Head + Neck
- Eye: dilator pupillae muscle → mydriasis; ↑ palpebral fissure; relax ciliary ms
- Glands: ① lacrimal → vasoconstriction
  ② salivary: trophic secretion viscid
- Skin: - eccrine glands - erector pilae ms - B.V
- cerebral vessels: • vasoconstriction • arterial blood pressure
  blood flow =

2) Cardio pulmonary : thorax
- Heart: cardiac properities ↑ - muscle
  coronary vessels: direct constriction, indirect ↑ metabolism dilatation
- Lung: - bronchodilation, while pulmonary vessels vasoconstric → widining of air passage

3) Splanchnic : Abdominal + pelvic viscera
B.V: contact, relax liver

Abdominal

GIT tract: walls relax, sphincters constriction

GIT secretion

splanchinic vessels - vaso constric
- vaso dilatation . liver

Spleen 250 ml poured into circulation

liver glycogenolysis + lipolysis vasodilatation
↑ blood clotting factor

endocrine . pancreas . inhibition insulin secretion

Kidney renal blood vessels vasoconstric
renin ↑↑↑

adrenal medulla . (80% adrenaline + 20% noradrenaline)

pelvic

urinary bladder wall of urethral sphincter ... inhibition

rectum ... = = anal = ... inhibition

female sex organ inhibit . utres + fallopian tube
late pregnancy in exception

male sex organ ejaculation

Somatic Skin + blood vessels of skeletal muscles

Subject:

skin

sweat glands Eccrine copius

Apocrine thick odoriferous secretion

cutaneous blood vessels vasoconstriction

Erector pilae muscles contraction

skeletal muscle blood vessels vasodilatation + ↑ metabolic process (↑ contraction, ↓ fatigue, ↑ recovery) orbelli phenomenon

Sympathetic nervous system functions during rest

Sympathetic tone of blood vessels mild vasoconstriction to maintain bP

Sympathetic tone to Adrenal medulla basal amounts of catecholamines

parasympathetic Cranio_sacral outflow
1973 Lateral horn cells
2,3,4

cranial outflow 1973

GIt inhibit sphenctar

10 Vagus cardiac coronary vessels direct vasodilation indirect vasoconst
pulmonary bronchoconstriction ↑ secretion, vasodilation of vessel → ↓ air pass

9 glossopharyngeal
لعابي لساني
parotid salivary gland

7 facial nerve. lacrimal glands vasodilator
submaxillary salivary glands (true secretion)

3 oculomotor @ constrictor pupillae (miosis) @ ciliary muscle contract
يعمل بالعين

salivary glands → submaxillary — facial nerve
parotid — Glossopharyngeal nerve

Sacral flow lateral horn cells 2,3,4
Defecation contract rectum / relax sphincter
Micturation contrat urinary bladder, relax sphincter
erection vasodilation erctile tissue of peins

parasympathetic tone (vagal tone)
vagal tone of Heart
vagal tone of GIT
vagal tone of bronchi

constrictor pupillae muscle parasympathetic
dilator pupillae muscle sympathetic

Sensory Division

- Stimulus transducer receptors → action potential
- sensory receptors at the termination Afferent nerve on dorsal root ganglia

sensory Receptors

According mode of stimulation (طريقة التحفيز)

1. Meehano — in touch (اللمس), audetoriy, muscles, ligaments joints + baroceptors (مستقبلات الضغط)
   - pacinian corpuscle
2. chemo — taste (التذوق), olfactory (الشم), O_2 lack, CO_2 excess, osmoreceptors
3. Electromagnetic — light in retina (الضوء موجات كهرومغناطيسية)
4. Therme — skin + Hypothalamus
5. Noci — free nerve ending receptors for pain sensation

According to site

- exteroceptors
  - found in skin
  - therme
  - touch
  - pain
- Teloceptors
  - special sence
  - distanto receptor
    - visual
    - olfactory
    - auditary
- Interoceptors
  - Viscro
    - -strech
    - -O_2 lack
    - -baro
  - hypothalmic
    - - thermo
    - - glyco
    - - osmo
  - proprio
    - ones own motion
    - - muscles
    - - lig
    - - joints

properites of sensory receptors

- Excitability
- specificty
  - modality
  - Muller's law
- effect of intinsity of stimuli
  - Webber Fechner law
- Adaptation

Excitability specificity modality Muller's law effect of intinsity of stimuli Webber Fechner law Adaptation

Adaptation → rapidlly, moderetelly, slowely

▷Excitability: stimuls → sensory receptor → generatore potential /sensory potential (local depolarization ~~propegated~~ caused Na+ influx) ~~absolute refractory peiod~~ (once stimuli continued (summation) > 5ms → nerve → action potential → nerve → CNS

