Cardiac Output and its Measurements

by

Dr Gul Muhammad

Outlines

- General consideration of cardiac output and its measurement
- Importance of cardiac output measurement
- Methods of measuring cardiac output
- Types of cardiac output measurement
- What is cardiac reserve?
  Mechanisms of cardiac reserve:
- What is Cardiac index

Cardiac output

- Is the volume of blood pumped into the aorta each minute by the left vetricle .
- It is the determinant of global oxygen transport to the body
- It reflects the efficiency of cardiovascular system
- There is no absolute value for cardiac output measurement

Cardiac Output Measurement

- **AIM : hemodynamic monitoring and support in the critically ill so as to optimize oxygen delivery to the tissues**

- **Oxygen delivery is determined by Cardiac Output and amount of oxygen carried in the blood**

- **Allows us to assess the blood flow to the tissues, and provides information on how to best support a failing circulation**

Why should we measure ?

1. The recognition that in many critically ill patients, low cardiac output leads to significant morbidity and mortality

2. The clinical assessment of cardiac output is unreliable/ inaccurate

When should we monitor?

- **High risk critically ill surgical patients in whom large fluid shifts are expected along with bleeding and hemodynamic instability**

- **An important component of goal directed therapy (GDT), *i.e.*, when a monitor is used in conjunction with administration of fluids and vasopressors to achieve set therapeutic endpoints thereby improving patient care and outcome**

Features of an ideal Cardiac Output monitor

- Safe and accurate
- Quick and easy to use both in terms of set-up and interpretation of information
- Operator independent i.e. the skill of the operator

  doesn't affect the information collected

- Provide continuous measurement

Methods of measuring cardiac output

Invasive

1. **Fick principle**

2. **Dye dilution**

Non-Invasive

1. TEE / Esophageal Doppler

2. Trans Thoracic Echocardiography (TTE)

Methods of measuring cardiac output

Simple method:

- **CO(L/min) = HR (beats /min) x SV (L/beat.)**
- **SV : volume ejected during each beat , depends on venous return, can be equal to venous return.**
- **CO (at rest) = 5-6 L/min.**
- **HR =72 beats/min , SV=0.07L/min (70ml) .**
- **CO increases when metabolic need (eg. Exercise), Therefore, H R or S V or both can increase.**

Methods of measuring cardiac output

Fick Principle: "Gold standard"

$$CO\ (ml/min) = VO_2\ (ml/min) / a\text{-}v\ O_2\ diff\ (ml/l)$$

- Fick Principle relies on the **total uptake of a substances by peripheral tissue** is equal to the product of blood flow to the peripheral tissue and arterial – venous concentration difference of the substances

