Cardiac output. Frank-Starling curve. Cardiac work.

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Cardiac output

- The volume of blood pumped by the heart per minute.

$$CO = HR \times SV$$

$$BP = CO \times SVR$$

- *At rest, CO is 5lt/min*
- *In exercise it may rise to 35 L/min.*
- *CI=CO/BSA=2.5-4.2 L/min/m2).*

The CO is pivotal in maintaining arterial BP (BP=CO X SVR) and oxygen delivery.

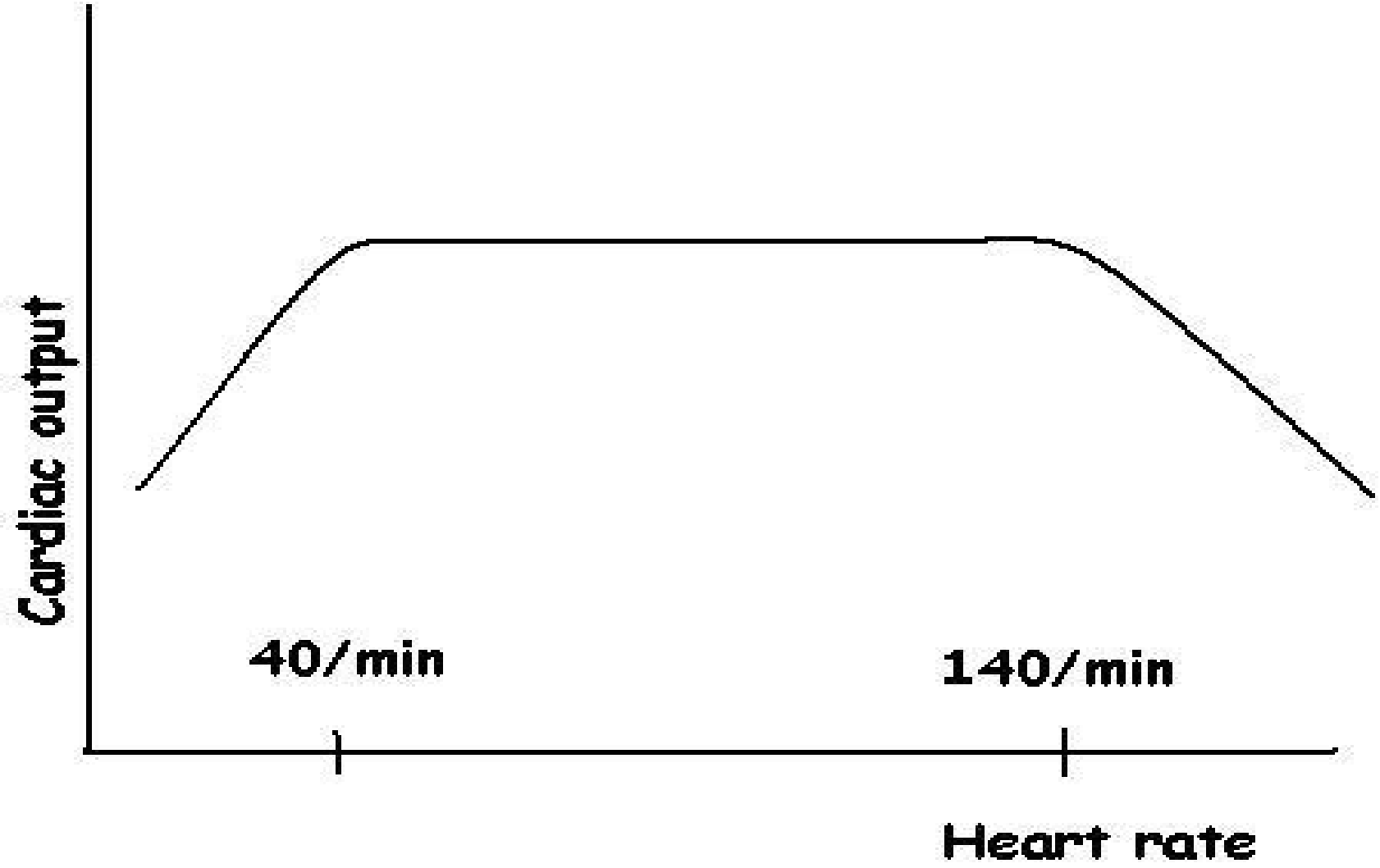
- *DO2= CO x Hb x 1.31 x % Sat*

mlO2/min lt/min gr/lt mlO2/grHb

Distribution of CO

- Heart 5%
  Brain 14%
  Muscle 20%
  Kidneys 22%
  Liver 25%
  Rest of the body 14%

HR effect on CO (Treppe effect)



Stroke Volume

Stroke volume: the volume of blood ejected by the ventricle with a single contraction.

SV = EDVol – ESVol

EF=SV expressed as a percentage.

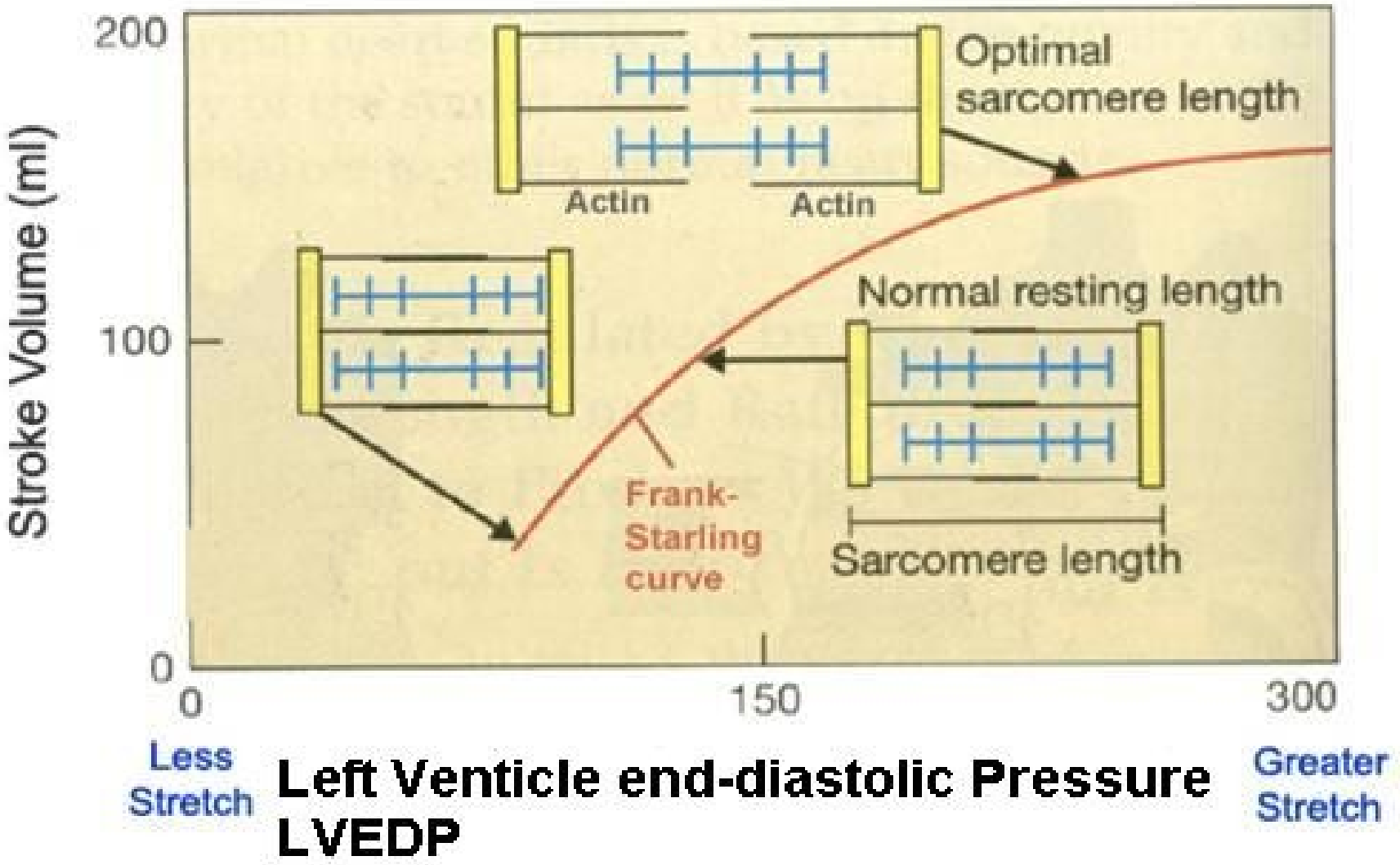
Determinants of SV:

