Blood Pressure Responses to Exercise. 16/03/23.

- Upperbody Response
- Lower body Response
- Steady state Ex. Jogging cycling:
  - > Arterial BP ↑

(Why) ↑. Active Muscle Vasodilatation.

① · Blood supply ↑
· Total Peripheral resistance ↓

② Muscle alternate contraction & Relaxation.

③ Phenomena :- Sy. BP. ↑ initially slightly
↓.

④ ∴ Diastolic B.P. par koi fark nahi padta.

Steady state Exercise:

During Rhythmic muscular Activity.

(e.g.) - Jogging, cycling, swimming, running in steady state.

↓

[vasodilation in active muscles.]

↓

[Decrease in Total Peripheral Resistance]

↓

[Increase in Blood flow large portion of peripheral ~~musculature~~ / vasculature (vessels.)]

&

- Alternate Muscle contraction & Relaxation. And also provides an effective force to propel Blood forward through vascular circuit & return into heart.
- Increased Blood Flow during steady state ex. Rapidly ↑ systolic B.P. during 1st few min. of ex.

↓

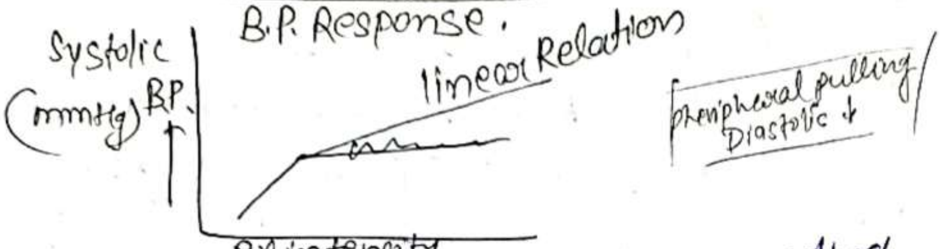
As ex. continuous, systolic Pressure gradually

declines. due to
Reason: Peripheral Return less.

Reducing pheral Blood Resistance to flow.

· Diastolic B.P. Remains unchanged through out the steady state ex.

Graded Exercise



After initial rapid rise from resting level systolic B.P. ↑ linearly c̄ ex. intensity. Diastolic B.P. remains stable or ↓ slightly at higher level.

Resistance Exercise:

(HTN) isometrics Not allowed. (small ms.)
long term ex. for strenthening nahikarani hai.

B.P. Response:

· Straining ex, particularly concentric (contract) or static phase of muscle action, mechanically compresses the peripheral Arterial vessels that supply active muscles.

· Arterial vascular compression dramatically ↑ses total peripheral resistance & reduces ~~peripheral~~ muscle perfusion.

· In an attempt to restore the muscle Blood flow, substantial ↑ occurs in SNS activity, cardiac output, Mean arterial pressure (MAP).

- The magnitude of hypertensive response relate directly to intensity of effort & quantity of ms activation.

- Resons.

Increase in Arterial BP due to:

1. Greater Stimulation of cardiovascular centre by active areas of motor cortex.
2. Large peripheral feedback to the centre from contracting muscle mass.

Upper Body Ex's

Blood Pressure response:

Ex's c̄ arms produces considerably higher Systolic B.P & Diastolic BP. than leg exercise performed at a given persantage of VO_2 max. In each form of exercise.
∴ This occurs b'cz the smaller arm mus. mass & vasculature offer greater resistance to Blood flow then larger leg mass & Blood supply.

B.P. Responses In Recovery

Vital at base line (Returning) (HR/RR/Breathing/Pulse) 5 min.
Hypotensive Response (10hr) me milta hai.

IN Recovery

upon The complition of single bout of submaximal exercise. B.P. Temporarily fals below pre-exercise level for Normotensive & hypertensive individuals.
(Reson: peripheral dilatation)
Hypertensive individuals from an unexplained ~~from~~ peripheral vasodilatation.

The hypotensive response can lasts upto 12 hours.
Another explaination for post exs hypotension is that significant quantity of Blood ... remain cooled in visceral organs & skeletal muscle vasculer beds during recovery.

