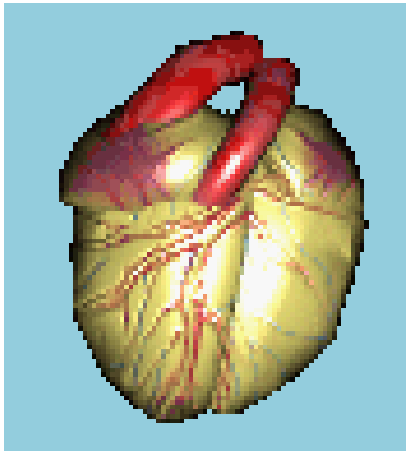
CARDIAC OUTPUT

Dr Balapala



- **Cardiac output (CO) – is defined as the volume of blood ejected by each ventricle per minute.**

- **Cardiac output = stroke volume X HR**

- **Normal value = 5- 6 litre/min**

- **Stroke volume (SV) – is defined as the volume of blood ejected by each ventricle per beat.**
- **Normal value = 70-80 ml.**

- **CO = SV x HR**

  = 70 X 72

  = 5.040 Lit/min

- **Cardiac index – is CO divided by BSA (Body surface area).**

- **CI = $\frac{\text{Cardiac output (5L/min)}}{\text{BSA(1.73m}^2\text{)}}$**

  ≈ 3L/min /m^2

- **Measures myocardial performance.**

- **EDV (End diastolic volume) – the vol. of blood remaining in each ventricle at the end of diastole. (= 130ml).**

- **ESV (End systolic volume) - the vol. of blood remaining in each ventricle at the end of systole. (= 50ml).**

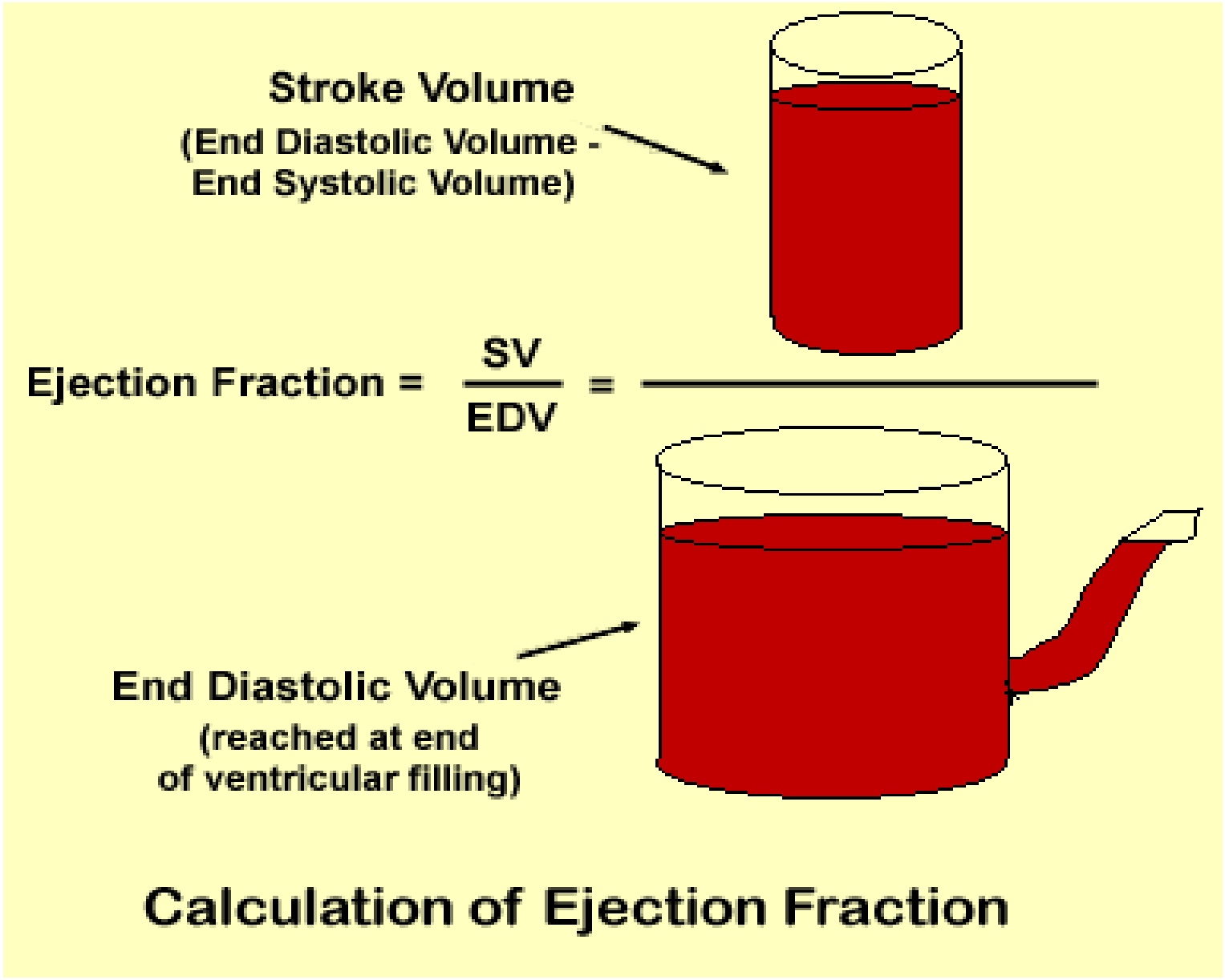
- **SV = EDV – ESV.**

- **Ejection Fraction – the percentage of EDV ejected per beat.**

- **EF = SV / EDV**

  ≈65 % (normal value 55 – 70%)

- **Is an index of myocardial performance.**


Stroke Volume
(End Diastolic Volume -
End Systolic Volume)
Ejection Fraction = SV / EDV =
End Diastolic Volume
(reached at end
of ventricular filling)
Calculation of Ejection Fraction

Physiological conditions that alter CO

- Increased CO

i. Exercise
ii. Anxiety
iii. Emotion & excitement
iv. Increased environmental temperature.
v. After eating
vi. pregnancy

Conditions that decrease CO

i. Standing from lying position
ii. Excessive sweating.

Conditions that do not change CO

i. Sleep
ii. Mild to moderate change in environmental temp.

MEASUREMENT OF CARDIAC OUTPUT

- Direct methods

i. Electromagnetic flow meter.

ii. Cardiometer.

iii. Doppler with echocardiography – in humans

- **Indirect methods**

1. Fick method
2. Indicator dilution method.
3. Thermodilution method.
4. Ballistocardiography.
5. Echocardiography.

Fick Method

- **Fick principle is defined as - the amount of substance taken up by an organ or by the whole body per unit time is equal to the arterio-venous difference of the substance times blood flow.**

- **CO can be measured by measuring the amount of oxygen consumed by the body in a given period and dividing this value by A-V oxygen difference across the lungs.**

- **Output of left ventricle (Bd flow) (CO)=**

$$\frac{\text{Oxygen consumption (ml/min)}}{\text{Arterio-venous oxygen difference}}$$

- Oxygen consumption at rest =250 ml O_2 / min
- Arterial O2 Content = 200 ml /lt
- Venous O2 Content =150 ml/lt
- A-V difference of O2 = 50 ml /lt
- CARDIAC OUT PUT =

$$\frac{250 \text{ ml/min}}{50 \text{ ml/lt}}$$

$$= 5 \text{ L/min}$$

Advantages

- Result is accurate.
- No chemicals injected.

Disadvantages

- Catheterization should be done.
- Hospitalization is required.
- Patient may be apprehensive of catheterization that increase CO.
- Simultaneous measurement of O2 consumption makes the process difficult.

Indicator dilution method

- Principle - in this method a known amount of an indicator (a dye or a radioactive isotope) is injected into circulation through an arm vein and the concentration of the indicator is measured in serial samples of the arterial blood.

- The output of the heart is equal the amount of the indicator injected divided by its average conc. in arterial blood after a single circulation through the heart

Procedure - this method is popularly k/a Hamilton's dye dilution method.