1. Preload
2. Afterload
3. Contractility

Preload

1. ***Preload**: The initial length of the muscle fibre before contraction.*
   - *The filling pressure of the ventricles.*
   - *Described by the Frank-Starling curve.*
   - *In clinical practice CVP(RV) and PAWP(LV) are used. (Several limitations).*
     - *In normal individuals, CVP correlates with filling pressures of both right and left atria, this is not true in the following disease states:*
     - *Left ventricular failure with pulmonary oedema*
       Interstitial pulmonary oedema
       Chronic pulmonary disease
       Valvular heart disease

The Frank-Starling curve

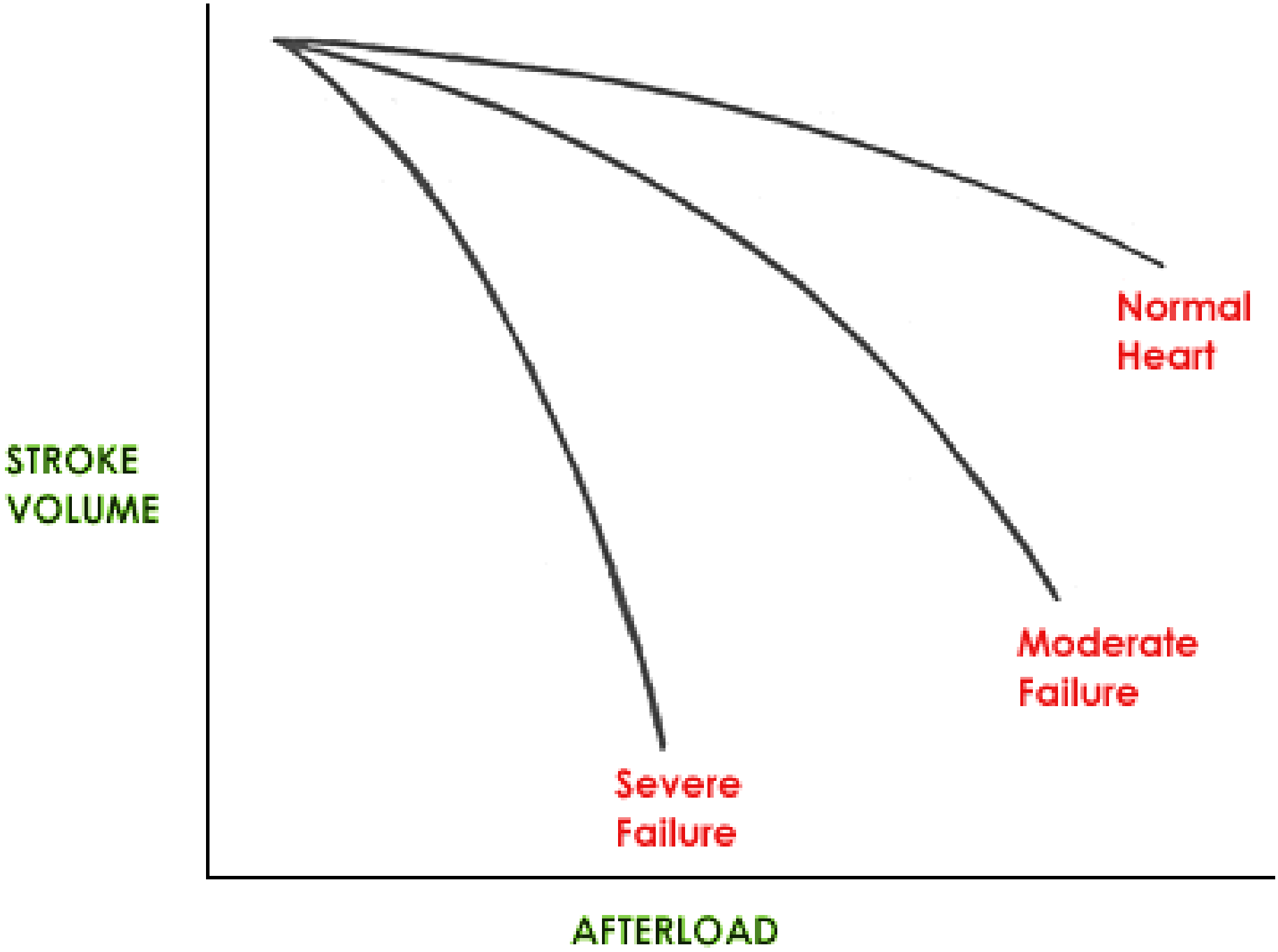


Frank-Starling law

- In isolated myocytes the tension developed during contraction is dependent on the initial length of the muscle fibre.
- Laplace's law: $P=2T/R$
- For an isolated muscle fibre it translates into a relationship between SV and LVEDP

Afterload

2. **Afterload:** The tension developed in the ventricular wall during systole.
   - Affected by preload, SVR, and external pressure (for example Positive Pressure Ventilation)
   - How forcefully the ventricles have to contract to eject blood.


STROKE VOLUME
Normal Heart
Moderate Failure
Severe Failure
AFTERLOAD

3. *Contractility:* *The amount of mechanical work that is done for a given preload and afterload.*

Increased contractility

1. *Sympathetic stimulation*
2. *Positive inotropes*

Reduced contractility

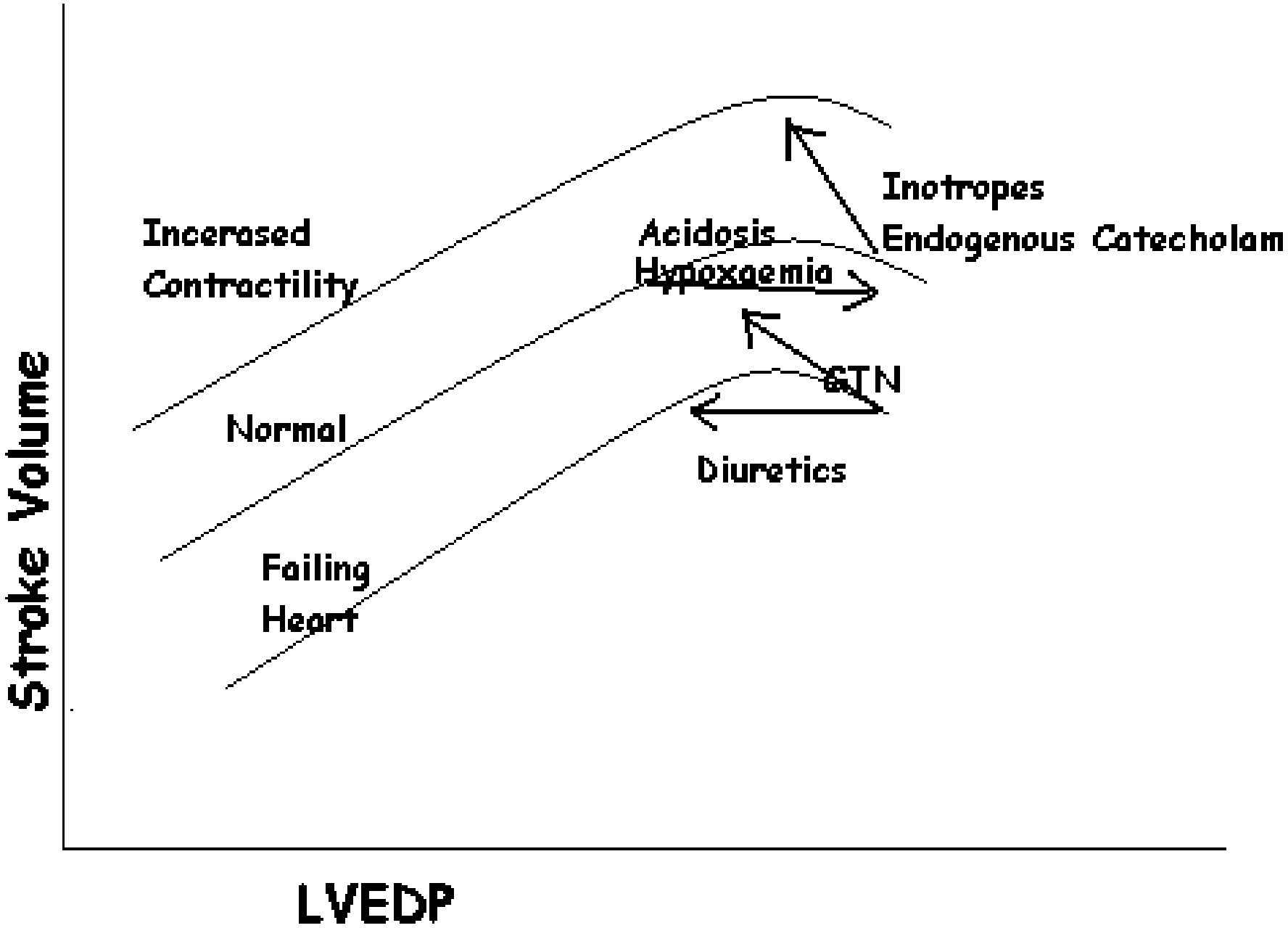
1. *Parasympathetic stimulation*
2. *Negative inotropes (b-block)*
3. *Ischaemia/Infarction*
4. *Hypoxaemia*
5. *Acidocis*
6. *HypoCalcaemia*