$$CO\ (ml/min) = VO_2\ (ml/min) / a\text{-}v\ O_2\ diff\ (ml/l)$$

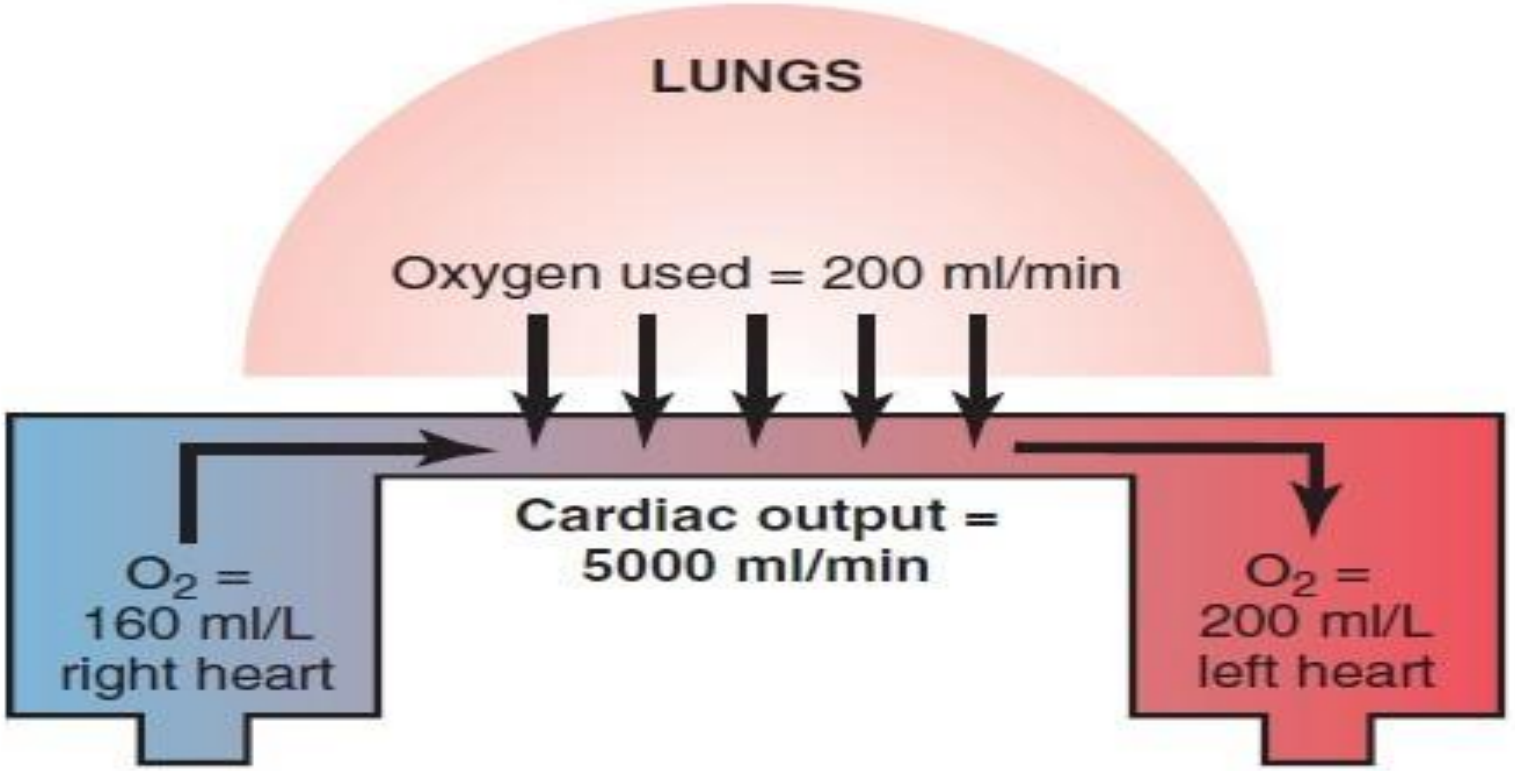


Figure 20-19. Fick principle for determining cardiac output.

calculation

- 200 ml Oxygen consumed in one minute
- Oxygen concentration found in arterial blood= 40ml/L
- Total 200 ml oxygen distributed in how much blood at the concentration of 40ml/L
- 200/40=5 so 5x1000=5000 ml OR 5 litre total amount of blood
- i.e Cardiac output is 5 litre /min

$$CO\ (ml/min) = VO_2\ (ml/min) / a\text{-}v\ O_2\ diff\ (ml/l)$$

Mixed venous blood is usually obtained

- Through a catheter inserted into:
  - The brachial vein of the forearm,
  - Through the subclavian vein,
  - Down to the right atrium,
  - The right ventricle or pulmonary artery
- SYStemic arterial blood can then be obtained from

- Fick cardiac outputs are infrequently used because difficulties in collecting and analyzing exhaled gas concentration in critically ill patients because may not have normal VO_2 value.

Indicator dilution method

- Based on how fast the flowing blood can dilute the substances introduced into the circulation

- A known amount of a substance, such as a dye, is injected into a large systemic vein or, preferably, into the right atrium.
- The concentration of the dye is recorded as the dye passes through one of the peripheral arteries, giving a curve