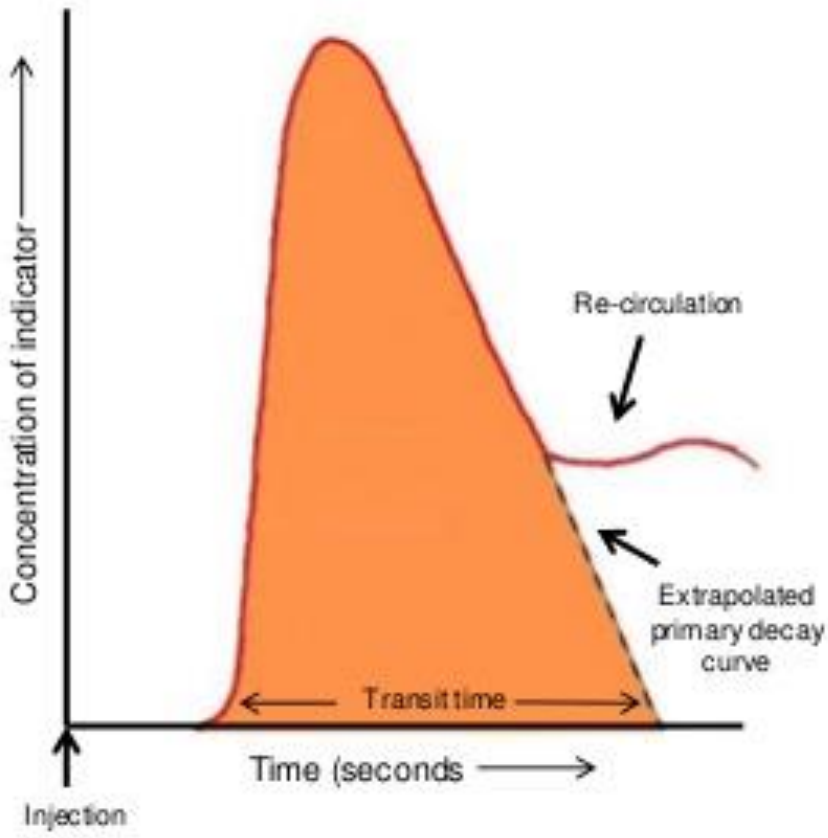
- Dye used – **Evans' Blue or Cardio-green**.
- After injecting the dye , the conc. of the dye is recorded as it passes through one of the peripheral arteries.(i.e by collecting serial arterial samples at regular intervals)

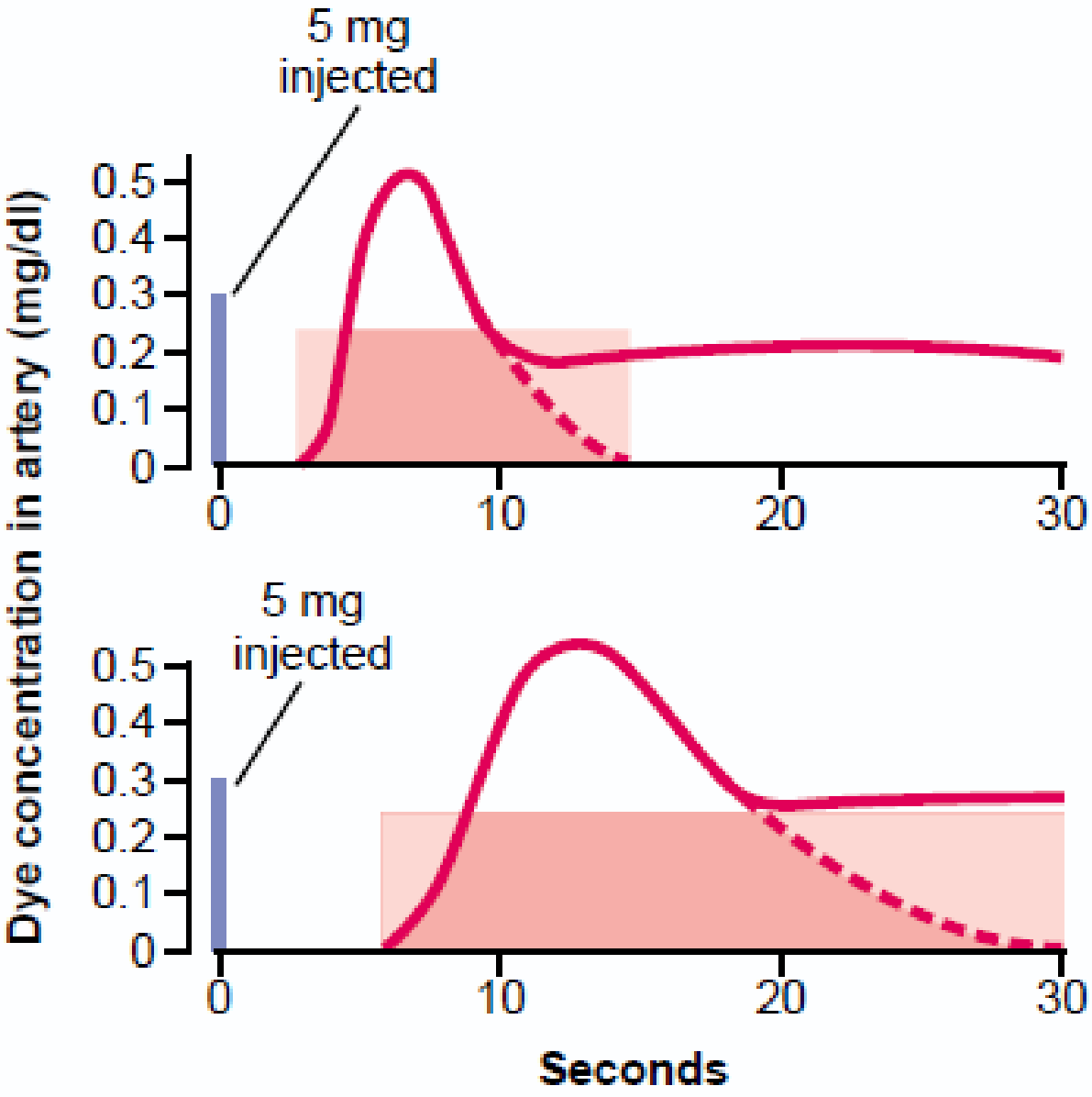
Cardiac output measurement: Indicator-dilution

- Described initially by Stewart, later by Hamilton
- When an indicator is injected into the blood stream, its concentration at a downstream sampling site initially increases and then falls proportional to blood flow
- As the indicator re-circulates, its concentration rises briefly and then reaches a steady state if it is not excreted
- The area under the extrapolated primary decay curve (*coloured area in the figure*) is equal to blood flow (cardiac output):

$$\mathbf{Q} = \frac{I}{\int_0^\infty C_t dt}$$

Q = cardiac output; I = amount of indicator; $C_t dt$: integral of indicator concentration over time




5 mg
injected
Dye concentration in artery (mg/dl)
0.5
0.4
0.3
0.2
0.1
0
0
10
20
30
5 mg
injected
0.5
0.4
0.3
0.2
0.1
0
0
10
20
30
Seconds

Indicator dilution method

$$F = \frac{Q}{C \times t}$$

where ,

F= blood flow (Lit /seconds)

Q = total amount of dye injected

C = mean concentration of dye

t= Duration in seconds of the first passage of dye

Advantages

- Is an accurate method.

Disadvantages

- Shoudn't be repeated in short time as the conc. of the dye of the earlier use may give errors