Clinical application

- *In low CO states leading to hypoperfusion, the*

1. *HR,*
2. *preload,*
3. *afterload and*
4. *contractility should be addressed- **in that order**.*

(No point trying to pharmacologically improve the contractility of a heart that is either too empty or too distended to eject).


Stroke Volume
LVEDP
Incerased
Contractility
Normal
Failing
Heart
Acidosis
Hypoxaemia
Inotropes
Endogenous Catecholam
GTN
Diuretics

Methods of measuring CO

- Invasive

Dilution/ thermodilution techniques

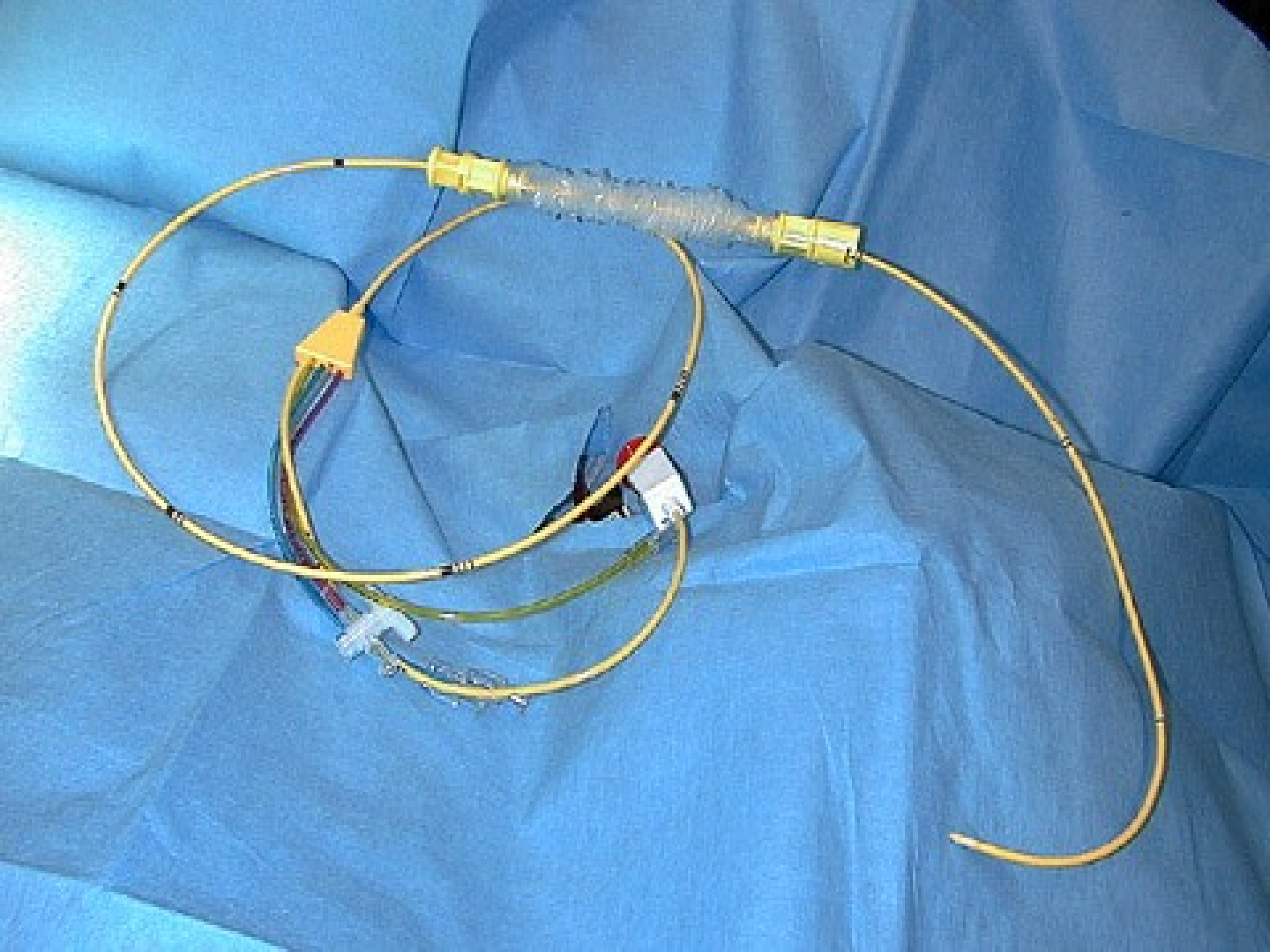
- Non invasive

TOE

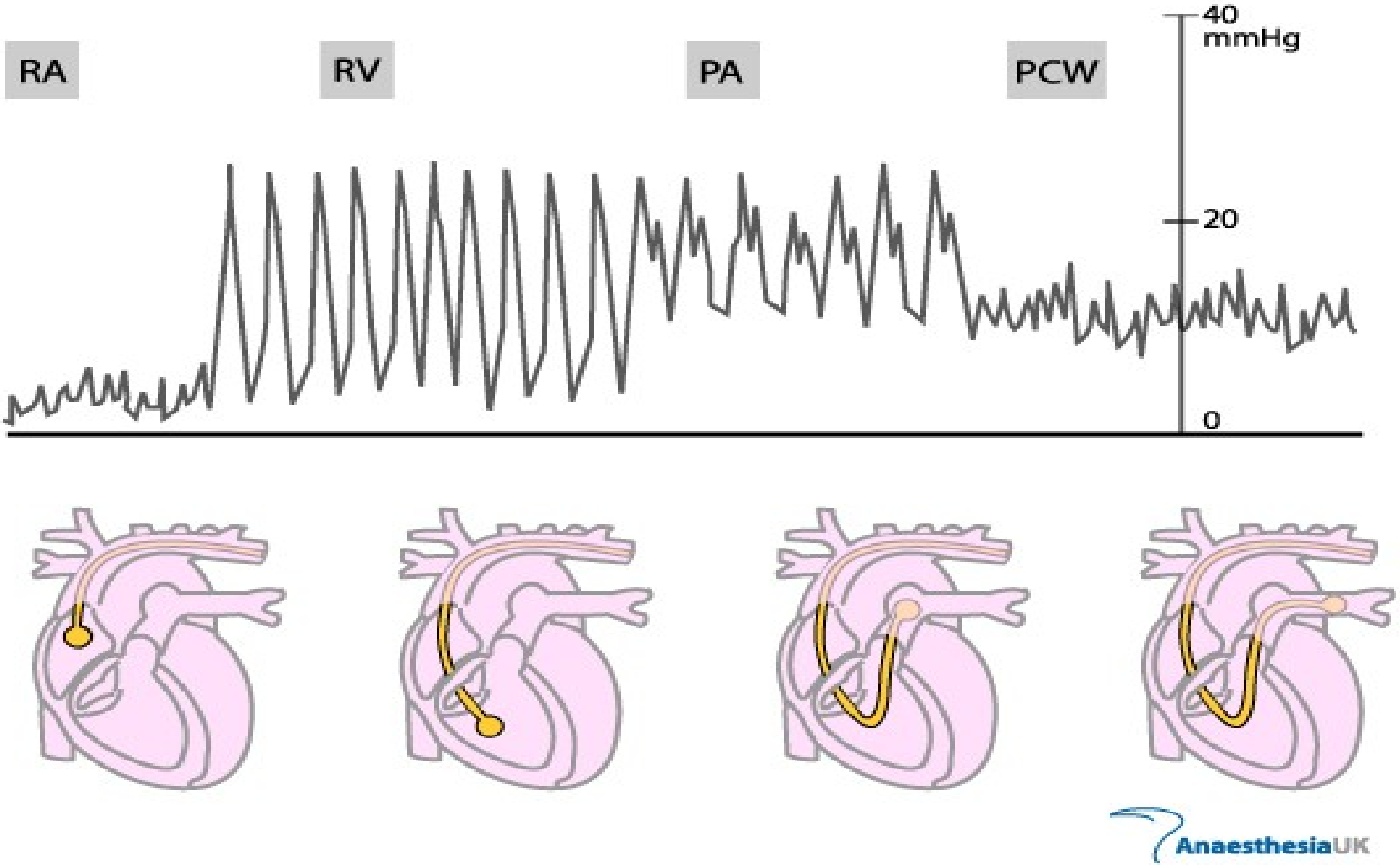
Transthoracic Echo

PA catheter

- Proximal lumen: 25 cm from tip, lies in right atrium, measures central venous pressure (CVP).
- Distal lumen: at tip of catheter, lies in a branch of the pulmonary artery, connected to a pressure transducer.
- Balloon lumen: permits introduction of 1.5 ml of air into the balloon at the distal tip.
- Thermistor lumen: bead situated 4 cm from the tip of the catheter and measures temperature.