~~all or non law~~

-Sense organ (sensory receptor + non nueral cell) – {free nerve ending}

Muller's law of specificty

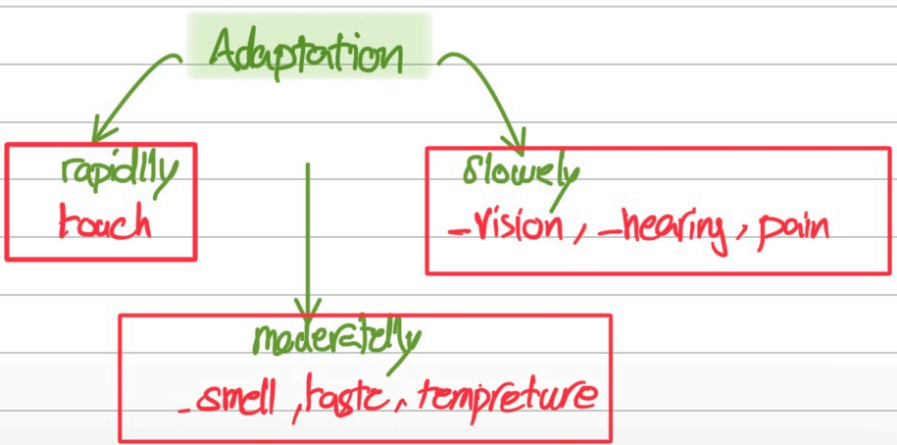
"each receptor is very sensitive to one specific type of stimuli called the "adequate stimulus" which can stimulate the receptor with least amount of energy".

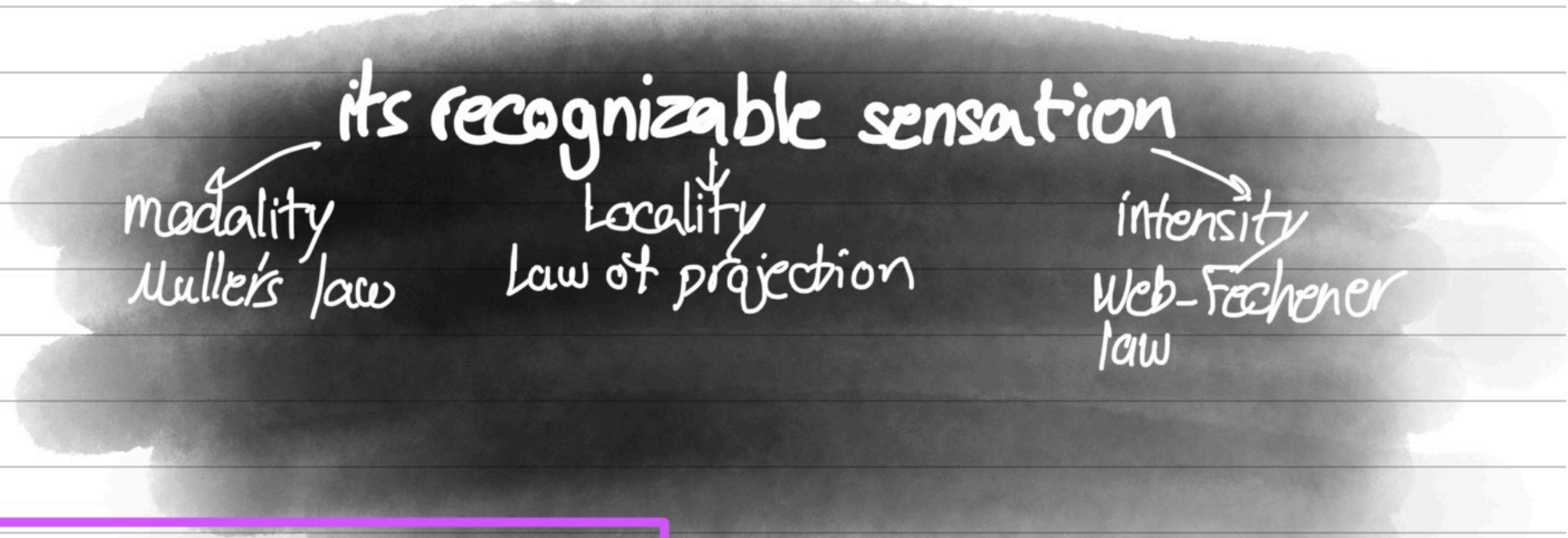
Weber Fechner law of intensity

"The frequency of the action potentials conducted a long the afferent nerve fiber is directly proportional to logarithm of the intensity of the stimulus applied to the receptor"

Adaptation

the decrease in the intensity of the sensation due to continuous constant stimulation.





Law of projection **Locality**

If we stimulate sensory pathway along its course to the sensory cortex, the conscious sensation produced is perceived to be from the location of receptors.