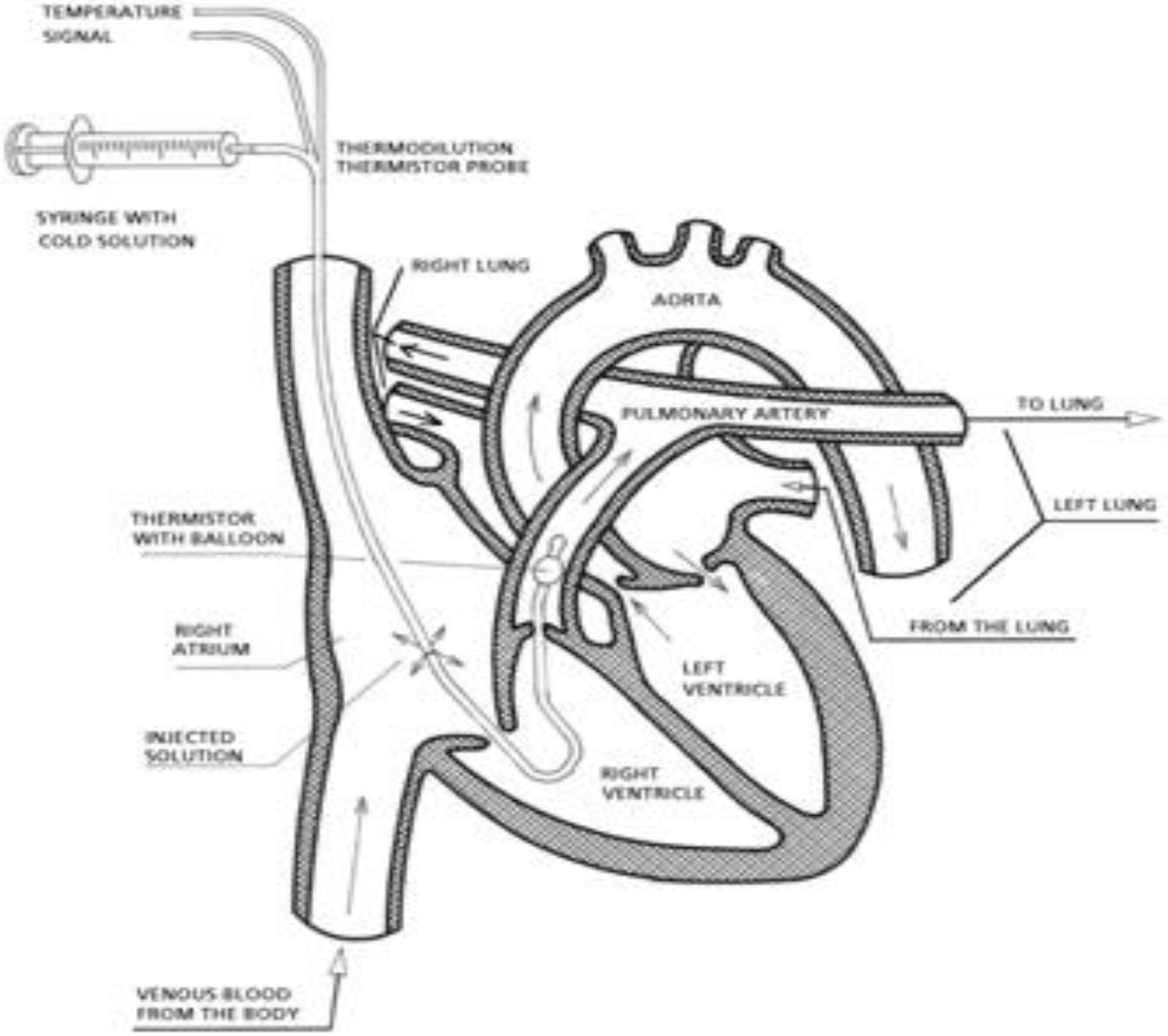
Thermodilution method

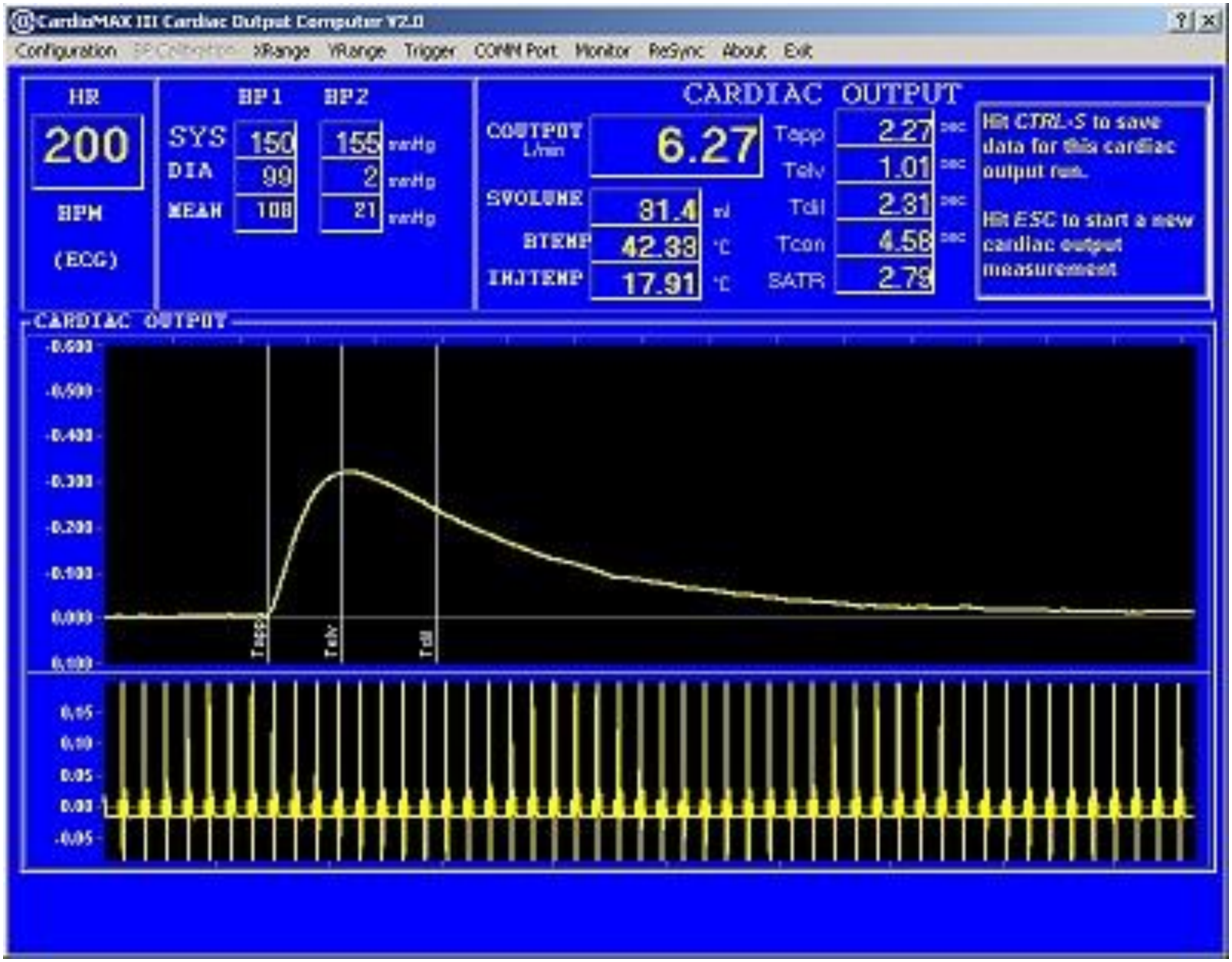
- Cold saline is used.
- Change in blood temperature is measured by thermistor.
- The CO is measured by determining the resultant change in the blood temperature in the Pulmonary artery

- Other indirect methods

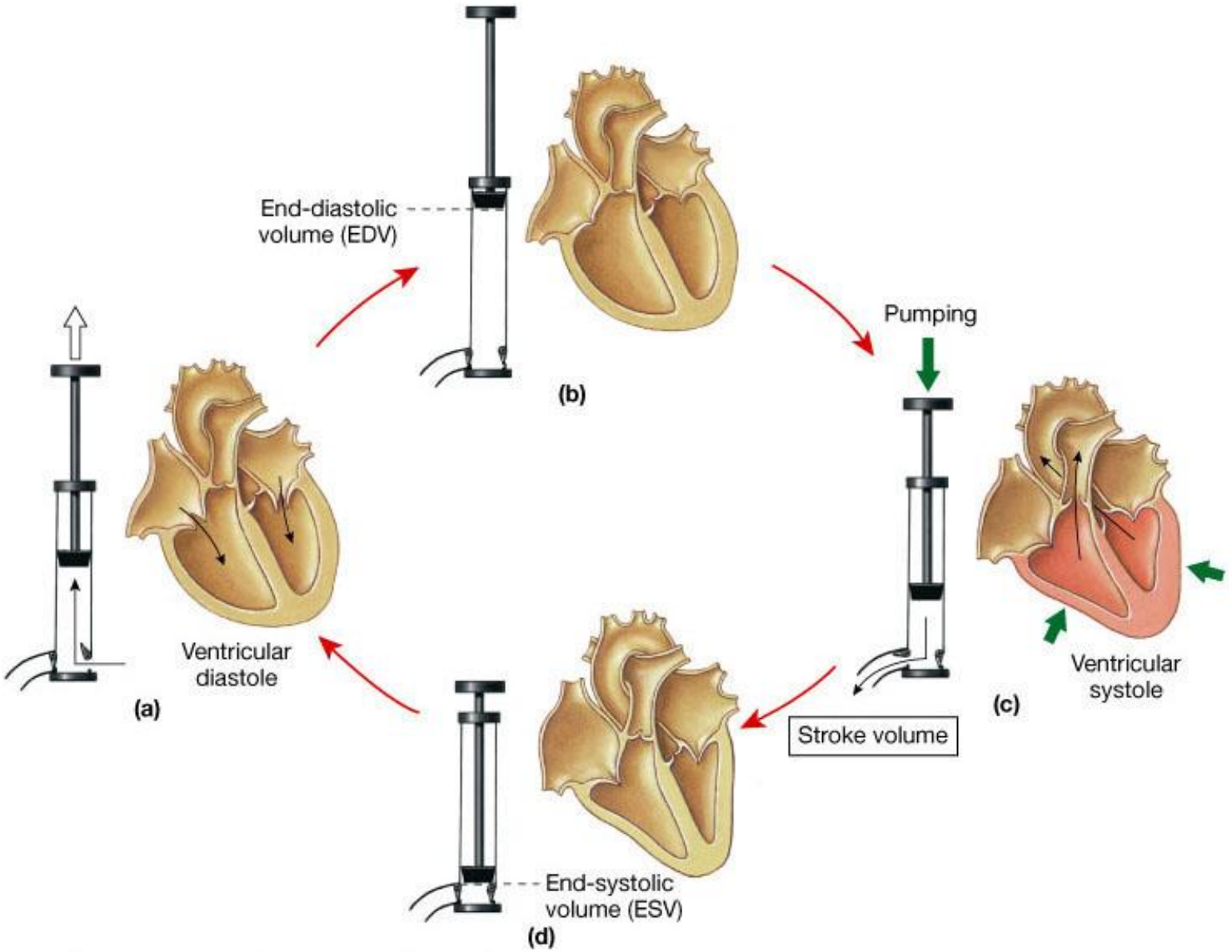
  Echocardiography

  Ballistocardiography etc.