Stewart-Hamilton Equation

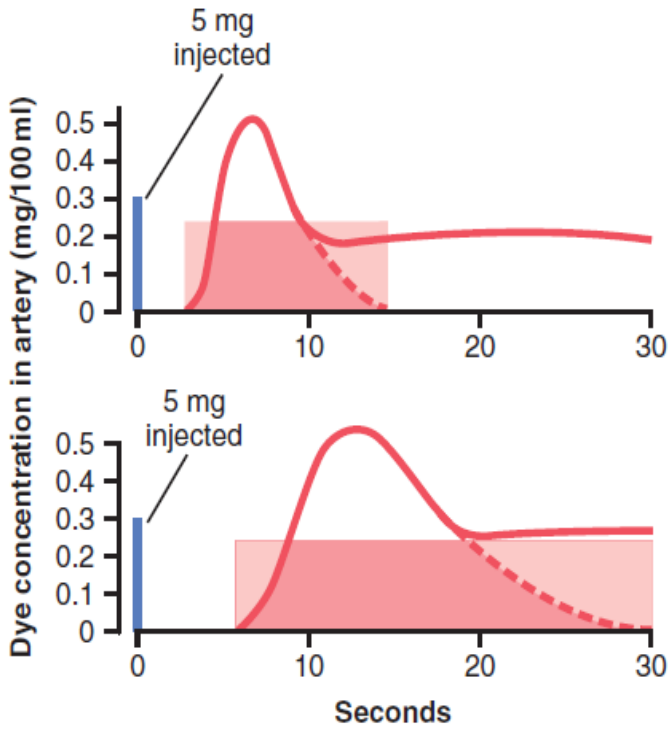


Figure . . Extrapolated dye concentration curves used to calculate two separate cardiac outputs by the dilution method. (The rectangular areas are the calculated average concentrations of dye in the arterial blood for the durations of the respective extrapolated curves.)

$$\text{Cardiac output (ml/min)} = \frac{\text{Milligrams of dye injected} \times 60}{\left(\begin{array}{l}\text{Average concentration of dye}\\ \text{in each milliliter of blood}\\ \text{for the duration of the curve}\end{array}\right) \times \left(\begin{array}{l}\text{Duration of}\\ \text{the curve}\\ \text{in seconds}\end{array}\right)}$$

Calculation of dye dilution method

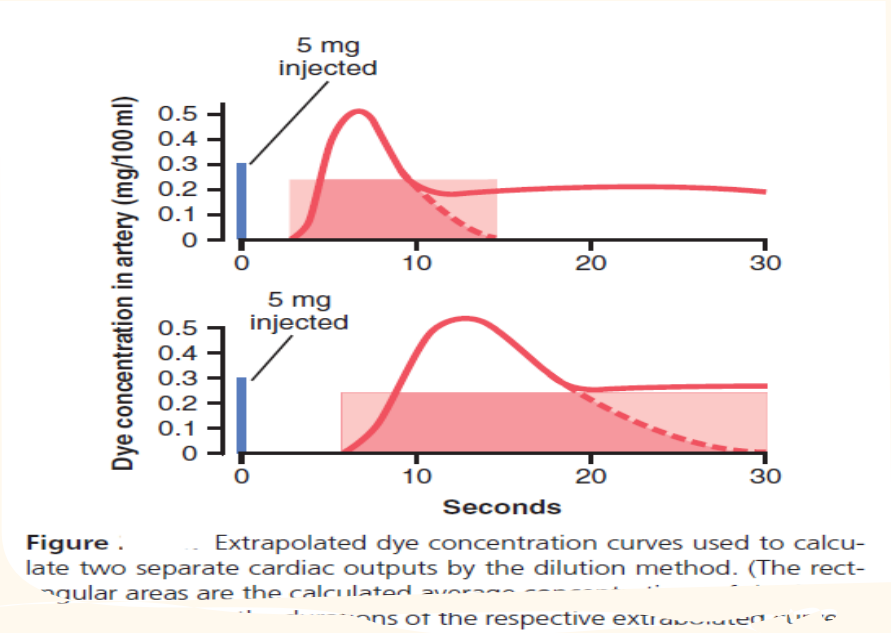


Figure . Extrapolated dye concentration curves used to calculate two separate cardiac outputs by the dilution method. (The rectangular areas are the calculated average concentrations of the respective extrapolated curves

- 5 mg total injected dye
- Average Blood level of dye detected 0.25/100ml
- Time taken by the distribution dye = 12 sec
- What is the ratio of detected dye with total injected dye= 5/0.25=20 (means 20th portion is present in 100 ml,)
- it means-20x100=2000ml in 12 sec (time taken by dye distribution)
- If we want to know the total blood amount in body for 5 mg total dye dilution in 12 sec,, then 2000x 60/12=10,000ml

OR = 10 litre. Total cardiac output. In this case.