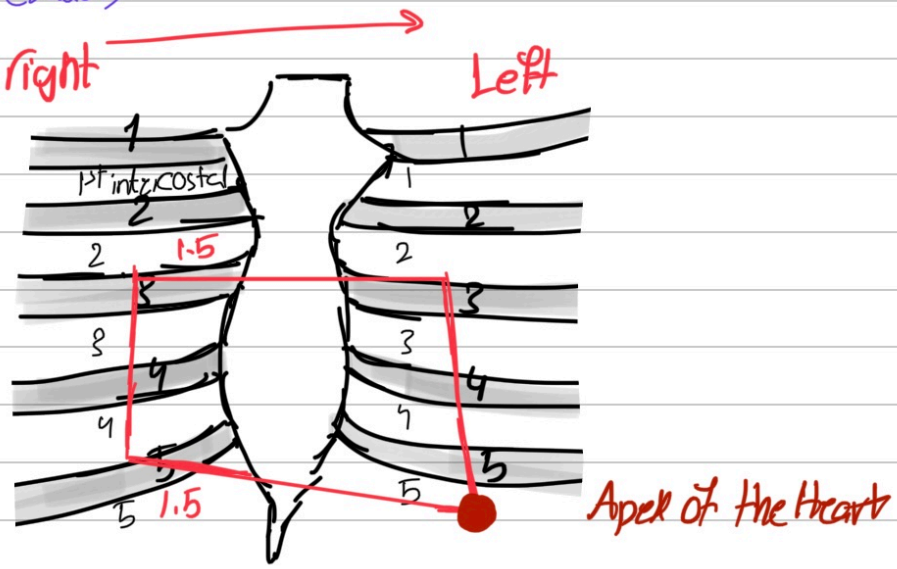
according dermatomal distribution

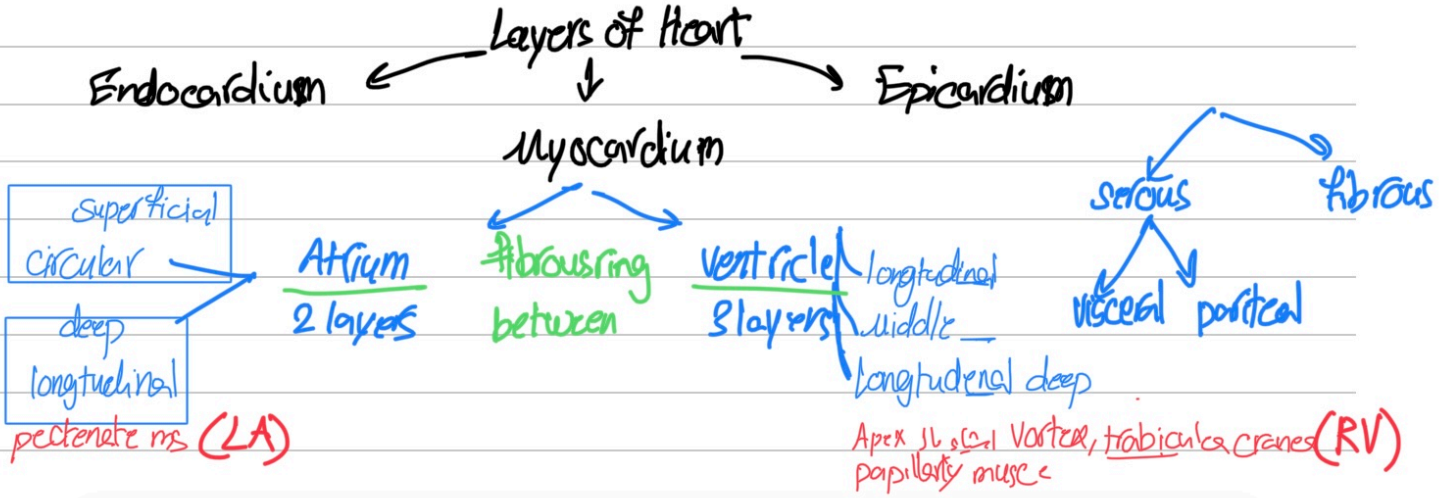
* Atrioventricular valves : Tricuspired valve + Mitral (bicusbed valve)
open : Atria fills (pressure aginist valve) as atria contract it continoue open
close; ventricle contract (blood aginist valve) + papillary muscle + cordenate tindinea, tighten