TEMPERATURE
SIGNAL
THERMODILUTION
THERMISTOR PROBE
SYRINGE WITH
COLD SOLUTION
RIGHT LUNG
AORTA
PULMONARY ARTERY
TO LUNG
LEFT LUNG
FROM THE LUNG
THERMISTOR
WITH BALLOON
RIGHT
ATRIUM
LEFT
VENTRICLE
INJECTED
SOLUTION
RIGHT
VENTRICLE
VENOUS BLOOD
FROM THE BODY


CardioMAX III Cardiac Output Computer V2.0
Configuration
XRange
YRange
Trigger
COMM Port
Monitor
ReSync
About
Exit
HR
200
BPM
(ECG)
BP1
BP2
SYS
DIA
MEAN
150
99
108
155
2
21
mmHg
CARDIAC OUTPUT
COUTPUT
L/min
6.27
SVOLUME
81.4
ml
BTEMP
42.33
°C
INJTEMP
17.91
°C
Tapp
Telv
Tdil
Tcon
SATR
2.27
1.01
2.31
4.58
2.79
sec
Hit CTRL-S to save data for this cardiac output run.
Hit ESC to start a new cardiac output measurement
CARDIAC OUTPUT
0.600
0.500
0.400
0.300
0.200
0.100
0.000
-0.100
Tapp
Telv
Tdil
0.15
0.10
0.05
0.00
-0.05

A Simple Model of Stroke Volume



- https://www.youtube.com/watch?v=vFRkSB46bl8

Factors **Affecting & Regulating** Cardiac Output

Factors Affecting CO

- CO = SV X HR
- So factors affecting SV and HR will affect CO.

A. Factors affecting stroke volume

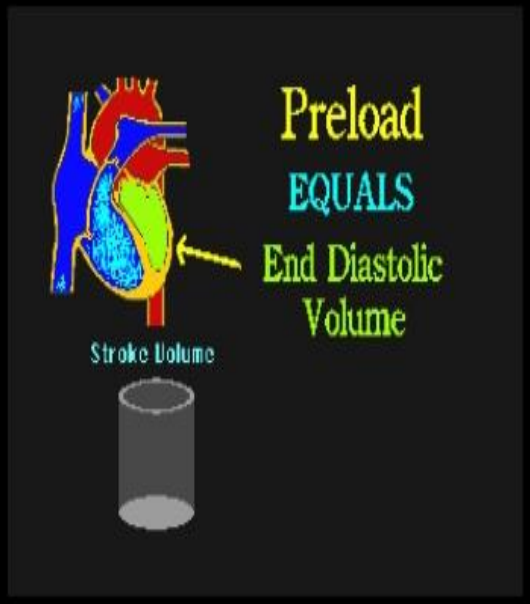
1. Preload.
2. Afterload.
3. Myocardial contractility.

1. Preload (EDV)

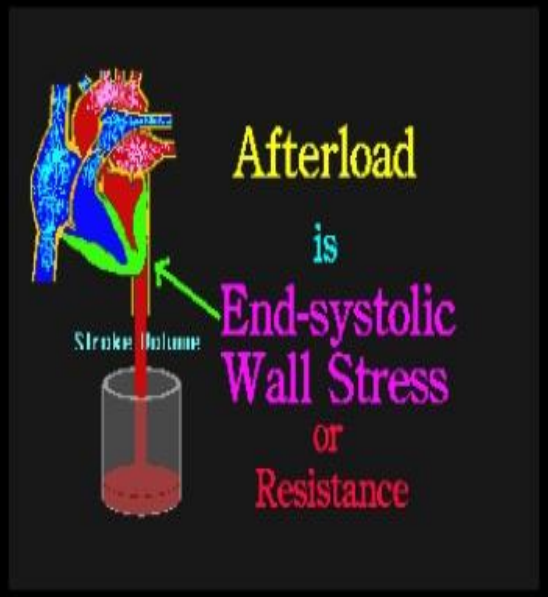
- EDV is considered as preload.
- ↑ in EDV ↑es stroke volume and viceversa.
- This can be explained by **Frank-Starling's Law-**

'it states that within physiological limit, the force of contraction is directly proportional to initial length of muscle fiber'

load



afterload



- The initial length of muscle fiber means the fiber length prior to ventricular contraction(i.e just before the onset of systole).
- Initial length prior to contraction depends on the extend of ventricular stretch which in turn depend on degree of end diastolic filling of the ventricle.

- ↑ed ventricular filling ↑es the fiber length that inturn increases the force of contraction.

- So the ↑in SV due to ↑in initial length of muscle fiber is called as Heterometric Autoregulation of cardiac output.(i.e SV varies at various ventricular muscle length)

Frank-Starling Law of Heart

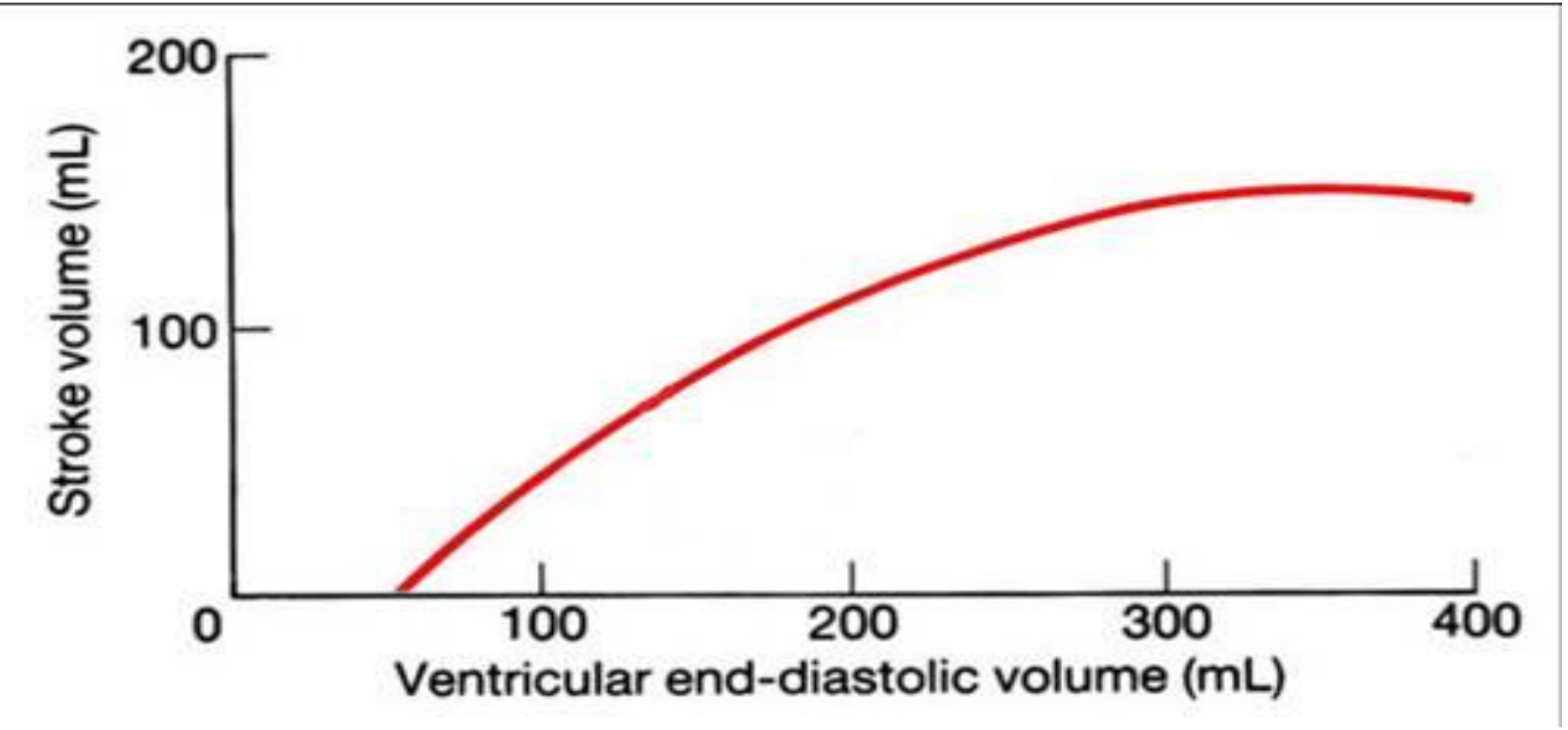
- **Mechanism-**

1. **With ↑ed EDV, the initial length of the muscle fiber ↑es due to increase in chamber size that stretches the muscle fibers.**
2. ↑ed stretch → ↑ed interaction b/w thick and thin filament → ↑ed force of contraction