[Venous cooling]
↓
[↓ Central Blood volume]
↓
[Reduces arterial filling pressure]
↓
[Lowers systemic arterial B.P.]

vasodilation — to maintain Blood supply: [Release of arterial Natriaceuretic peptide hormone.]
→ Potent vasodialator hormone.
Does not account for post exercise hypotension.

Heart Blood supply

Coronary arteries

Right main coronary artery
- Arises from upper part of ascending Aorta.

Rt. coronary artery supply predominantly Rt. ventrical / Rt Artium.

Left main coronary artery
Arise: upper part of ascending Aorta
Supplies: Lt ventrical
Lt Artrium.
Mild part Rt ventrical.

Muscle Supplies and their use

▷ Myocardial supplies:

At Rest myocardium uses (extracts) about 70-80% of oxygen from blood in the coronary vessels. During Vigorous exercise coronary Blood flow ↑ses 4-6 Times above resting level.

Blood flow ↑ses b'cz:

- Elevated myocardial metabolism dialates coronary blood vessels.
- Sympathetic mediated artiriolar vasodilation.
- Arterial B.P. also facilitates coronary bloodflow.

Effects of Impaired Blood supply

BP↑ Arteriosclerosis ⇒ Loss of fibers. walls hardening.

Impaired Blood supply ← Artherosclerosis → deposition of Plaque.

- The myocardium depends on an adequate Oxygen supply because it has limited anaerobic energy generating capacity.
- Tissues hypoxia provides a potent stimulus to myocardial blood flow.
- Impaired coronary blood flow usually causes chest pain termed as Angina pectoris. (condition: MI)

Rate Pressure Product (RPP) (Estimate of myocardial product) (O_2 consumption)

"sports rpp"

$$RPP = SBP \times HR$$

- It is an estimate of myocardial work load.
- It is the product of peak systolic pressure.

at brachial artery & Heart Rate.

- It relates closely to directly measured myocardial oxygen consumption & coronary Blood flow in healthy individuals.

- At Rate Rest = 6,000 (value)

  120 X 50 = 6000 mmHg/bpm.

  In Athletes = 40,000.

  at ex's. 200X200 = 40,000 mmHg/bpm.

Upper body ex's (↑ HR)

Resistance
↑ ex's.

UNIT - Microbiology on the horizon

- Upper body ex's produces higher Heart Rate & BP responses than more rhythmic ex's with lower extremity.

23/March/23.

Cardivascular Regulation & Integration.

SA-Node (Sinoatrial Node.) Pacemaker whole tissue cld.

Sinus → coronary sinus.
Drainage artery coronary tissue.

Property: Autorhythymacity.

SA → cause ayenge pacemaker tissues
↓
AV (Atrioventricular).
↓
Bundle of Hiss
(Rt) (Lt)
↓
Purkinje fibers.

Some part of the Hrt Not function but Heart will not stop.
Rhytham zero Nahi hogi.

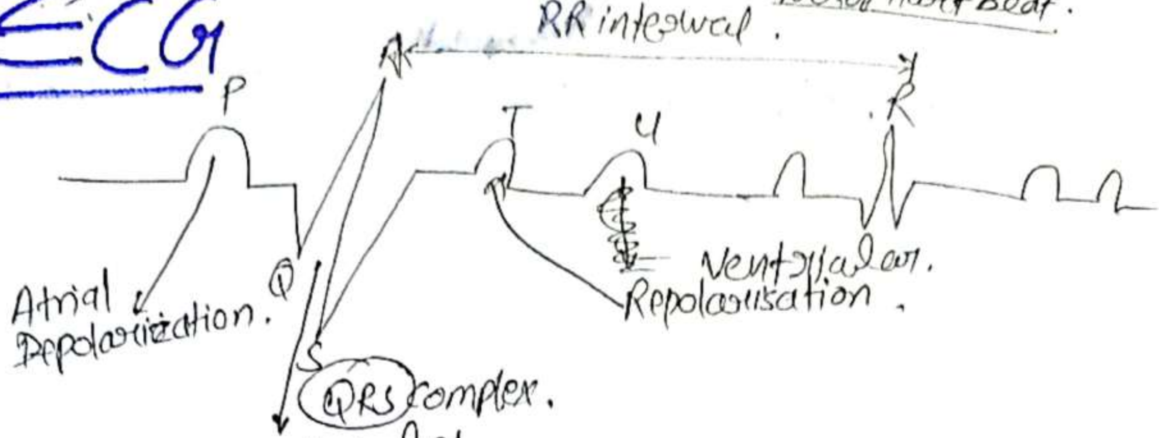
Ethiovian Triangle.