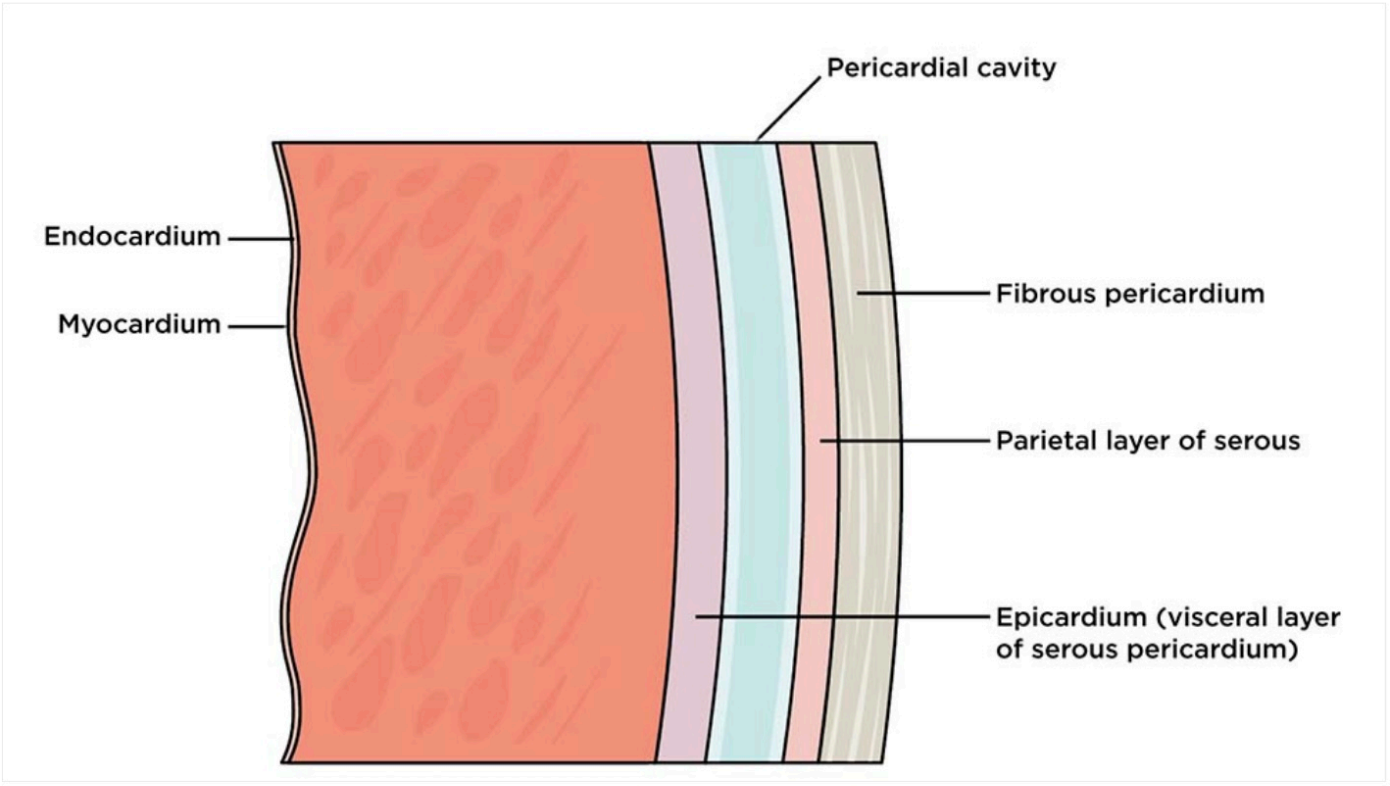
* Semilunar values : pulmonary valve + aortic valve
open : ventricle contract (intraventricular pressure ↑)
close : ventricle relax (intraventricular pressure ↓)

* clinical topography of heart

Holotopy	ēgo
Syntopy	Anterior, posterior (esophagus + vesocleture), superior (greater blood vessels), inferior, lateral
Sceleotopy	relations (borders)







* Fuel of the Heart

Glucose, Lactate, Fatty acid, Amino Acid

Glucose, Lactate → (كسر) → pyruvate

Fatty acid → (كسر) → beta oxidation

Amino Acid → non essintial

→ Ketogenic (Lycine, leucine)

صايم >< → Ketone bodies

Angina Pectoris ذبحة صدرية

cause: sternous activity → Ishemia نقص تروية

علت نشاط رشفه قليل احتاج دم واكسجين اكثر

Nitroglycrine هي تحت اللسان

Myocardial Infarction MI (Heart Attack) احتشاء القلب

cause: death of Myocardial Muscle (اعتلال) → Scar tissue

Frank starling mechanisim and mecanical efficiency

Frank starling mechanisim "preload + strok volume" S.V depends on EDV

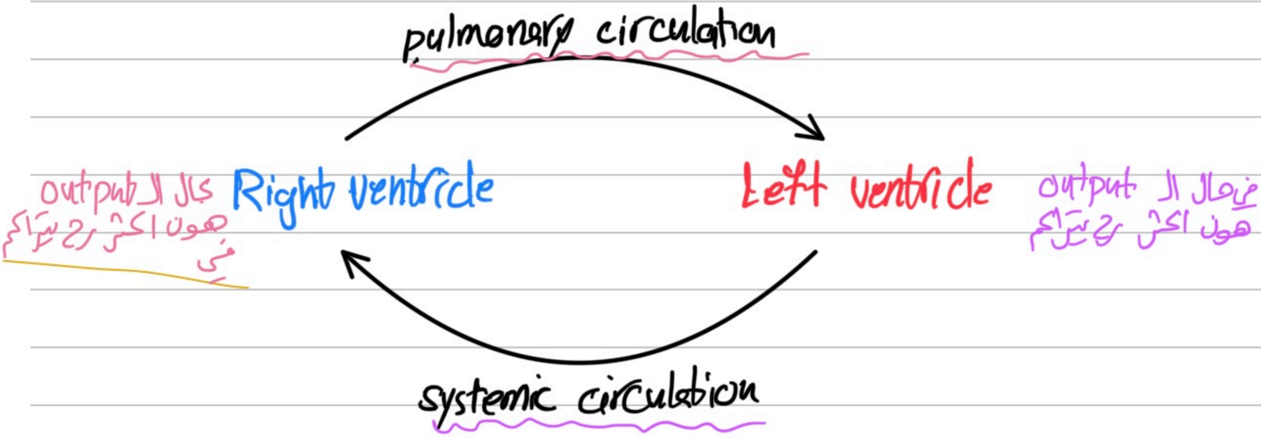
"Volume of blood ejected by the ventricles depends on the volume present in the ventricle at the end of diastole"

underlying principle: there is relationship ( ) Length of sacromere + tension of muscle fiber