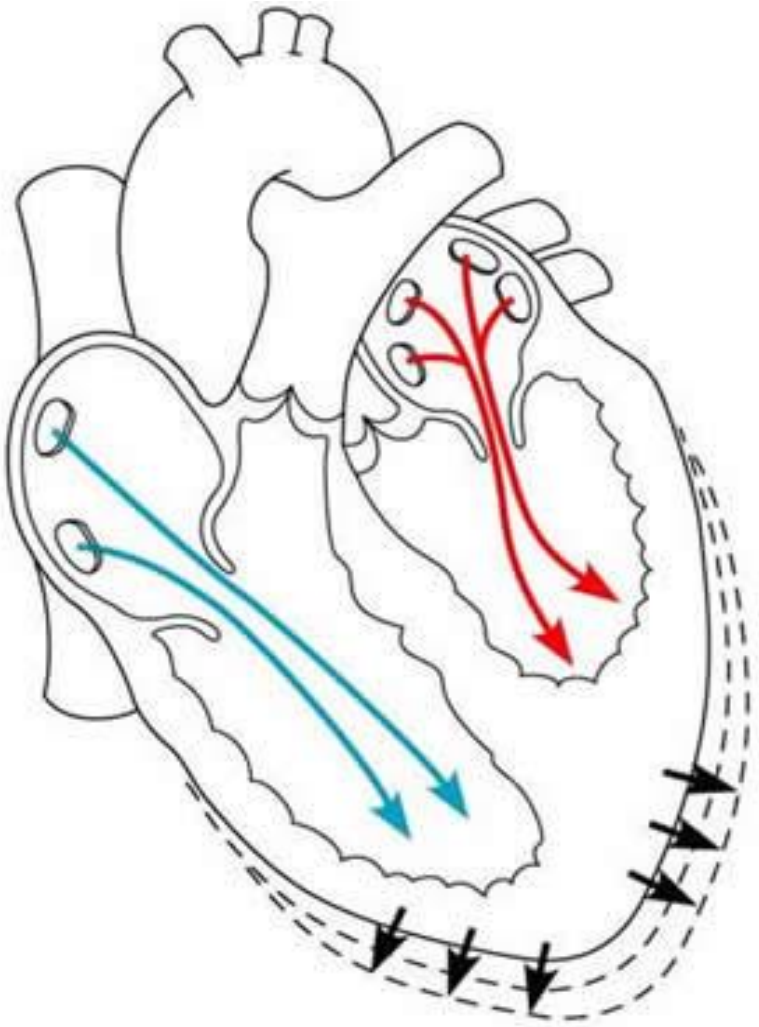
3. Stretch→ opens up the stretch sensitive Ca+2 channels on muscle cell membrane → Ca+2 influx into myocardial cells increases → ↑ed force of contraction.

4. Stretch→ increases intracellular Ca+2 ion conc. due to increased Ca+2 influx from ECF which in turn causes Ca+2 induced Ca+2 release from sarcoplasmic reticulum.

Is the curve rising constantly ??

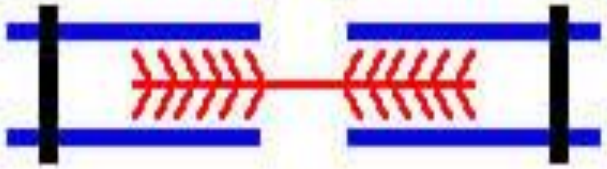


The Frank-Starling Law of the Heart refers to the length-tension relationship of cardiac muscle where length (stretch) is determined by EDV (end diastolic volume) and tension = contractile force at a given length will determine SV (stroke volume). As the ventricles become overfilled (to the right on the curve beyond EDV = ~250 mL), the heart becomes inefficient and stroke volume levels off and eventually declines.

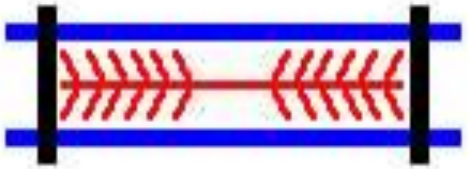


(a) Preload direction

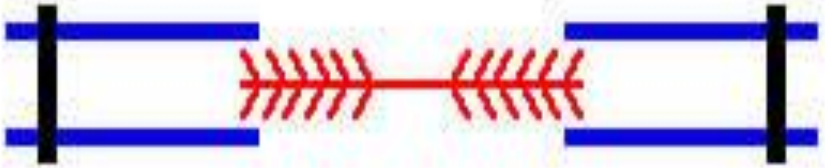
Normal Sarcomere Resting Length



Contracted Sarcomere Length



Pre-loaded Sarcomere Length



Preload cont…

- The EDV(end diastolic filling pressure) depends on 3 major factors

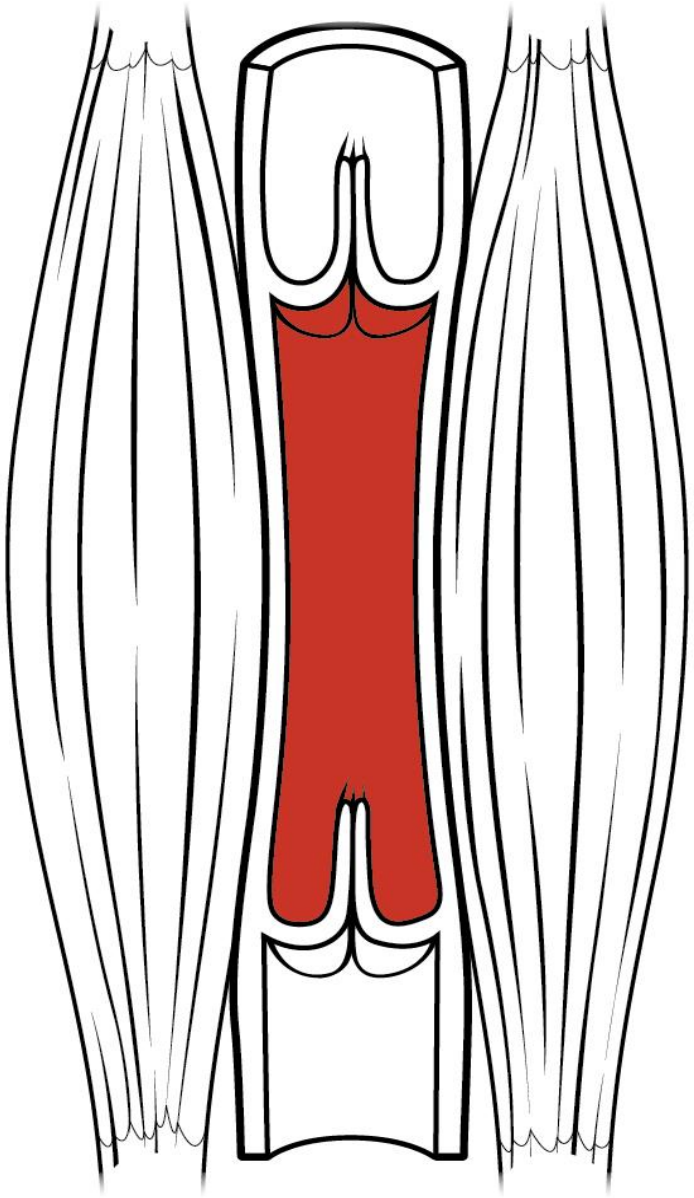
I. Venous return
II. Atrial pump activity
III. Ventricular compliance.

I. Venous return(VR)

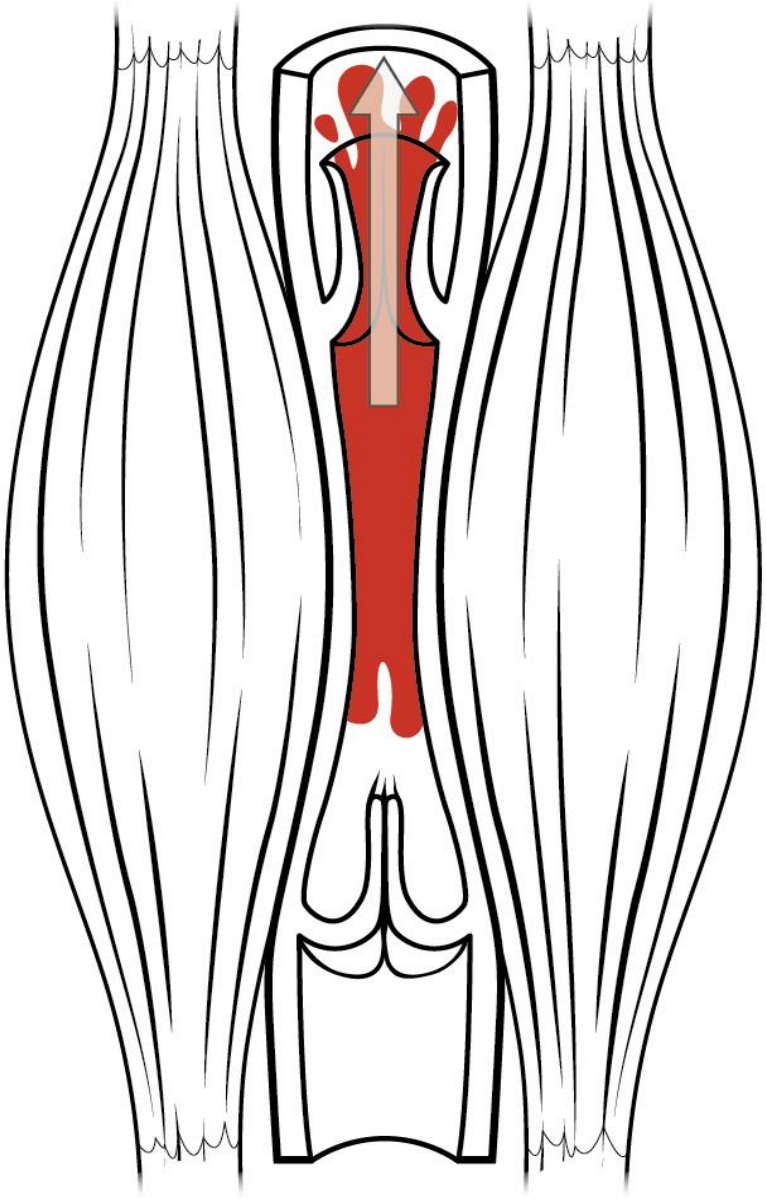
- VR- is the amount of blood that returns to the RA from systemic venous circulation.
- VR depends on the following factors