Non Invasive Methods for cardiac output measurement

ECHOCARDIOGRAPHY

- **Trans-esophgeal echo cardiography**
- **Trans-thorasic echocardiography**
- **Doppler US**

Trans Esophageal Echocardiography (TEE) / Doppler)

- A widely used monitor in perioperative setting
- Is an important tool for the assessment of cardiac structures, filling status and cardiac contractility
- Aortic pathology can also be detected by TEE

Doppler technique

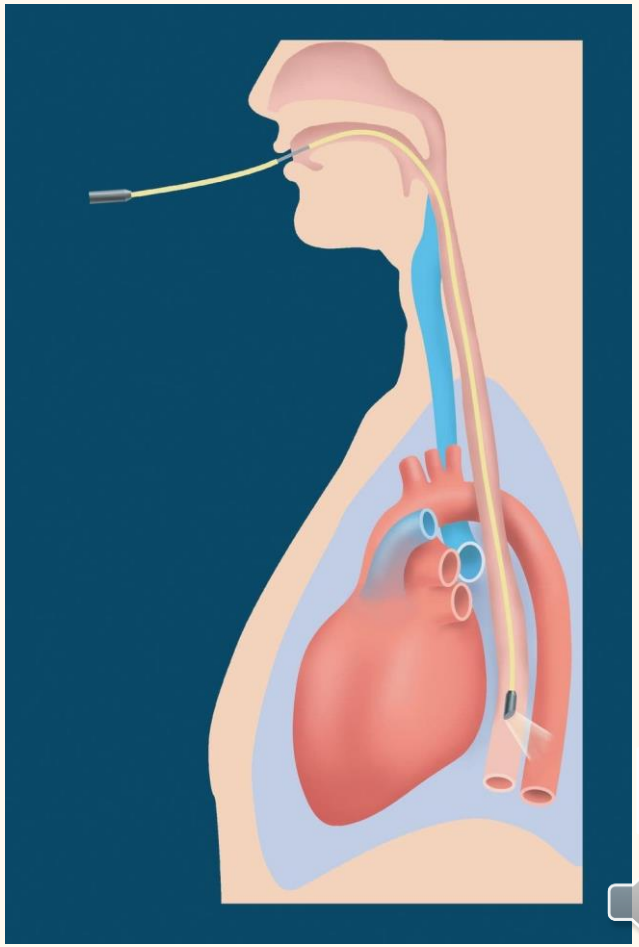
- is used to measure CO by Simpson's rule measuring SV multiplied by HR

- Measurement can be done at the level of pulmonary artery, mitral or aortic valve
- TEE can quantify Cardiac Output more precisely by measuring both the velocity and the cross-sectional area of blood flow at appropriate locations in the heart or great vessels