Characteristic intracardiac pressure wave forms during passage through the heart



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Complications

1. Arrhythmias on insertion
2. Knotting of catheter in right ventricle
3. Balloon rupture
4. Pulmonary infarction
5. Infection

The Fick principle

- Fick described the following relationship in the 19th century:

  $Q = M / (V - A)$

- Q is the volume of blood flowing through an organ in a minute.
- M the number of moles of a substance added to the blood by an organ in one minute,
- V and A are the venous and arterial concentrations of that substance.
- This principle can be used to measure the blood flow through any organ that adds substances to, or removes substances from, the blood. The heart does not do either of these but the CO equals the pulmonary blood flow, and the lungs add oxygen to the blood and remove carbon dioxide from it.

Fick's method

PVein[O2]=200ml/lt

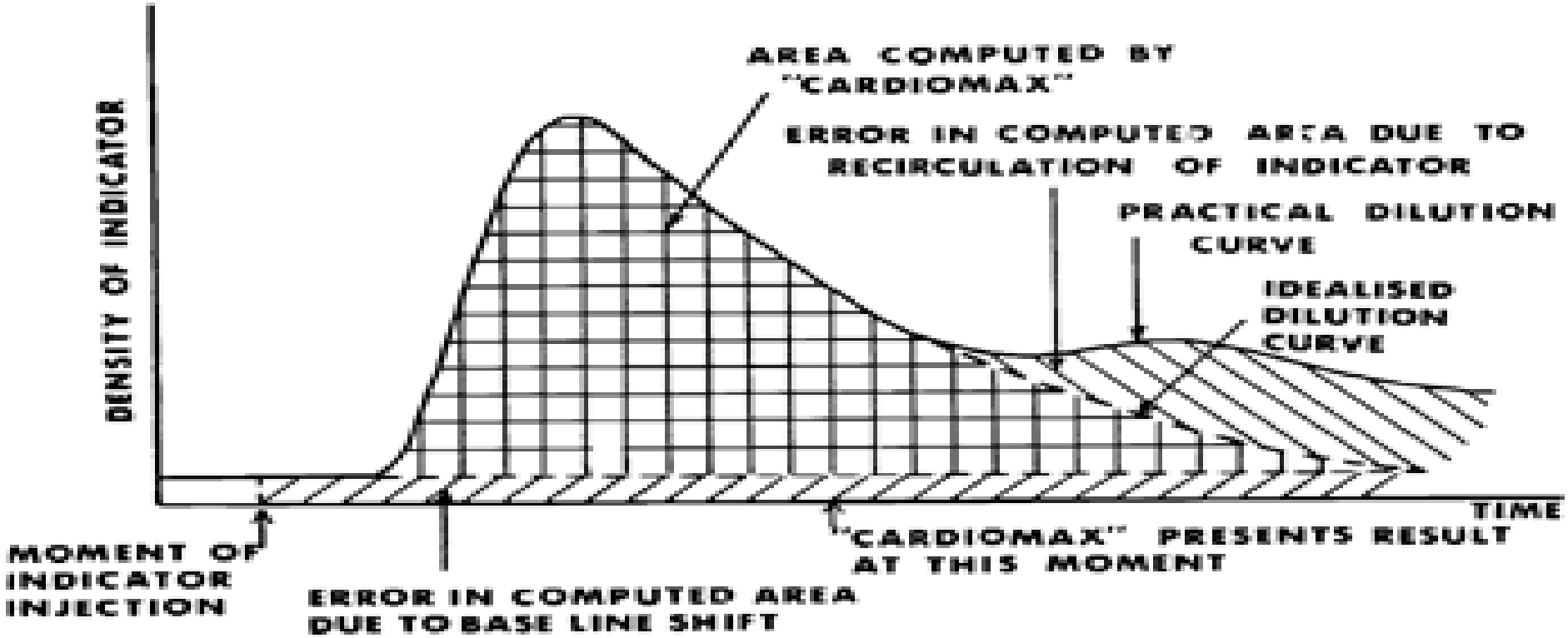
PArtery[O2]=150ml/lt

At rest the blood takes 250ml/min of O2 from the lungs.