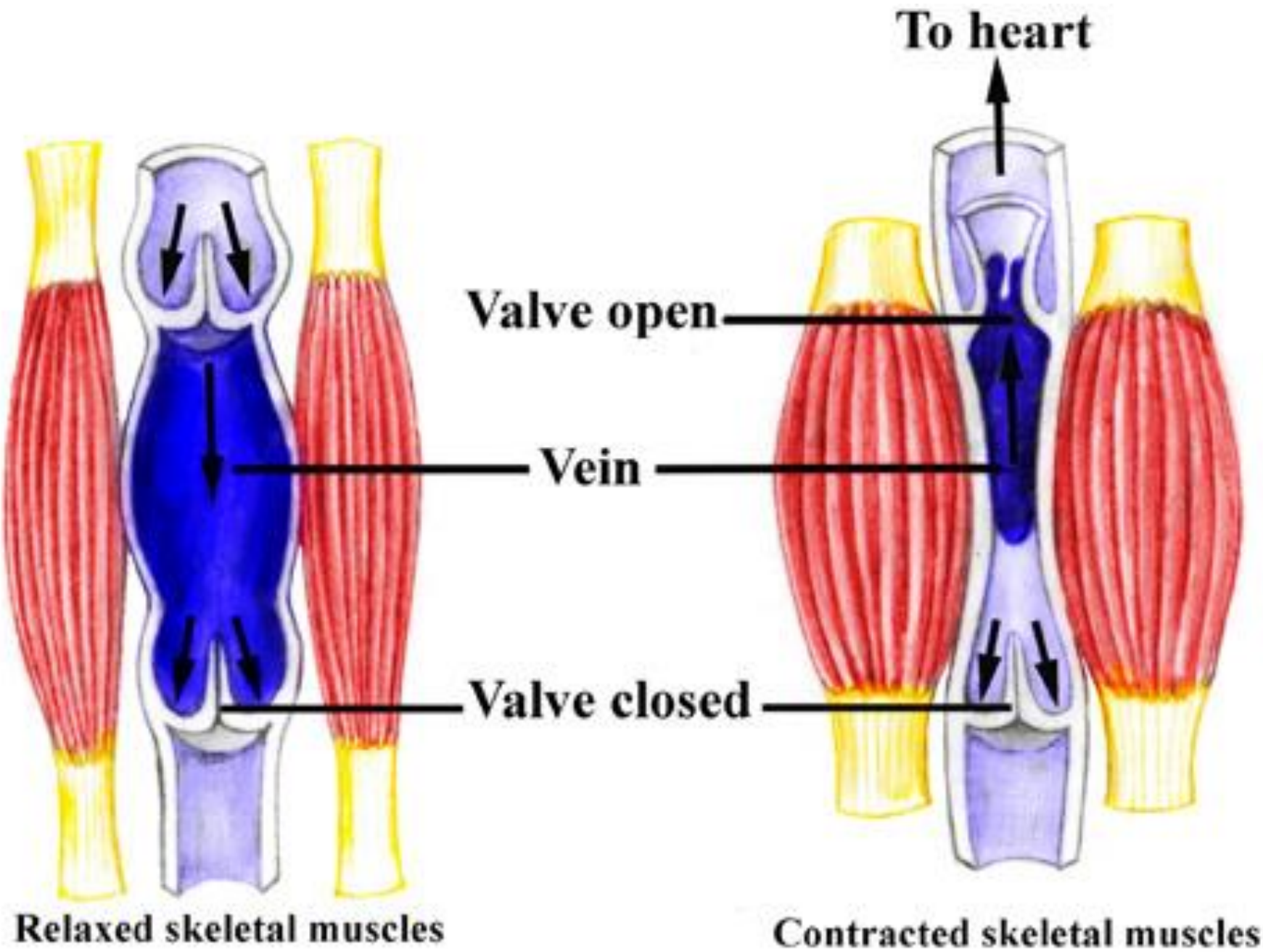
i. **Muscle pump -**
ii. **Cardiac pump –**
iii. **Thoracic pump –**
iv. **Abdominal pump –**
v. **ECF volume –**
vi. **Sympathetic activity (veno constriction**)

Muscles relaxed,
valves closed



Muscles contracted,
valve above muscle opens




To heart
Valve open
Vein
Valve closed
Relaxed skeletal muscles
Contracted skeletal muscles

Cardiac pump

- The cardiac pump influences the VR by 2 types of forces- '**vis-a tergo**' and '**vis-a-fronte**'.
- **vis-a tergo-** refers to forward push from behind i.e the propelling force which pushes the blood from veins into Rt.atrium.
- vis-a tergo- results from myocardial contraction during systole + Elastic recoil of the arterial wall (Windkessel Effect)

- **vis-a-fronte-** refers to suction force acting from the front , which pulls the blood from great veins towards Rt. Atrium.
- The suction force is created by ventricular contraction and has 2 components

i. Ventricular systolic suction

ii. Ventricular diastolic suction

II. Atrial pump activity.

- Atrial contraction contributes to only 20-25 % of ventricular filling **at rest.**
- Atrial systole becomes important in situations which demand ↑ed CO **e.g Exercise.** In this case atrial systole contributes significantly to the end-diastolic filling of the ventricle.

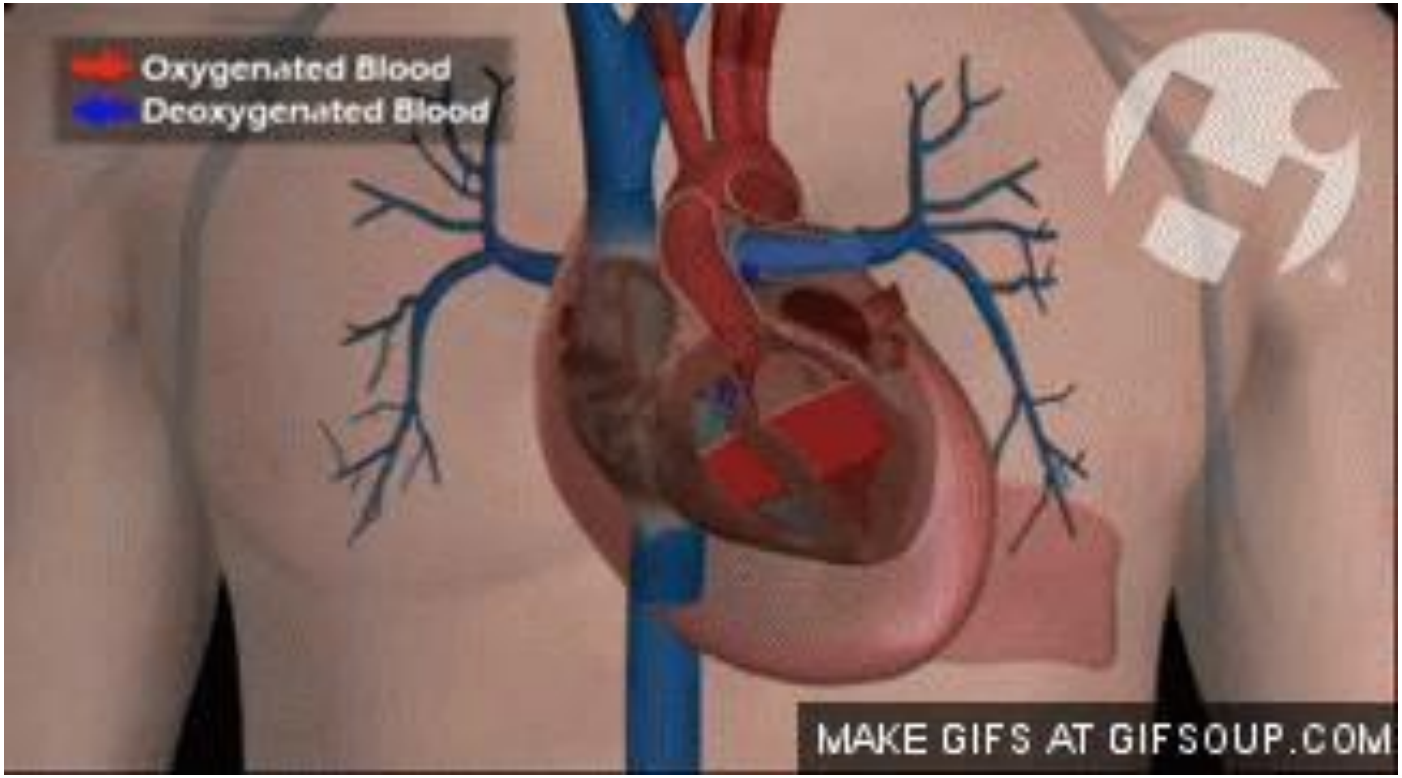
III.Ventricular compliance.