12 leads - Total

Augumented limb leads.

L = Lt arm aV_L	I	V_1
f = foot aV_F	II	|
R = Rt arm aV_R	III	V_6
(6) leads.	(6) leads	

ECG



ventricular.
Depolarisation.
forced contraction
due to larger area.

This electrical activity is prominent that is seen.

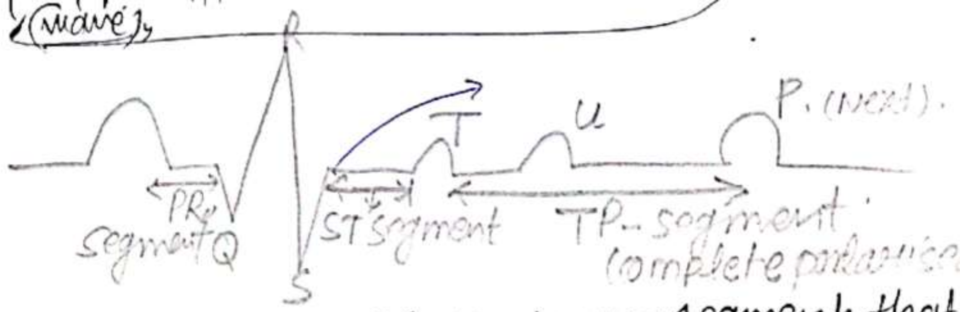
Papillary muscle → leads to the relaxation of heart.

$$HR = \frac{300}{\text{No. of BigBoxes b/w RR interval}}$$ (Big Box)

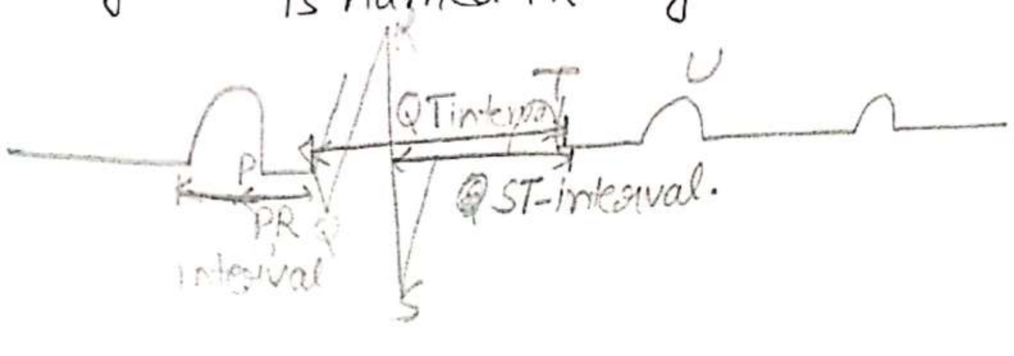
$$HR = \frac{1500}{\text{No of smallBOX b/w RR interval}}$$ (SmallBox)

1 — Box.
↓
(5)

PP = Atrial contraction Rate. (wave)



PR segment → B'cz R is the larger segment that's why it is named PR-segment.



27/03/2023

Dr. Hina (Cardiopulmonary).

Distribution of Blood

Sedative hone ke vajah se food, food pipe me rehjata hai, or Aspirate hone ke chances ↑↑sed many times.

Food digestion ↓ses, Aspiration ↑es.

Most of the Out of The Blood, where it is not much necessary such as, Hormone release, Skin supply, etc. & it is provided to the required muscles. Aerobic metabolism ↑↑eses, Amount of Blood constant (Hb), Plasma is constant.

→ food become the most prominant factor of Aspiration.

So, there should be No food in b/w (or) before exercises.

→ So we know drink contents are given in Ex's session, instead of solid food.

Distribution of Blood

(20 liters of Blood) exists in all vessels and muscles.