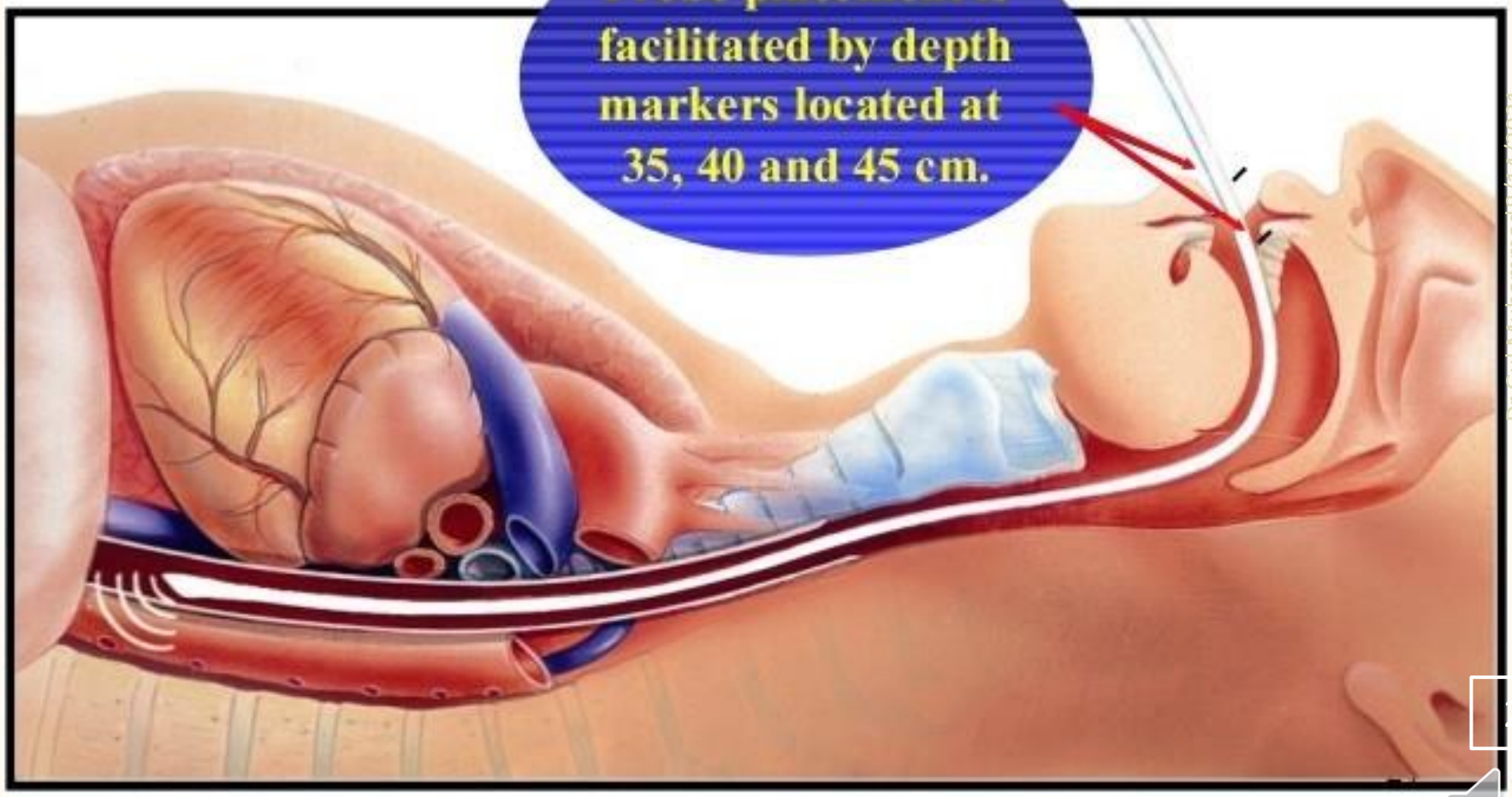
  i.e. Flow = CSAX Velocity

- SV=flow X ET (Systolic Ejection time)

- CO=SV X HR



Probe Placement



Trans Thoracic Echocardiography (TTE)

- Echocardiography is cardiac ultrasound
- Can be used to estimate Cardiac Output by direct visualization of the contracting heart in real time
- Gaining acceptance as one of the safest and most widely used cardiac output monitors in the critically ill

Cardiac reserve

- Cardiac reserve refers to the heart's ability to adjust to the demands placed upon it.

Cardiac reserve =

[cardiac output during stress - cardiac output at rest.]

- In the normal young adult the resting cardiac output is about 5-6 L/min and the cardiac reserve is nearly 30 L/min.

Mechanisms of cardiac reserve:

Short term mechanisms

-Rapid onset.

- Increase CO according to moment to moment

increase in body needs.