- Normally ventricular muscles are compliant (stretchable), adequate to accommodate enough blood during diastole.(EDV =130 ml).

- Pathological conditions where ventricular compliance decrease –

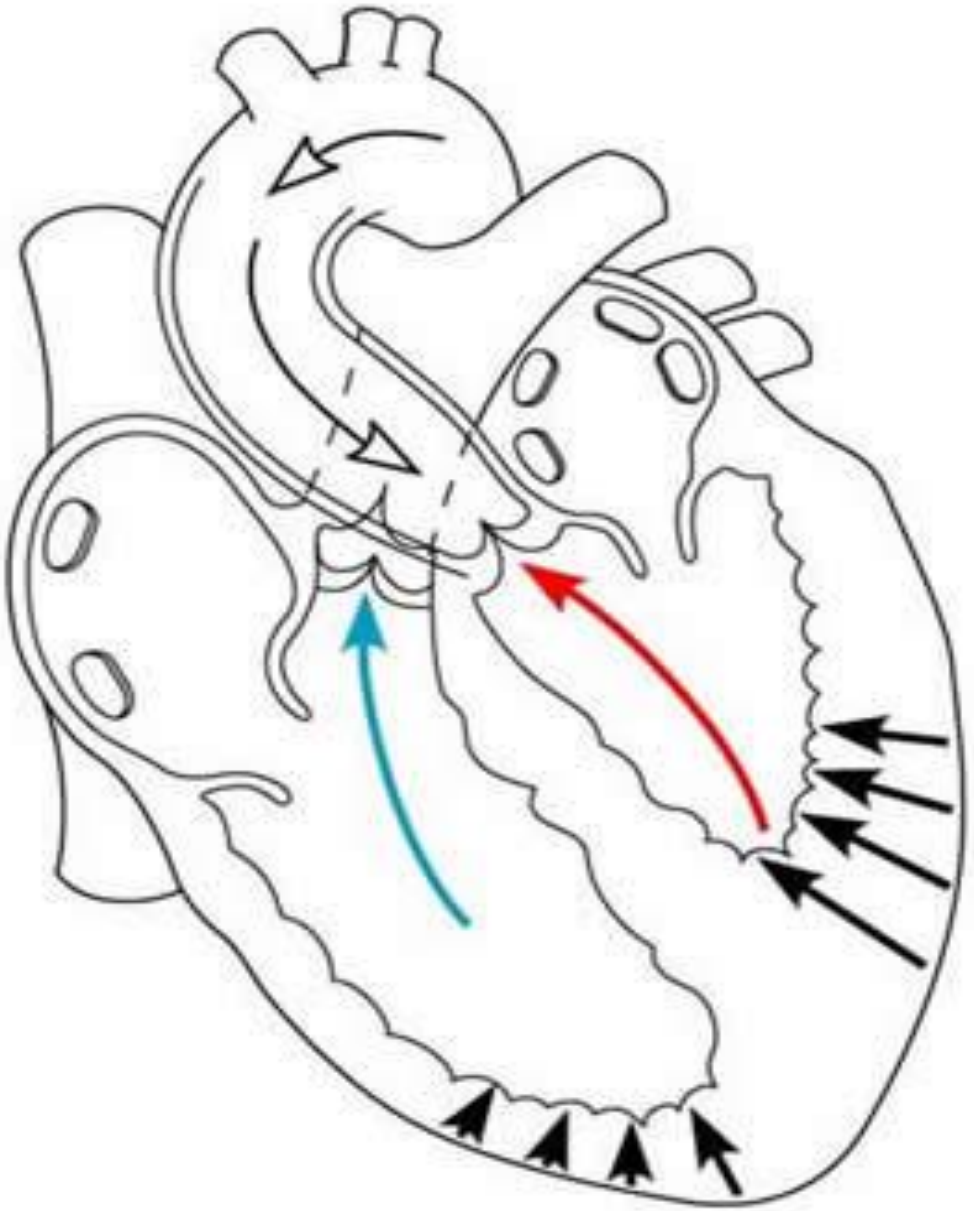
  - pericardial effusion

  - cardiac temponade = reduced ventricular filling and subsequent hemodynamic compromise due to effusion


Oxygenated Blood
Deoxygenated Blood
MAKE GIFS AT GIFSOUP.COM

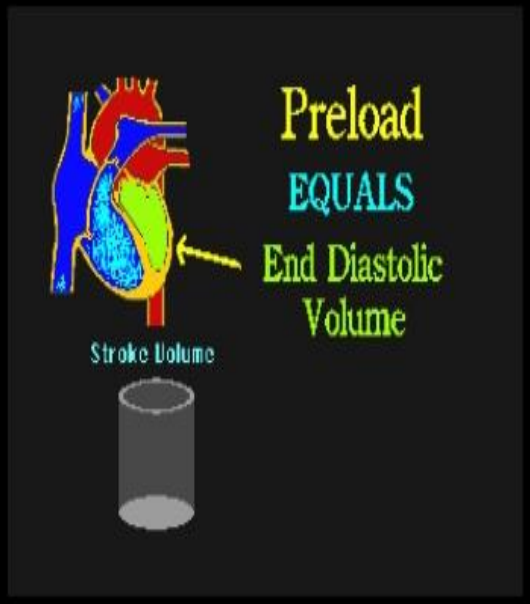
2. Afterload

- **Afterload is the force against which ventricular muscle fibers contract**.
- It is the resistance offered against the ejection of blood from ventricles.
- **Peripheral resistance is considered as Afterload**.
- Cardiac output is **inversely** proportional to afterload.

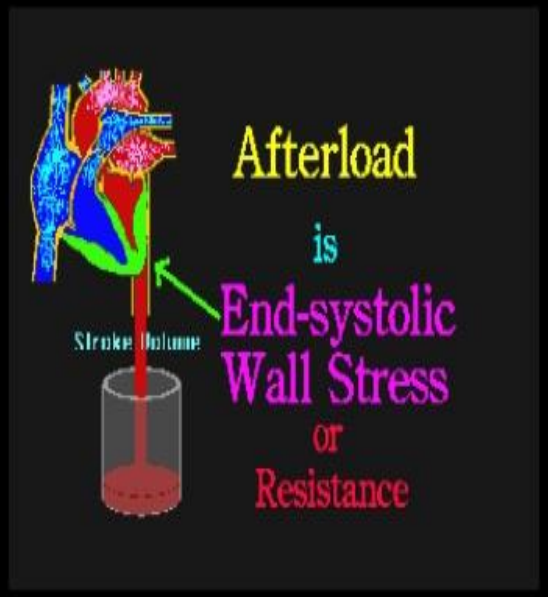


(b) Afterload direction

load



afterload



- ↑in peripheral resistance ↓es cardiac output and viceversa.
- cardiac output (or SV) changes without change in ventricular muscle length. This type of regulation of cardiac output is called as **Homometric Autoregulation .**
- This is also known as **Arnep effect** (as described by Arnep in 1974)

- Afterload (peripheral resistance) depends on 2 factors-

i. <u>Vessel diameter-</u>

- vasoconstriction ↑es peripheral resistance that ↓es SV.
- Vasodilatation ↓es peripheral resistance or afterload that ↑es SV

ii. <u>Viscosity of blood.</u>

3.Myocardial contractility

- Has major influence on CO.
- Factors that ↑myocardial contractility are k/a **positively inotropic** .
- Myocardial contractiliy depends on the following factors-

i. Ventricular muscle mass-

- decreased in MI, Cardiomyopathies (so CO is decreases)

- increased in regular physical exercise (CO increases);physiological hypertrophy of myocardium.

ii. **Autonomic activity** –

- ventricles are supplied by sympathetic fibers.
- Sympathetic stimulation ↑es myocardial contractility & sympathetic inhibition ↓es contractility.
- Ventricles are sparsely innervated by parasympathetic fibers(vagus)

iii. hormones

- + vely inotropic
- Catecholamines
- Insulin
- Glucagon
- thyroxine

- negatively inotropic
- Acetylcholine

iv. Chemical factors

Xanthines

- Caffeine , theophylline are +vely inotropic

Inhibiting factors

- Hypoxia
- Hypercapnia
- Acidosis
- Toxins
- General anaesthetics

V. **Drugs** –

- Digitalis -↑es myocardial contractility by inhibiting Na-K ATPase activity on myocardial cell membrane..
- Other drugs that inhibit contractility –

  quinidine,barbiturates etc.

B. Factors affecting Heart Rate(HR)

HR is normally influenced by autonomic activity.

- **Sympathetic** stimulation **↑es** and **parasympathetic (Vagal**) stimululation **↓e**s HR.
- HR depends on the balance b/w symp.& para symp. activity.
- Normally ↑ in HR should result in ↑in CO (as CO =SV X HR)

- Unless associated with ↑ed venous return (EDV) ,↑ in HR may not ↑ CO propertionately.