CO (at rest)=5lt/min

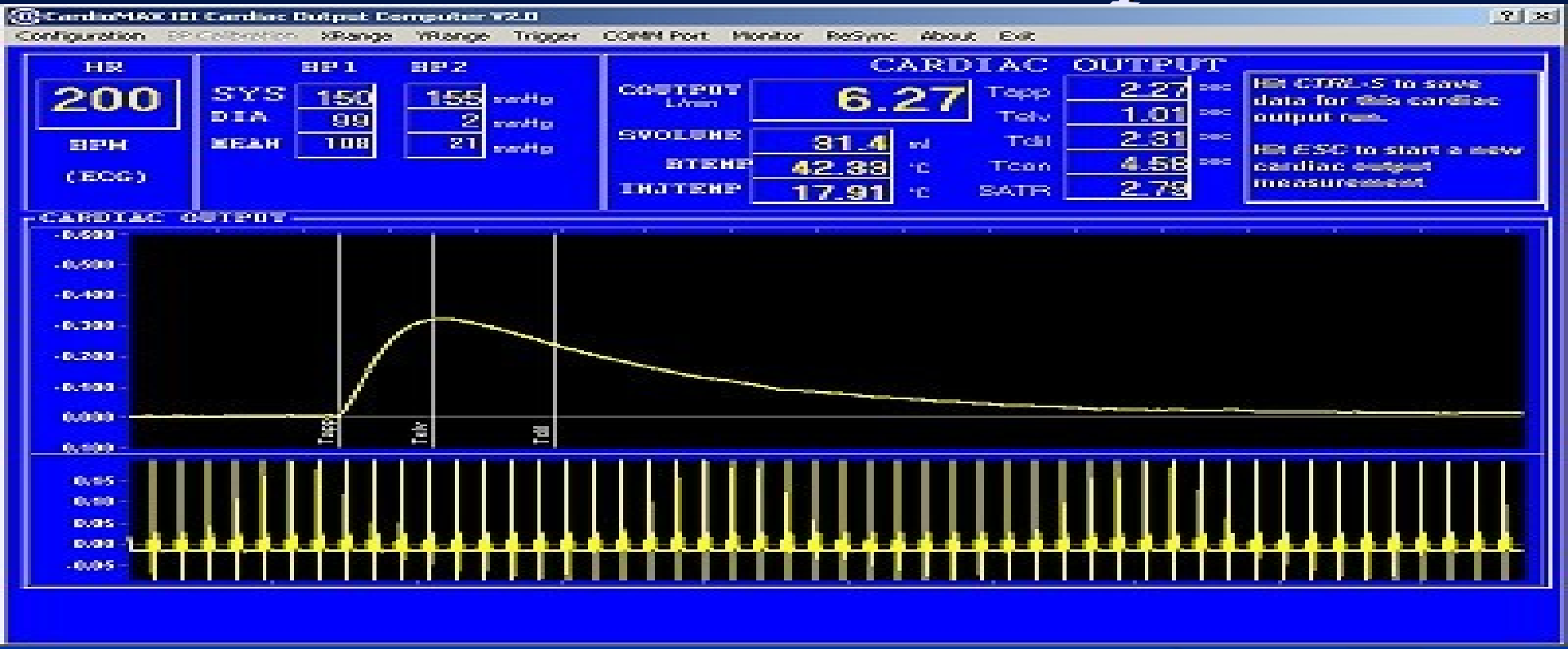
Adolf Fick 1829-1901

Dilution techniques



- *A known amount of dye is injected into the pulmonary artery, and its concentration is measured peripherally. Indocyanine green is suitable due to its low toxicity and short half-life. A curve is achieved, which is replotted semi-logarithmically to correct for recirculation of the dye. CO is calculated from the injected dose, the area under the curve (AUC) and its duration. (Short duration indicates high CO).*

Thermodilution technique



- *5-10 ml cold saline injected through the port of a pulmonary artery catheter. Temperature changes are measured by a distal thermistor. A plot of temperature change against time gives a similar curve to the dye curve (but without the second peak).*
- *Calculation of CO is achieved using the Stewart-Hamilton equation.*
- *Application of this equation assumes three major conditions; complete mixing of blood and indicator, no loss of indicator between place of injection and place of detection and constant blood flow.*

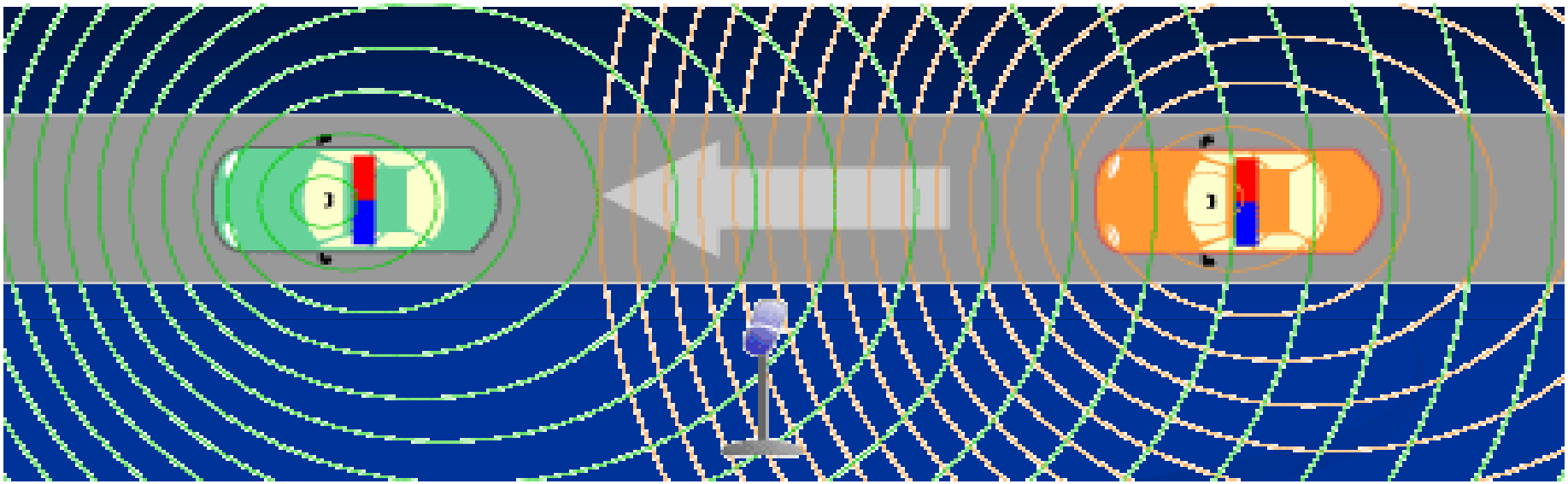
Measured values

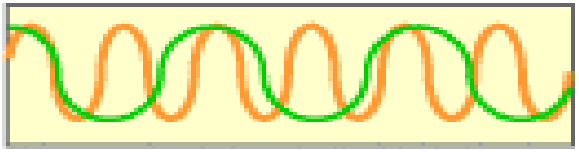
- **CVP**: Central Venous Pressure 1-6 mm Hg (reflects right atrial pressure).
- **PAP**: Mean Pulmonary Artery Pressure Systolic 15-30mm Hg, Diastolic 6-12mm Hg.
- **PCWP**: Pulmonary Capillary Wedge Pressure 6 - 12mm Hg. Estimates left atrial heart pressure and left ventricular end diastolic pressure.
- **CO**: Cardiac Output 3.5 - 7.5 L/min
- **Sv02**: Mixed venous partial pressure of oxygen (70 - 75%). Drawn from the end of the pulmonary artery catheter. Used to calculate how well oxygen is extracted by the tissues.

Non-invasive (TOE)

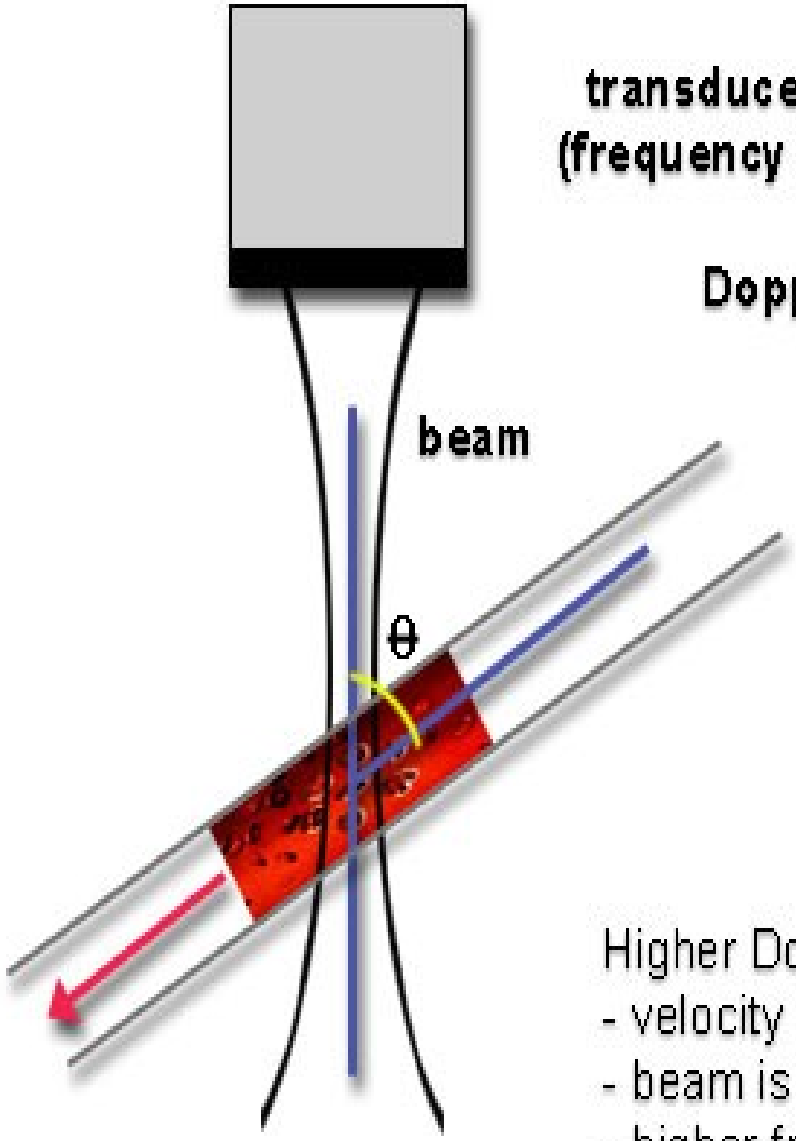
- *These machines transmit an ultrasonic vibration into the body and record the change in the frequency of the signal that is reflected off the red blood cells.*
- *Doppler techniques measure **velocity**, not flow; the flow could be obtained by integrating the signal over the cross-sectional area of the vessel.*
- *Transoesophageal echocardiography (TOE) can measure the **length of blood in the ascending aorta in unit time.** This is multiplied by the cross-sectional area of the aorta to give stroke volume.*