Long term mechanisms

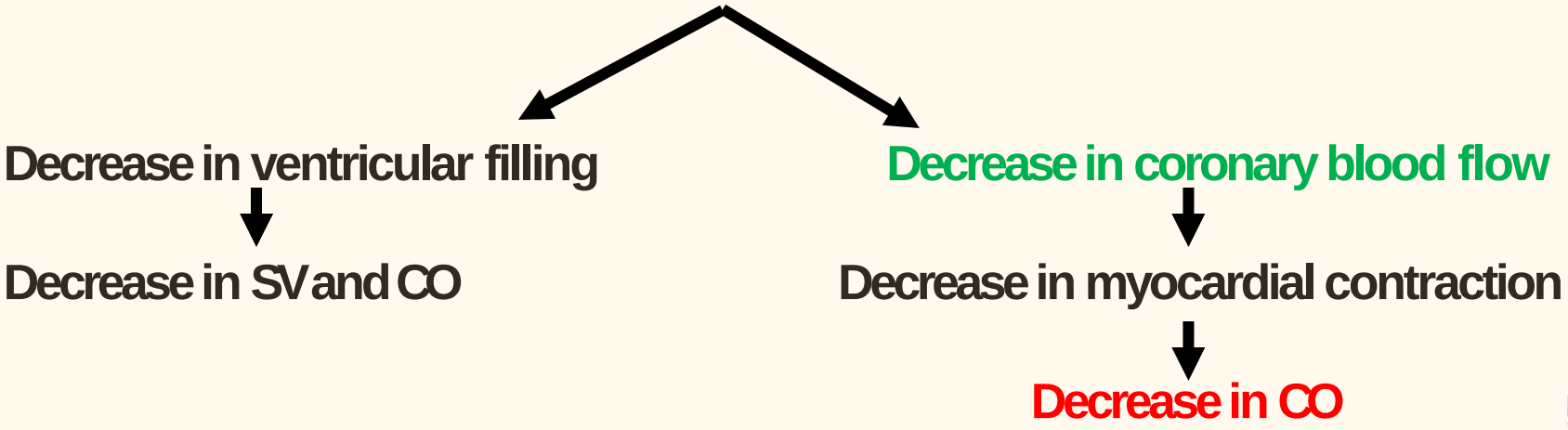
-Slow and gradual.

-Occur in cases of prolonged excess work done by heart eg: Increased arterial blood pressure (hypertension).

Short term mechanisms:

Heart rate (HR) reserve:

- The possibility of the increase of heart rate up to 2 – 3 times.
- Heart rate reserve is limited because the increase in heart rate above 180 beats/min causes decrease in the CO.
- As this marked increase in the heart rate will be associated with marked decrease in diastolic period causing:



Stroke volume reserve:

- Increase in SV causes increase of CO.
- This mechanism is mediated by either:

Increase of the EDV (EDV reserve)

Decrease of the ESV (ESV reserve)

End diastolic Volume (EDV) reserve:

- Increase of the venous return (as in strenuous exercise) increases EDV stretch of ventricular muscle fibers *ncreases force of contraction of cardiac muscle and increases SV and CO (Starling law.)*
- **But** This mechanism is limited as the marked increase in EDV causes overstretch of ventricular muscle fibers which will decreases the force of contraction in cardiac muscle, and a decrease of cardiac output will occur.

ESV reserve:

- Increased sympathetic stimulation to the heart increases the force of cardiac muscle contraction and decreases the ESV. This will increase SV and CO.

- This mechanism is also limited as the marked decrease in ESV causes myocardial injury (athletic injury).

Longterm mechanisms:

1. Cardiac muscle hypertrophy:

- It is the increase in the size of the individual cardiac muscle fiber (increase its protein content). This increases the force of contraction of cardiac muscle increase SV and CO.
- Marked hypertrophy of the myocardium is **not associated with parallel increase in coronary blood flow.**
- This cause myocardial ischemia with subsequent decrease in the force of contraction of cardiac muscle. This will decrease the SV and CO.