* ↑ EDV → ↑ number of streching myocardial fibers → ventricle stimulated to contract

↑ tension of muscle fiber → ↑ force of contraction → ↑ S.V → ↑ CO

* Right ventricular output = left ventricular output



- $S.V = EDV - ESV$
- $CO = (EDV - ESV)\,HR$ (flow Q)
- $CO = S.V \times HR$

$S.V \propto$ preload + contractility , $S.V \propto \frac{1}{\text{Afterload}}$

(streching قديش رح يتمدد) (T3, T4, glocagon ∝) (work to pump)

$BP = CO \times TPR$

$V_{velocity} = \frac{CO}{A}$ (flow / cross sectional area)

Aorta ↑diameter/↓Total corss sectional area/↑ velocity

Arteiriole ↓diametere/↑↑Total corss sectional area/↓↓ velocity

the velocity is slowest in capillary & ↑total cross sectional A

$$Q = V \times A$$

flow/cardia output remains constant at all vessels

Hypoproteinemia

نقص البروتين بالدم

Causes (albumin, lipoprotein, antithrombosis III, gout)

1- Nephrotic syndrome
2- liver disease: Liver is produced plasma protein
3- Malnutrition: ~~protein~~
4- Malabsorbtion: protein الامتصاص
5- serve burns

Motor

Reflex action: sudden + involuntary action as response to stimuli

Reflex arch: nervous pathway of Reflex action

Reflex arch: receptor → Afferent neuron → Center → Efferent neuron → Effector organ

Types of Reflexes

Monosynaptic reflex arch

~~inter neuron~~

Strech reflex

polysynaptic reflex arch

interneuron ✓

light reflex

properities of Nervous Reflexes

1. UNIDIRECTIONAL Law of forward direction
   (Afferent (pre-synaptic neuron) Synaps Efferent (post-synaptic neuron)
2. LOCALIZATION (specifisty) specific stimulus → specific receptor → specific stimulus
3. TOTAL REFLEX Time monosynaptic → very short time / polysynaptic → long time
   central delay 0.5ms central delay > 0.5
4. IRRADIATION extent of reflex responce depends on intensity of stimulus
   Flexion, Flexor withdrawal reflex, crossed extensor reflex (عكس رزنيم)
5. FATIGUE انخفاض الاستجابة بتكرار الحدث
6. RECIPROCAL INNERVATION — { positive supporting reflex } support body weight
   contraction of one group muscle + relaxation inhibition of antagonistic group
7. RECCRUTMENT AND After Discharge to study it (reflex tetanus + Motor tetanus)

Reccrutment: gradual activation of Anterior Horn cells ↑AHCs

After discharge: presistant discharge from efferent neuron after stimulation stops of stimulation

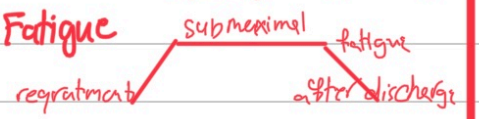
اسمرار التفريغ من العصب الحركي حتى بعد توقف المحرّ

REFLEX TETANUS	MOTOR TETANUS
maximal repetitive stimulation of Afferent	maximal repetitive stimulation of Efferent

REFLEX TETANUS

maximal repetitive stimulation of Afferents

- latent period ↑
- Recruitment (gradual)
- After discharge (gradual relax)
- Fatigue



MOTOR TETANUS

maximal repetitive stimulation of Efferents

- latent period ↓
- muscle contraction (RAPID)
- rapidly relax

HUMAN REFLECSES

perephiral reflexes

like GIT re-exes

- entric reflex
- gangilionic reflex
- axon reflex

Central reflexes

condition (corticol)

- intact cortical cerebral cortex
- specific stimulus

Uncondition (inborn)

- subcortex
- fixed stimulus

spinal cord ← ↓ → Hypothalamus

brainstem

medulla ← → pons/midbrain

Resistance and capacitance

- mechanical effeciency of Isotonic contraction = 20-25%
- Ejection fraction = 60%

(percentage of total blood that is pumped by your heart while each beat)

$$Force = \frac{\Delta P}{R}$$ Ohm's law ← من وين جابوا كانون خفيف الدم؟

$$Flow(CO) = \frac{BP}{R} \rightsquigarrow BP = CO \times (R)\ Resistance$$

$$R = \frac{8 n L}{\pi r^4}$$ (posillyus law)

n: viscosity (polycythemia n↑) (animea n↓)