Doppler effect





- *is the apparent change in frequency and wavelength of a wave that is perceived by an observer moving relative to the source of the waves.*



$$\text{Doppler frequency } (f_d) = \frac{2 \cdot f_t \cdot V \cdot \cos\theta}{c}$$

fd - doppler shift
c - is speed of sound in tissue
ft - transmited beam
V - velocity of the blood
0 - angle of incidence between the ultrasound beam and the direction of the flow

Higher Doppler frequency obtained if:
- velocity is increased
- beam is more aligned to flow direction
- higher frequency is used

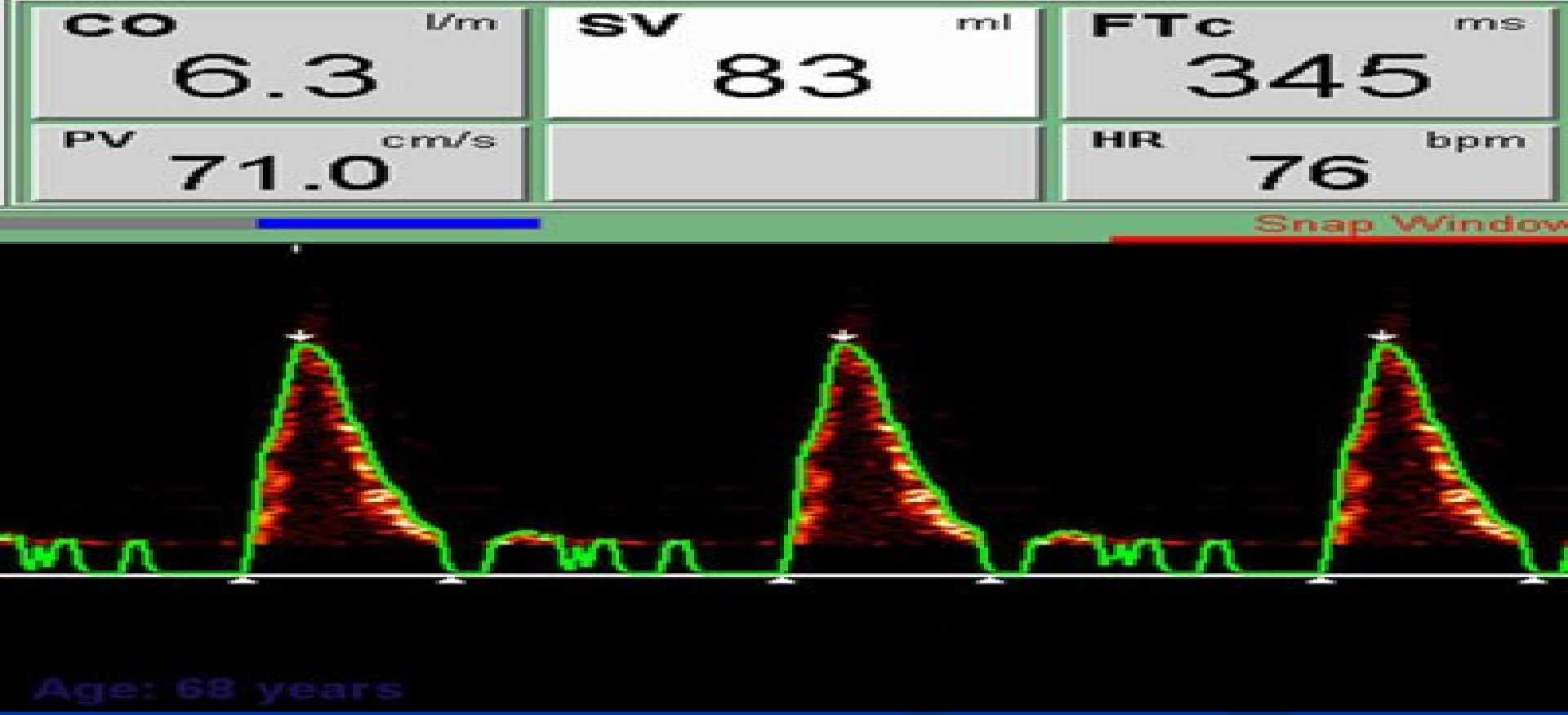
Indications for oesophageal doppler monitoring

- **Intensive Care Unit**

  Post-operative monitoring
  Unstable Haemodynamics
  ARDS

  General Surgery

  Patients over 65
  Fluid loss >500ml
  ASA III
  Procedures exceeding 2 hours



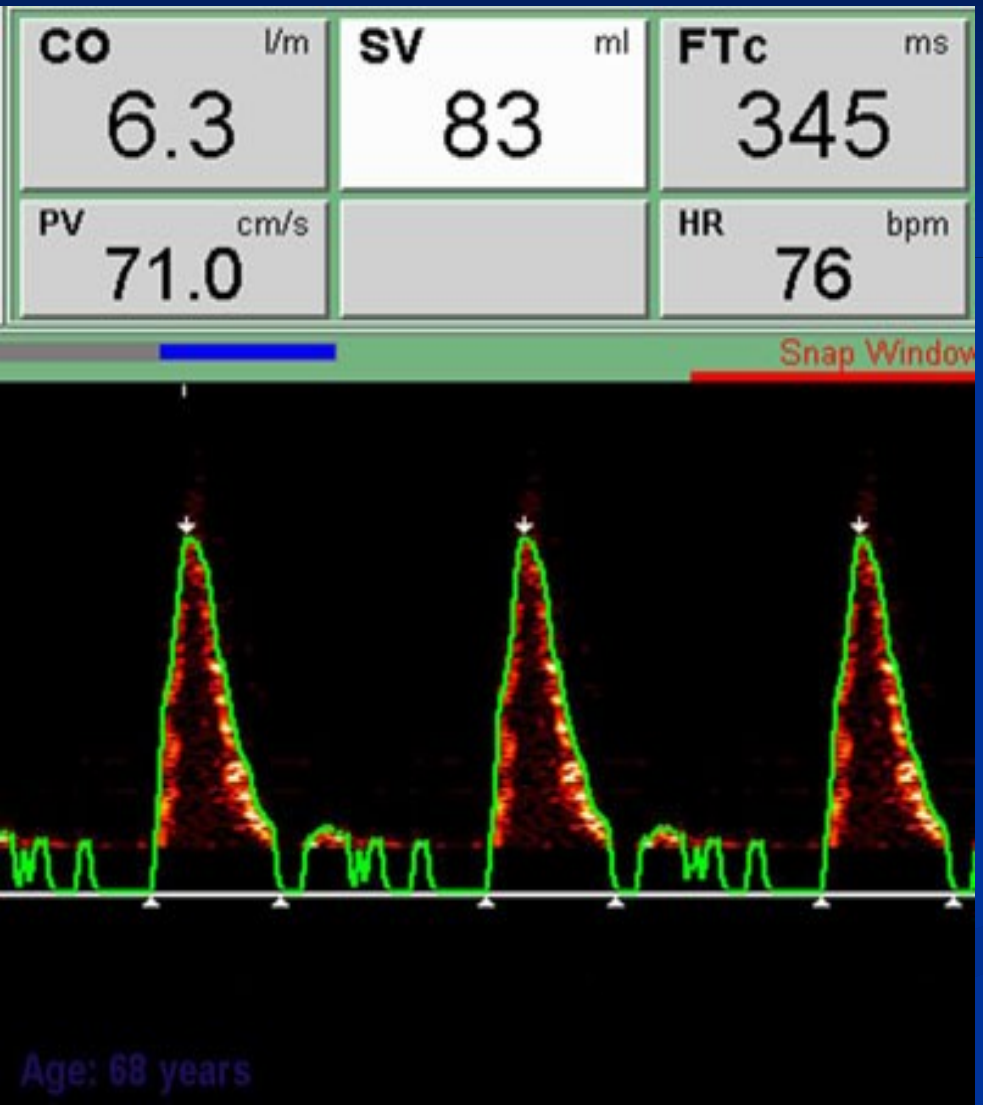
- *The key preload parameter of interest is the **flow time; the time required from the start of the waveform upstroke to return to baseline.** Flow time represents the duration of left ventricular systole and makes up one third of the cardiac cycle or cycle time. Since the flow time (FT) is heart rate dependent it is typically corrected (FTc) to a rate of 60 bpm and compensates for the change in duration of systole.*
- ***Low FTc value (<330 msec)** is seen with a **vasoconstricted** circulation e.g. hypovolaemia, excess arterial constriction (e.g. vasoconstrictors), major circulatory obstruction (e.g. pulmonary embolus).*
- ***Raised FTc (>360 msec)** is seen with a **vasodilated** circulation e.g. sepsis, hyperpyrexia*