- If fully dialated the body could hold 20 lit. of Blood.
  (Blood vessels)

- The capacity of large portion of vascular to contrict or dialate provides rapid blood redribution to meet metabolic demands.
- It also optimises B.P. throughout the vascular circuit → (vessels, arteries, veins).

Physical factors affecting Blood flow.

The volume of flow in any vessel relates to:

(i) Directly to the pressure gradient b/w the two ends of the vessels, not to the abosulte pressure in the vessel.

(ii) Inversity to the resistance encoutered to the fluid flow.

Three factors determines the resistance –

1. Blood thickness / viscosity of Blood.
2. Length of the conducting Tube.
3. Blood vessels radius.

06/04/23 Thur.
Dr. Hima Vaish
(Cardiopulmonary)

Nitric Oxide → potent vasodilator.
Somewhere it acts as a healer to.

Nitric Oxide dilates blood vessel and decreases vascular resistance.
L Argenine - is the precursor to release NO_2.
- Most living organisms naturally produce NO_2 from its precursor L-Argenine.
It rapidly spreads through underlined cell memb. to smooth muscle cells c̄ in the arterial wall.
Here, it binds c̄ and activates Guanylayl cyclase enzyme. This initiates cascade of reaction (series of rxn) and them attenuate sympathetic vasocontriction and induce arterial smooth muscle relaxation to ↑es blood flow in neighbouring blood vessels.

Hormonal factors

- With sympathetic activation, adrenal gland secreates large quantities of epinephrine & small amount of Non-epinephrine & Blood.
- These hormonal chemical messengers induced a generalise constrictor respone except in blood vessels of heart & skeletal muscles.
- Hormonal respons plays a minor role in control of regional blood flow.

10/Apr/23 Mon
Dr. Hima mam
(Cardiopulmonary)

Functional Capacity of Cardiovascular System.

Cardiac output = Stroke volume x Heart Rate.
♂ ≃ 0.70mL

Cardiac output ♂ > Cardiac output ♀

Endurance capacity ↑ = Cardiac output ↑
(Why?) Atheletes > Normal/untrained person.
bz → SV↑ HR↓

Functional capacity of CVS.

Cardiac output is the amount of blood pumped by Heart during 1 min. period.

[CO = SV x HR]
↓
(Q)

Cardiac output in untrained individuals.

- for untrained individual with HR = 70 bpm. & CO = 5 L/m, SV = 0.714 mL. (♂)
- SV & CO ♀ = Average about 25% below the values of for Men.

Endurance Athelets / cardiac output

Stroke volume is greater in endurance atheletes. b'cz muscles required hot and long run of blood. for longer activity.
(LT) v. more amt of blood pumped out every ms contracting more there is more amt of blood in the muscles will flow.
CO = ↑ more than sedentary individuals.

Two factors explain large S.vol. ↑ endurance in trained atheletes —

① Increased vagal tone ↑
② decreased sympathetic Drive.
both of these ↓ (HR).
③ ↑ed Blood vol. ⊕ myocardial contractility and complince of left ventrical = All 3 of these ↑es SV.
↳ change of pressure (ΔV/ΔP)

Cardiac output during exercise

Systemic blood flow ↑ directly c̄ ex's intensity.
CO ↑ rapidly during transition from rest to steady state exercise. → (Rhyt. exactive c̄ same speed)

- Thereafter CO rises gradually untill it plateues when blood flow meets the exercise metabolic requirements.

In sedentary young males CO during maximal exercise ↑es 4-times above the resting level.
(20-22 lit.) per minute during ex.

In endurance atheletes (trained endurance athetes). max^m CO can be achived of about 30-40 Liters/minute.
The endurance atheletes achieve a large maximal CO due to large SV.

SV↑ (why?)
① Diastolic filling (relaxation ke time filling hoti hai)
② Systolic contraction. (vol.↑)
Stroke vol. Jab badta hai to

Increase in SV : Diastolic filling v/s Systolic emptying.

① There is enhanced cardiac filling in diastole followed by a more forceful systolic contractⁿ.

② Neurohormonal influence governs the 2nd mechanism that involves normal ventricular filling with a subsequent forceful ejection & emptying during systole.

③ The 3rd mechanism comes from training adaptation. that expands blood volume & reduce resistance to blood flow in peripheral tissues.