2. Dilatation of cardiac chambers:

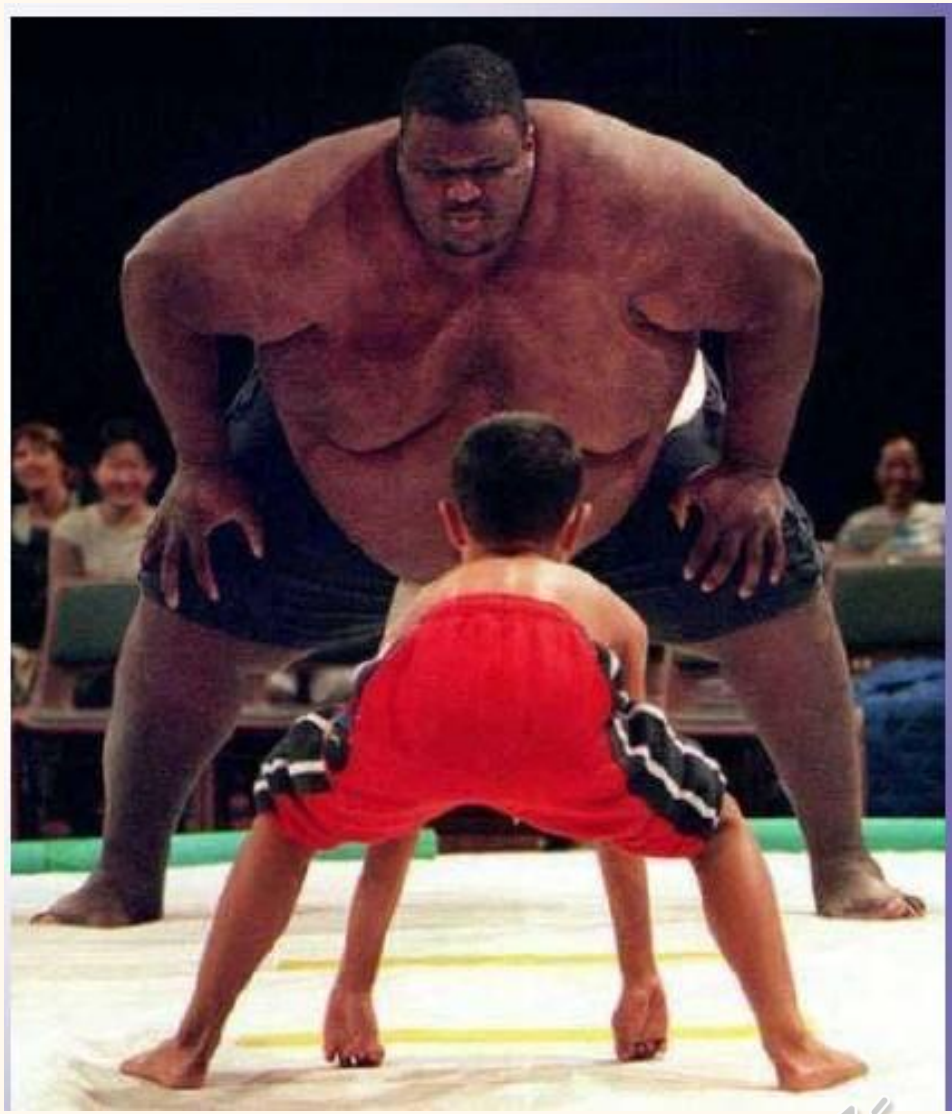
- This mechanism occurs when the blood accumulated inside the cardiac chambers as in case of heart failure.
- This dilatation of the cardiac chambers causes stretch of the cardiac muscle fibers Increases its force of contraction according to Frank-Starling law.
- Marked increase in EDV causes overstretch of ventricular muscle fibers and decreases force of contraction of cardiac muscle which will decrease the SV and cardiac output

- In severe heart failure the cardiac reserve can be markedly diminished or totally abolished

- The cardiac reserve is affected by a number of different factors like:
  - The percentage of fibrosis,
  - The degree of apoptosis or necrosis and
  - Presence of auto-antibodies against the β1 adrenoceptor in the heart.