Interpret the waveform

FTc is inversely related to SVR

PV can be used as an index of contractility

Concurrent shifts in FTc and PV can indicate changes in afterload



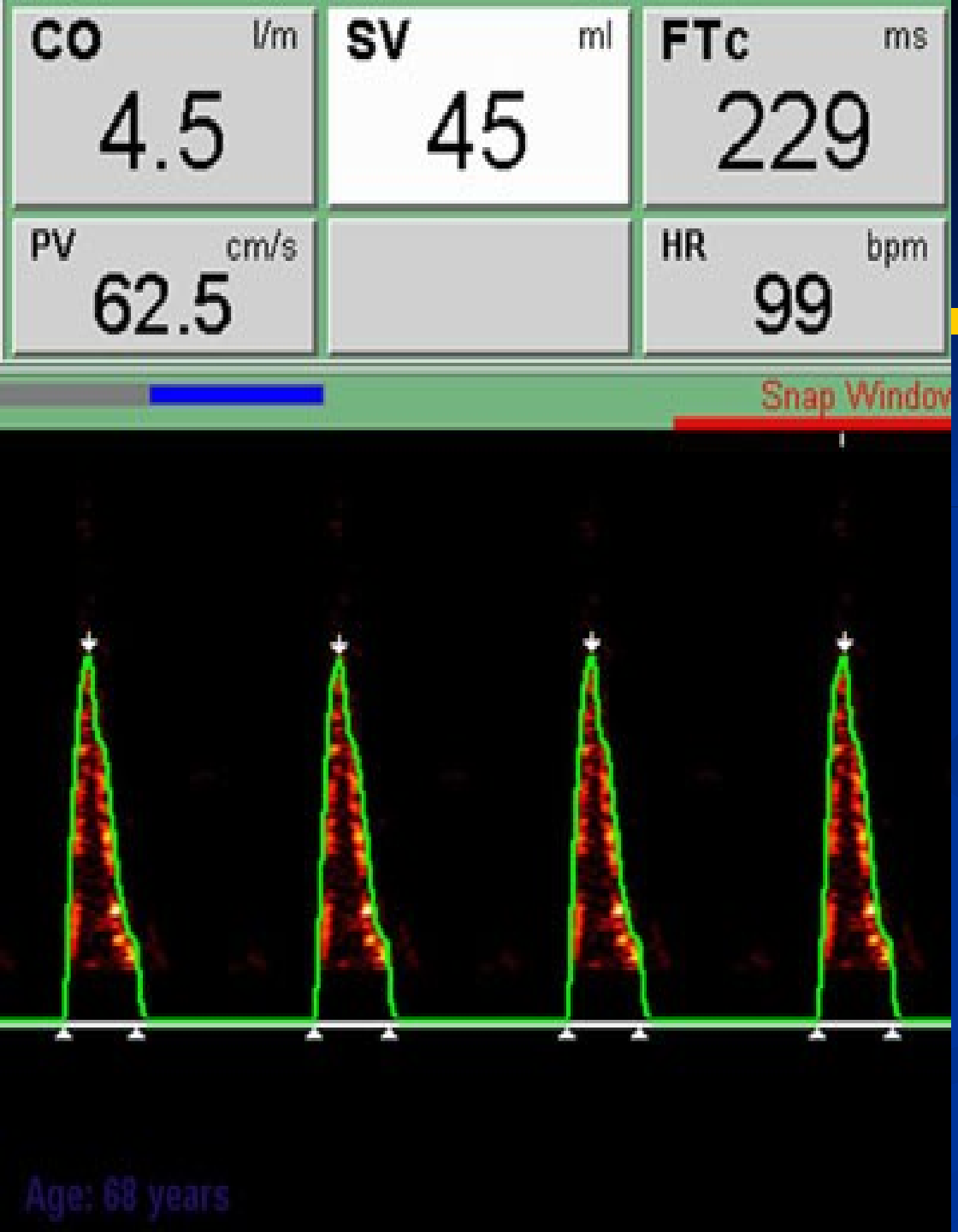
- ***Normovolaemia***

Whip crack sound

Wide bases – within normal ranges

Tall peaks – adequate Peak Velocity for age

All parameters appear to be within the normal limits



- *Hypovolaemia*

Narrow waveform thus low FTC

Good PV

Decreased CO, SV & borderline high HR



- *Hypovolaemia following fluid challenge 200mls*

Audible change;