L: length

r: radius (the main factor that is determine R) غير تغير

Tension = pressure x radius → mainteine tension constant

$$Compliance = \frac{\Delta V}{\Delta P}$$ (dilated cardiomyopathy / compliance ↑)

$$stiffness = \frac{\Delta P}{\Delta V}$$ (Ventricular hypertrophy / stiffenes ↑)

Compliance = distensability x original volume

(compliance of vein 24 times compliance of arterys)

* Capacitance Blood vessels are the veins (blood Resirvoar) compliance ↑↑

$$Conductance = \frac{1}{resistance} = \frac{Flow(CO)}{P}$$

← Blood flow →

laminar flow — turbelent flow

Blood flow

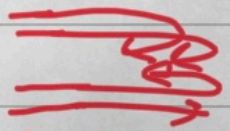
laminar flow

طبيعي silent

flow ↑ perfusion pressure ↑

low resistance

turbelent flow



مرض bruits

murmurs

flow ↓ perfusion pressure ↑ بصارعله

high resistance

BP

La place Law for myocardial O_2 demand (VO_2) : $VO_2 \propto T$

O_2 demand $\propto$ wall tension

wall stress (σ) $= \frac{\text{pressure} \times \text{radius}}{2(\text{ventricular wall thickness})}$ (force/unit area)

wall tension = pressure $\times$ radius (force in entire wall)

$BP = CO \times TPR$

$CO = HR \times S.V$

$R = \frac{8\eta L}{\pi r^2}$

* Pressure gradiant ΔP = Higher pressure P_1 − lower pressure P
* pulse pressure = Systolic pressure − diastolic pressure = $120 - 80 = 40$ mm Hg

ΔP (perfusion pressure) = MAP − CVP

Mean arterial pressure — center venous presure

$\Delta P = MAP$

(CVP central venous p) ("right artirial pressure (RAP)") = 3 → 8 mm Hg

MAP → systolic pressure 120 mm Hg
→ Diastolic pressure 80 mm Hg

MAP = 93 mm Hg

MAP = diastolic pressure + $\frac{1}{3}$(pulse pressure)

MAP = $\frac{\text{systolic pressure} + 2(\text{diastolic pressure})}{3}$

MAP = $\frac{1}{3}$ SBP + $\frac{2}{3}$ DBP

Microcirculation notes

MAP↑, P↑, R↑ (vasoconstriction)

Brian

MAP↓, P↓, R↓ (vasodilatation)

flow constant ($flow = \frac{P}{R}$)

flow constant ($flow = \frac{P}{R}$)

Other tissue

PO_2↓ → vasodilatation

(carcinoma)

Lung

PO_2 vasoconstriction to ↓ blood shunt

Metastasis in epithelial tissue is speared by lymphatics

Metastasis in connective tissue is speared by blood

sarcoma

Lymphoden__ lymph node

cortex: B cells

para cortex: T cells

medulla, plasma cells

medullary sinus: Macrophage

Subject: cardiac cycle + heart sounds

Remember:

* Systole → heart contract
* Diastole → heart relax

} 2 phases of cardiac cycle

* EDV quantity of Blood in the left or right ventricle at the end of diastole just befor systole starts
* ESV the amount of Blood remaining in the ventricles at the end of systole after the heart has contracted
* EDV − ESV = Stroke Volume S.V
* Stroke Volume: volume of blood pumbed by heart / Beat
* COP the amount of Blood pumped by the heart by minute

COP = S.V x H.R mL/min

* Ejection Fraction % Blood pumbed by beat

$E.F = \frac{S.V}{EDV} > 55\%$

ECG electro — depolarization of ventricle

الكهرباء تتبع العقل الحقيقي بالعلن

depolarization — QRS

(Late diastole)

Atrial Systole: Atrial contraction phase

① Atrial contraction phase 30% blood pumbed

70% → passivly without contraction

Atrial pressure ↑↑ 4 → 8 (contract) evacuation → 4

Ventricular pressure ↑↑ 4 → 8 (نبضي دم قاعه) accumulation caused by elasticity → 4

4th heart sound _ normally inaudible

② Rushing of blood in ventricles

② Contraction in Atrium caused vibration in atrial wall (تذبذب اهتزاز)

Semilunar valves closed / A.V valves _ opend

EDV = 140 mL EDV

0.1 second

B O O X

Ventricular Systole — 1st heart Sound

Isovolumetric contraction — closure of AV valves, 1st component

ventricles اكبر عضلة contracts اول مرحلة semilunar — 0.05 Second

valves مغلقة blood is incompressible

(muscle fiber length) ولا (تقصير) يعني الانقباض بدون ما يتغير طول

(AV) bulging — ventricular contraction

مرحلتها كامل 0.3 Sec

begins to contract Isometrically

4 valves: closed

atrial pressure ↑ bulging of valves

Ventricular pressure ↑↑↑ (contract) — regarding of blood — 4 → 80 mmHg

ventricular volume remains constant

Rapid Ejection phase — 1st heart Sound: vibration + rushing of blood Aorta, 2nd component