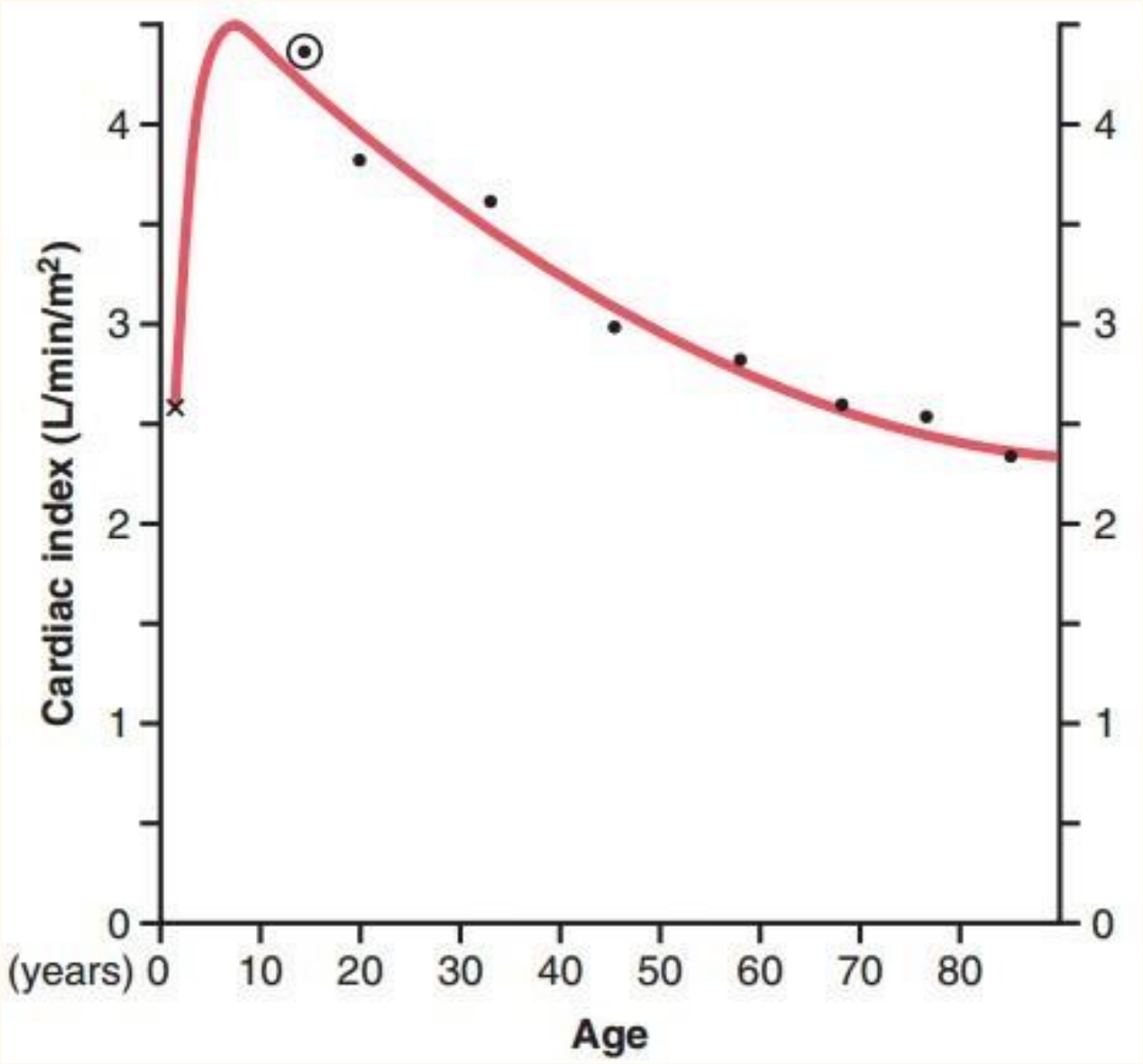
Cardiac Index

- **Both of these people's hearts pump 3 liters of blood per minute.**
- **Who feels better?**
- **The smaller one!**
- **Why ?**
- ***Less body surface area.***



Cardiac Index

- Is the *cardiac output per square meter of body surface area*
- **Cardiac output** is frequently stated in terms of the **cardiac index**
- Average human being who weighs 70 kilograms has a body surface area of about **1.7 square meters**, which means that the normal average cardiac index for adults is about **3 L/min/m²** of body surface area



Variation of cardiac index with age

THANK YOU !!!

- **Guyton and hall textbook of medical physiology, *13th edition***
- ***Ganong's Review of Medical Physiology - 23rd Edition***
- ***Through net,,***

  http://heart.bmj.com/content/79/3/289#BIBL