- In severe Tachyardia- HR ↑ but EDV is↓ed as duration of diastole shortens more than the duration of systole.(incomplete relaxation of ventricles) . So CO doesn't ↑ propertionately.

Regulation of CO

1. **Intrinsic regulation.**

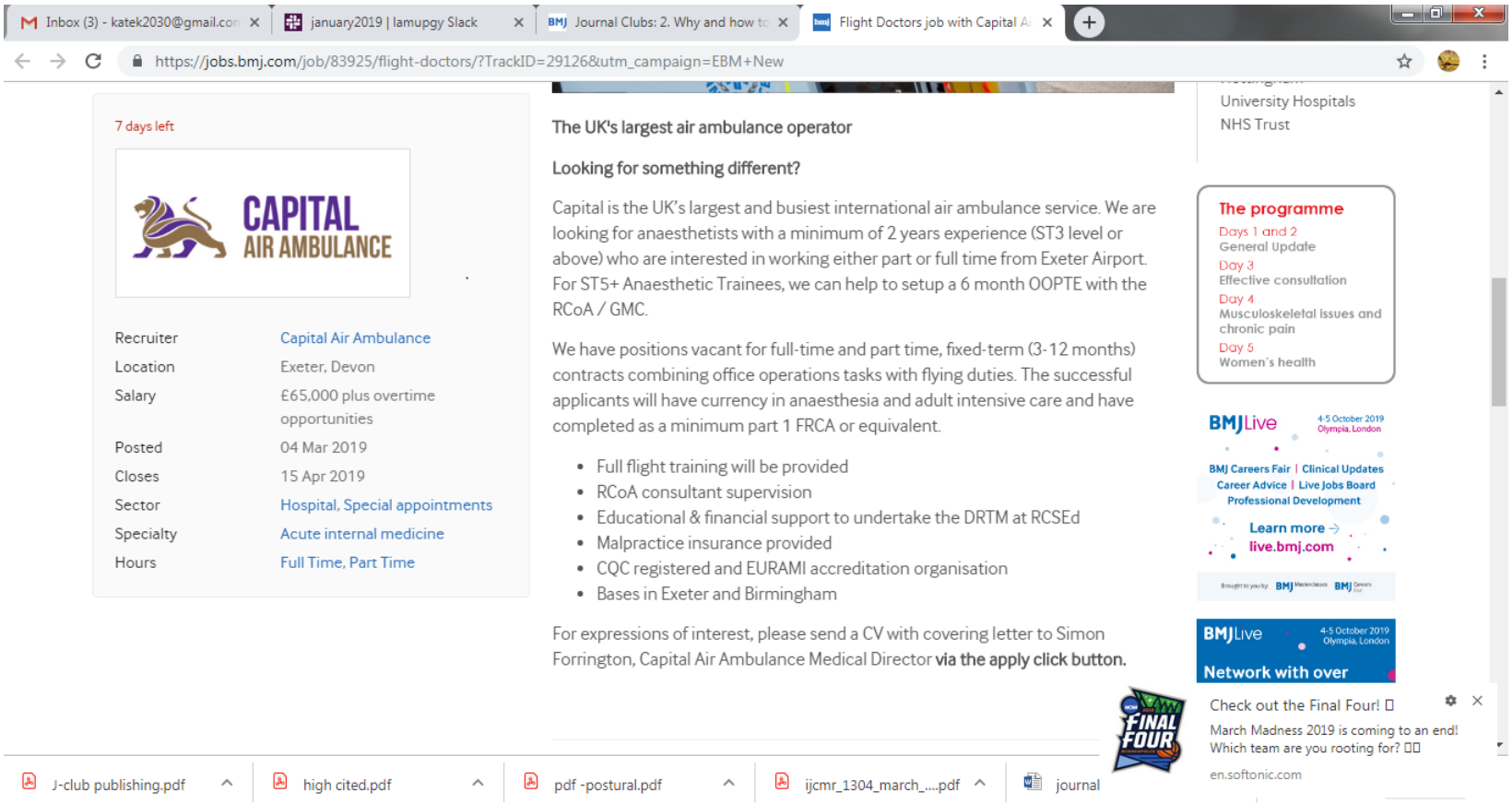
   Frank-Srarling Law

2. **Extrinsic regulation - After load**

   -neural control

   - hormonal regulation

   - chemical regulation


Inbox (3) - katek2030@gmail.com
january2019 | lamupgy Slack
Journal Clubs: 2. Why and how to
Flight Doctors job with Capital Ai
https://jobs.bmj.com/job/83925/flight-doctors/?TrackID=29126&utm_campaign=EBM+New
7 days left
CAPITAL AIR AMBULANCE
Recruiter Capital Air Ambulance
Location Exeter, Devon
Salary £65,000 plus overtime opportunities
Posted 04 Mar 2019
Closes 15 Apr 2019
Sector Hospital, Special appointments
Specialty Acute internal medicine
Hours Full Time, Part Time
The UK's largest air ambulance operator
Looking for something different?
Capital is the UK's largest and busiest international air ambulance service. We are looking for anaesthetists with a minimum of 2 years experience (ST3 level or above) who are interested in working either part or full time from Exeter Airport. For ST5+ Anaesthetic Trainees, we can help to setup a 6 month OOPTE with the RCoA / GMC.
We have positions vacant for full-time and part time, fixed-term (3-12 months) contracts combining office operations tasks with flying duties. The successful applicants will have currency in anaesthesia and adult intensive care and have completed as a minimum part 1 FRCA or equivalent.
Full flight training will be provided
RCoA consultant supervision
Educational & financial support to undertake the DRTM at RCSEd
Malpractice insurance provided
CQC registered and EURAMI accreditation organisation
Bases in Exeter and Birmingham
For expressions of interest, please send a CV with covering letter to Simon Forrington, Capital Air Ambulance Medical Director via the apply click button.
University Hospitals NHS Trust
The programme
Days 1 and 2 General Update
Day 3 Effective consultation
Day 4 Musculoskeletal issues and chronic pain
Day 5 Women's health
BMJLive 4-5 October 2019 Olympia, London
BMJ Careers Fair | Clinical Updates
Career Advice | Live Jobs Board
Professional Development
Learn more
live.bmj.com
BMJLive 4-5 October 2019 Olympia, London
Network with over
Check out the Final Four!
March Madness 2019 is coming to an end! Which team are you rooting for?
en.softonic.com
J-club publishing.pdf
high cited.pdf
pdf -postural.pdf
ijcmr_1304_march_....pdf
journal

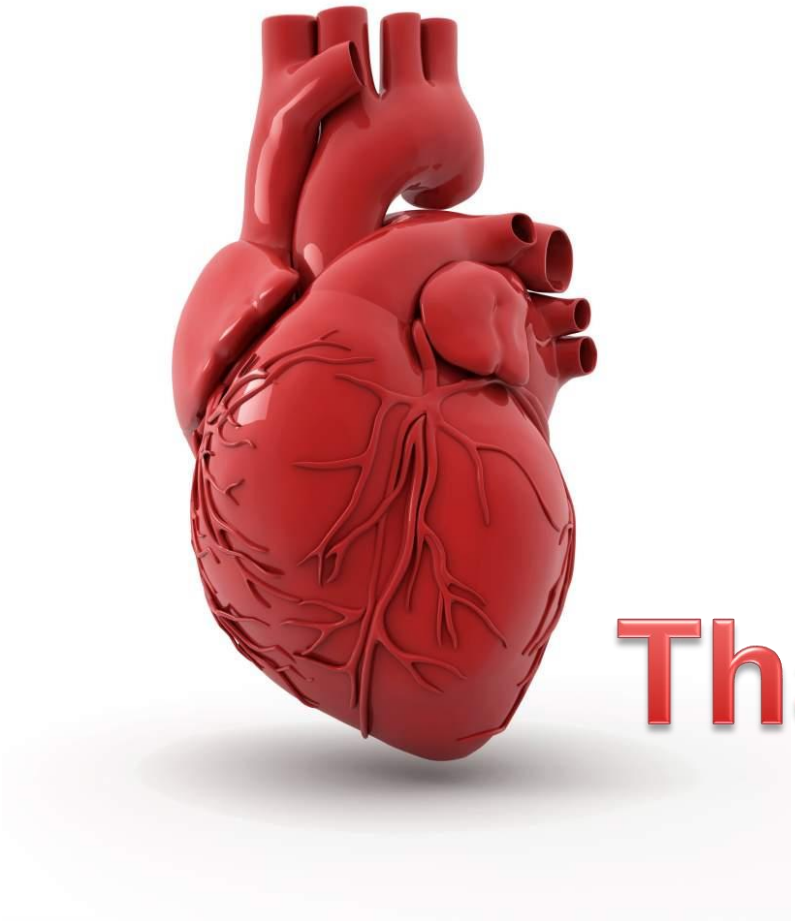
Learning outcomes

- Describe normal COP
- Discuss factors that affect SV
- Describe Frank starling law
- What happens to COP in exercise.

- https://www.youtube.com/watch?v=l4jxZGlnf0Q

Links

- https://www.youtube.com/watch?v=vFRkSB46bl8
- https://www.youtube.com/watch?v=LqOd4Sqc9Ts



Thanks