Enhanced Diastolic-filling

Any factor that ↑ses the venous return. or slows the HEART produces greater ventricular filling (Pre-load) during cardiac cycle diastolic phase

An ↑es in End-diastolic volume initiates a powerful ejection stroke during contraction.
- This ejects the normal SV + any additional blood that entered the ventricles in diastole and stretches the myocardium.
- The relationship b/w contractile force & resting length of the heart muscle fiber is explained by Frank Starling Law of the heart.

Frank starling Law of Heart:
"It states with in physiological limits, the force of contraction is α the initial length of the muscle fiber."

Greater Systolic Emptying

Progressive ↑es in SV during graded upright exercise ∅ in both children & Adults most likely results from combined effect of enhanced diastolic filling and more complete emptying after (during) systole.

Greater systolic ejection occurs despite ↑es resistance to blood flow in arterial circuit from exercise induced elevatn of systolic B.P. (After load).

25/Apr/23 (Tue)

Atheletes heart

→ Structural (or) changes due to training.

Strength & advance atheletes. both have changes.

cavity size ↑ } (ECO) can be visible
fibers size ↑ } SV ↑ CO ↑ O_2 deliviary ↑

Hypertrophy [ecentric hypertrophy.
[concentric.

Definition: Structural and functional changes that occure in heart of people, who trained for prolonged duration.

- Mainly there enlargment / hypertrophy of myo-cardium in response to repeated ex. stimuli.

features: cardiac enlagment to allow for ↑sed maximal (stroke volume. & cardiac (Q) output adaptations that derive ↑sed O_2 delivery in trained state.

Training causes cardiac remodelling. ~~there~~
There is ↑es in (Lt) ventrical, (Rt) ventricle & (Lt) Atrium. size and volume.
There is marked enlargement in (Lt) ventrical.
& mild ↑es in (Lt) ventrical wall ~~thy~~ thickness
Systolic & Diastolic functionation remains normal.
Changes are reversible c̄ sesation of training.
There may be ↑es in cavity size, but these values remain c̄ in except normal limits.

Adaptations differ by Training

① Eccentric (Lt) ventricle (LV) hypertrophy

This mainly occurs in endurance trained athletes -

eg. – long dist. runners.

features:

① Increase in Lt v cavity diamension proportional to $\uparrow$ in LV wall thickness to normalize myocardial strain.

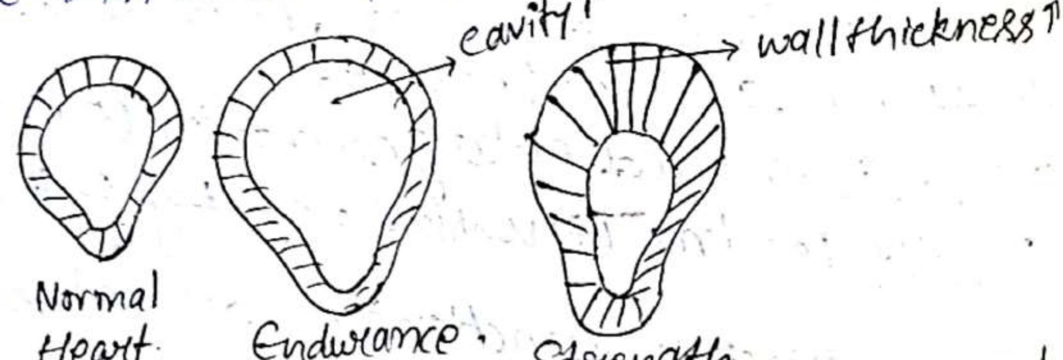
② Concentric LV Hypertrophy

This mainly ocurs in strength / Resistance trained athletes.

eg. - wrestlers.

features:

- $\uparrow$es in LV wall thickness. to Normalise $\uparrow$ed wall tension ~~c̄ $\uparrow$es to Normalize~~ c̄ $\uparrow$ in ~~tension~~ pressure.



Normal Heart.

Endurance Trained cavity $\uparrow$sed. eg.-

Strength Trained Athelete heart. wall thickness $\uparrow$sed. eg.-

Both endurance / strength trained Athelete's heart -
cavity $\uparrow$
wall $\uparrow$
eg.-