Semilunar opened — 0.15 Second

AV valves Closed

70% S.V ejected on cardiac cycle in one heart beat

ventricular pressure ↑ 80 → 120 mmHg

ventricular volume ↓↓ ejection

Isotonic contraction → shortening sliding

Atrial pressure ↓ (down displacement) of valves AV — bulging

Strok volume = EDV − ESV

Subject: 70 = 140 − 70

Reduced Ejection phase

30% remein blood of stroke volume دا للهوا دفعة دفعة

reach maximum ventricular + Aortic pressure — 0.1 SECOND

Begins of decrese pressure 120 mm Hg Escape of blood to peripheral circulation

Atrial pressure ↑ → venous return

تبدأ القلب يلهو فيتفضى هناك من ضخ محبا

Atrium يرجع IVC SVC يرجع دم من S.V

طلع كل الدم الذي يمكن اخراجه فيتبقى للاقل كمية دم

هيكون بيضل حوالي ESV 70 ml

Diastole عالم ال

Protodiastolic phase: 5th phase — 0.04 SEC

① فترة فاصلة بين نهاية ال contraction وال ventricle

closure of Aortic valve هتتسمى باللي بعدها

② ventrical volume remains constant

اللي طلع طلع ومفيش حاجة بترجع

closure of Aortic valve → dicrotic notch

ventricular pressure ↓ about (20 mmHg) قد ايش بتنقص

Aortic pressure ↓ but still > pressure of ventricle

phase 6

Iso_volumetric relaxation of ventricle

semilunar valves

volume of ventricle remain constant

Atrial pressure ↑↑ venous return atrial.

ventricular pressure ↓ 90 → 0

0.06 SEC

Second Heart Sound caused by closure of semilunar valve

All valves are closed

→ elastic recoil Aorta pressure ↑

phase 7

Maximal filling (rapid) phase

60%

open of AV values Atrial pressure > ventricular p

passively under pressure gradient (AV values

→ pressure in the ventricle = pressure in the atrium → 0

chambers

inaudible in adults ↓ Aortaic pressure

0.1 SEC

third Heart Sound caused by rushing of blood into the ventricles + vibration of ventricles wall

Atrial pressure ↓↓ ?

BOOK

▸ Subject : Heartsound

phase 8

Reduced Filling phese

10% slowly

↑↑ pressure in Ventricle gradually 0 → 4

0.2 SEC
longest duration
in cardic cycle

Cardiac cycle : period from the end of systole (Heart contraction) to the end of next sistole 0.8 SEC

HR = ~~75~~ beat/menit

cardiac cycle $\frac{60 \text{ sec}}{75 \text{ beat}}$

= 0.8 SECOND

Cardiac cycle = $\frac{60}{\text{heart rate}}$

0.8 (Ventrick (systole 0.3, diastole 0.5)
Atrium (systole 0.1, diastole 0.7)

cardiac cycle phases

1- Atrial systole 4th
2- Isometric Ventrick contraction 1st 1st
3- Rapid ejection 2nd 1st
4- Reduced ejection
5- proto diastolic
6- Isometric relaxation 2nd
7- Rapid filling 3
8- Reduced filling

▸ Subject : **Heart sounds**

First Heart sound 0.14→0.16

First component Iso volumetric contract

Closure of AV valves →

Vibration . valves . blood . Ventricle walls

Second component 1st part of Rapid ejecti

Vibration . artery walls . rushing of blood

Lubb 25_100 cycle/sec

Mitral 5th Left intercostal space at mid clavicular line (apex)

Tricuspeal lower right border of sternum

Second Heart sound 0.1

Isometric Relaxation phase

Closure of semilunar values

Dup 100-200 cycle/sec

splitting is very close heart as one

Aortic 2 ICS right (near sternum)

pulmonary 2 ICS Left (near sternum)

Third Heart Sound 0.05

Rapid filling phase

Vibration . ventrick → rushing blood

can be heard in children +

hyper dynamic circulation

Mitral area

Fourth Heart sound 0.04

Atrial contraction phase

atrial contraction

non audiable _{ atrial hypetrophy

Mitral area

at beginning in ventricular systole

Atrial contraction → Iso metric contraction → Rapid ejection → Reduced ejecti

S4 1st part S1 2nd part S1

→ prediastolyic → Iso metric relaxation → Rapid filling → Reduced filing

S2 S3

at begining in ventricular diastole

S1 → S2 systolic period S2 → S1 diastolic period

GMAN OTE BOOK

Heart sounds

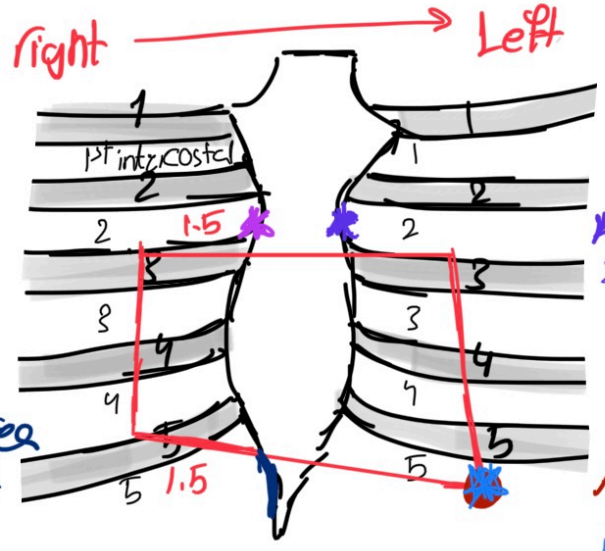
S4 — phonocardiogram (record)

S3 (0.05)	S4 (0.04)
in childeren	recorded
hyperdynamic circulation	Atrial hypertrophy

S1, S2, S3 – stetho – scope (near)

lubb	DUB
closure AV values S1	S2 closure semilunar values sippliting but it is heared as one
low pitched	High pitched (Weak)
soft	sharp

lubb	DUB
closure AV values S1	S2 closure semilunar values sippliting but it is heared as one
low pitched	High pitched (أعلى)
soft	sharp
long duration	shorter
0.14 → 0.16 sec	0.1 sec



* Aortic area
2nd right space near sternum

* pulmonary area
2nd left space near sternum

o Tricusped area
Lower right end of sternum

Apex of the heart * Mitral area
mid clavicular line ⊥ 5th intercostal space

* Aorta أصلا جاي من
* pulmonary artery لل

	S1	S2	S3	S4
Stethoscope site	Mitral Tricusped area	pulmonary Aortic area	Mitral area	Mitral area

Vascular Lec Notes

* why arterioles called resistance vessels?

cause they determine perphiregal resistance + arterial blood pressure

* which types of vessels determine Blood flow? arteriole by VC + VD

* why arterioles have drop in BP from 80 → 30 mm Hg? cause they have great resistance

remember • The greater the change in resistance at any point in the vasculature, the greater the loss of pressure at that point. ★★★★

Factors regulate arterioles diameter

Central/systemic Regulation

Neural regulation

- vasoconstricter fibers
- vasodilator fibers
  - sympathetic
  - parasympathetic
  - Antidromic vasodilator impulses * local axon mechanism substance P

* parasymphathetic
  - VC coronary
  - ↑ PO_2,
  - ↓ metabolites
* sympathetic inderict
  - VD coronary

Local Regulation Mechanisim

Hormonal regulation

- Catacholamine — VC
- Renin – Angiotensine system — VC
- Antidiuritic Hormone (vasopressin) ADH — VC
- endothial drived relaxing factor
- Kinin → EDRF / plasma Kinine → brady kinin, tissue Kinine → Kallidine — VD
- Atrial Natriuretic peptide (ANP) — VD

Local Regulation Mechanism

Local tempreture
- ↑temp → VD. (الاصل عند ارتفاع الحرارة)
- ↓temp → VC

PO_2
- ↓PO_2 → VD except lung VC
- normal → partial VC

Metabolites
- CO_2, K^+, adenosine
- osmolality
- acidosis

Autoregulation: intrensic mechanism
- Myogenic theory: ↑flow → stretch vessel, depolarization → contract → ↓flow
- Metabolic theory: ↓flow → ↑metabolites → VD → ↑flow

Local vasoconstriction substances
Seratonin which is produced by platlets to injured arterials

endothelium substances

Thrombaxine A2 | prostacycline
- platlets aggregration
- VC

- prevent platlets aggregration
- VD

endotheline
released when the vessels strech
VC

EDRF
endothelium drived relaxing factor

VD ← relaxing من اسم
↑ cGMP
EDRF → hypertension, atherosclerosis, impotence

☆ Thrombaxine a2 + sertonine is released by platlets both VC substances

V.C	V.D
Decrease CO_2 tension ,acidosis, osmolality, K^+, and adenosine. metabolites ↓ / ↑PO_2	Increase CO_2 tension ,acidosis, osmolality, K^+, and adenosine. ↑ metabolites / ↓PO_2
↑PO_2 High O2 Except Lung	↓PO_2 O_2 lack Except Lung
stretching the vascular smooth muscles Endothiline	
Drop in the tissue temperature	Increase in the tissue temperature
Thromboxane- A2, serotonin released by platelets	**Prostacyclin, EDRF** Endothelium drived relaxing factor
Sympathetic V.C Vasomotor tone	sympathetic innervations to bl. Vessels of Coronary vessels, Skeletal muscles, The splanchnic areas, Sweat glands
Parasympathetic **coronary vasoconstriction** **indirect effect**	Parasympathetic to **genital organs** (sacral out-flow).
adrenaline and nor-adrenaline, Angiotensin-II , ADH	**Antidromic local axon reflex**
	Kinins, ANP Atrial Natriuretic peptide