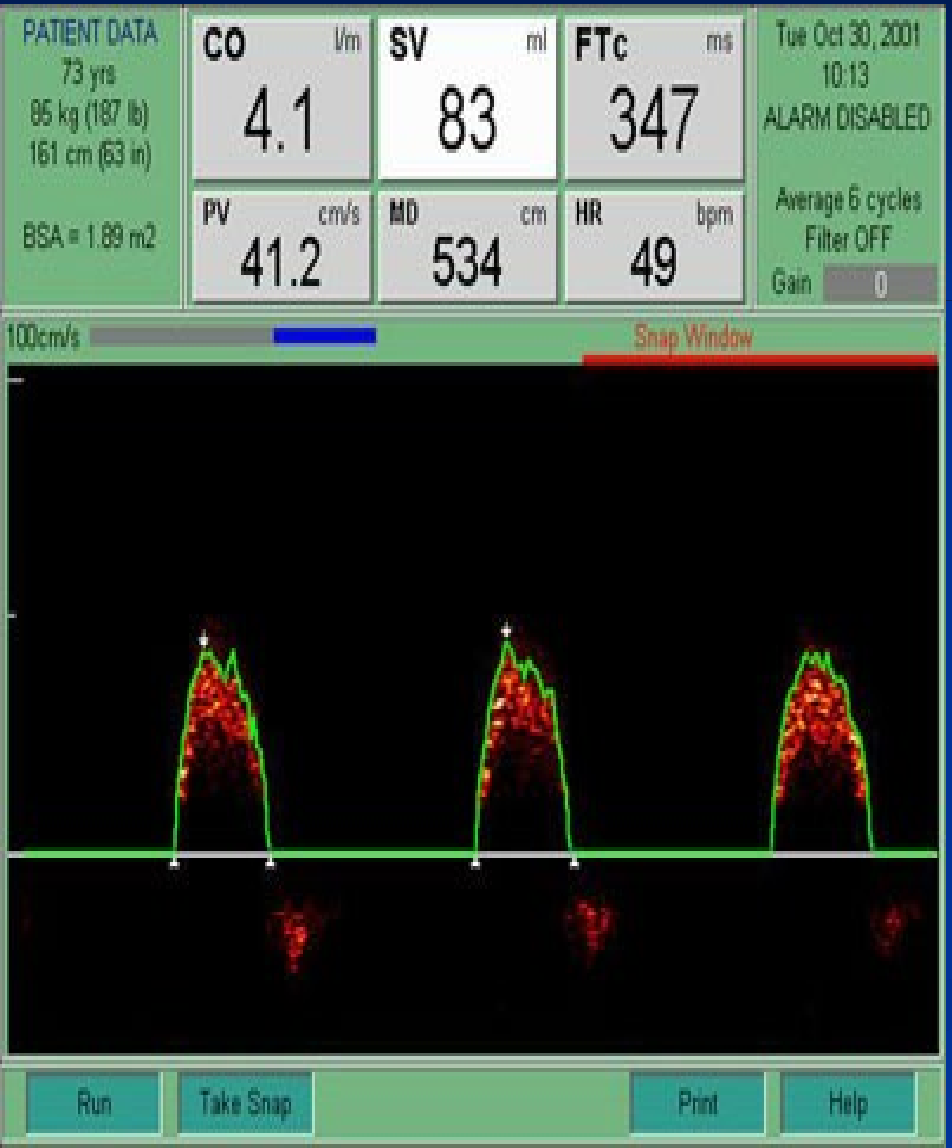
Widened bases – FTc improving

Good peaks – PV within range
CO, SV improving



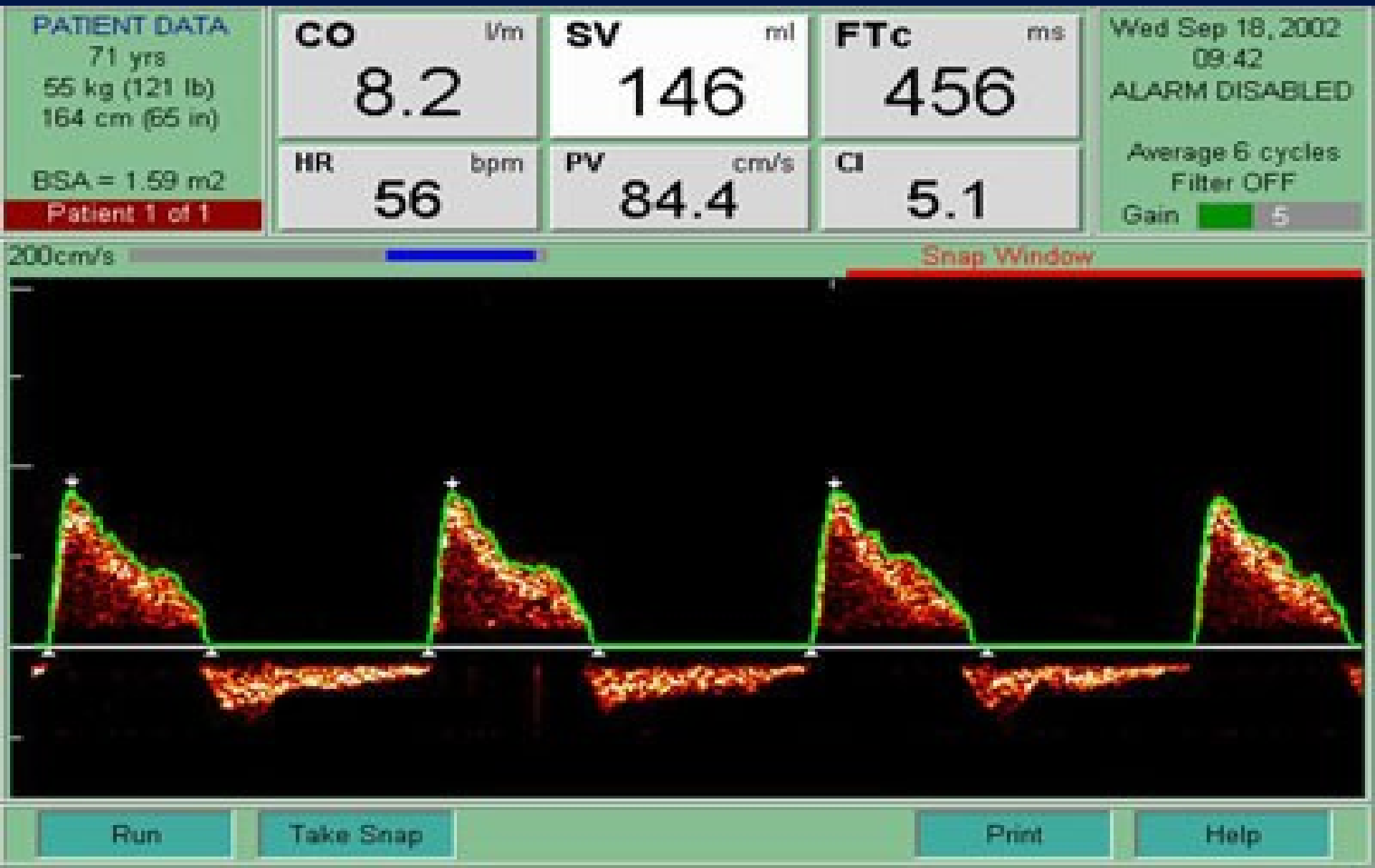
- *Hypovolaemia following fluid challenge 600mls*

Aortic stenosis pre/post AVR

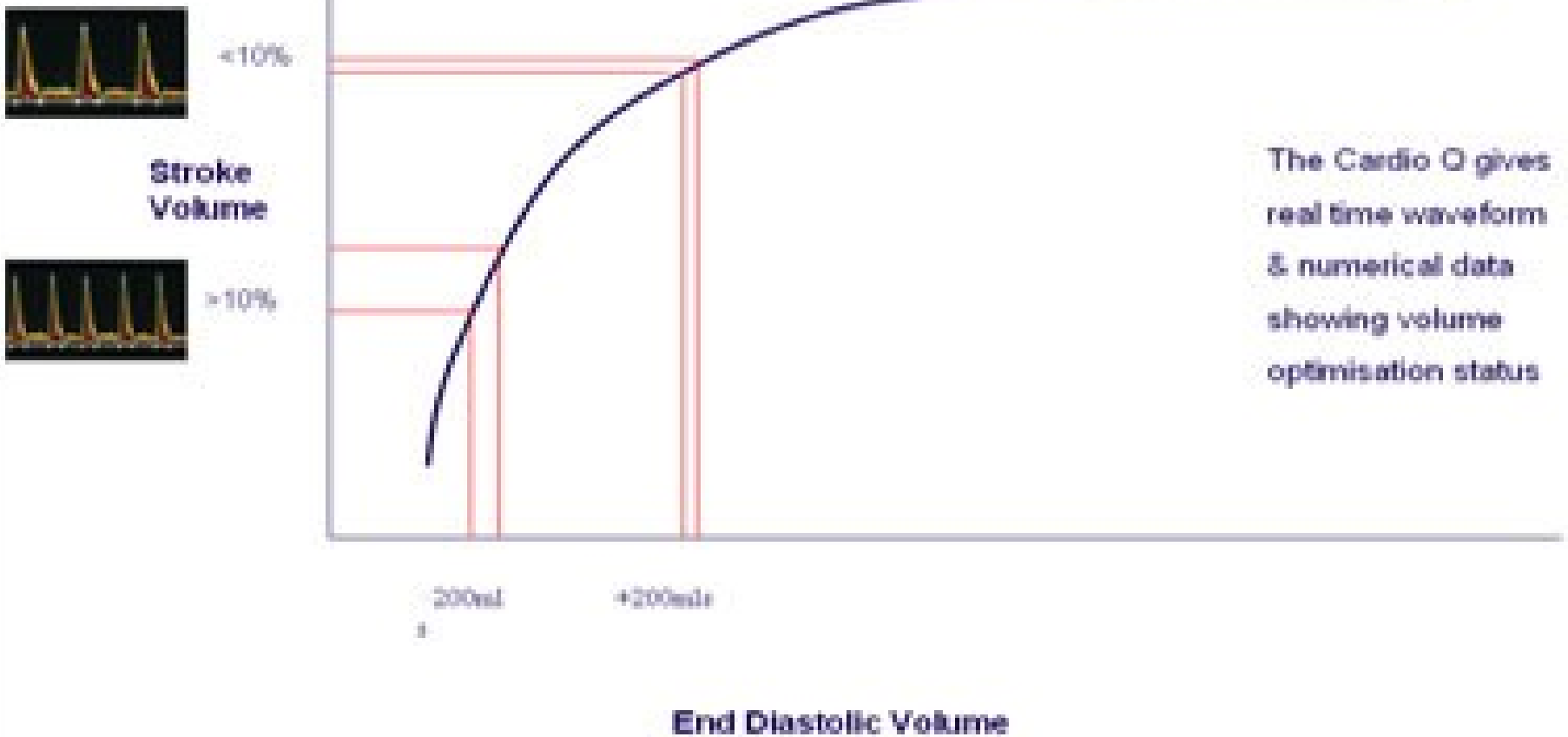




Aortic Regurgitation

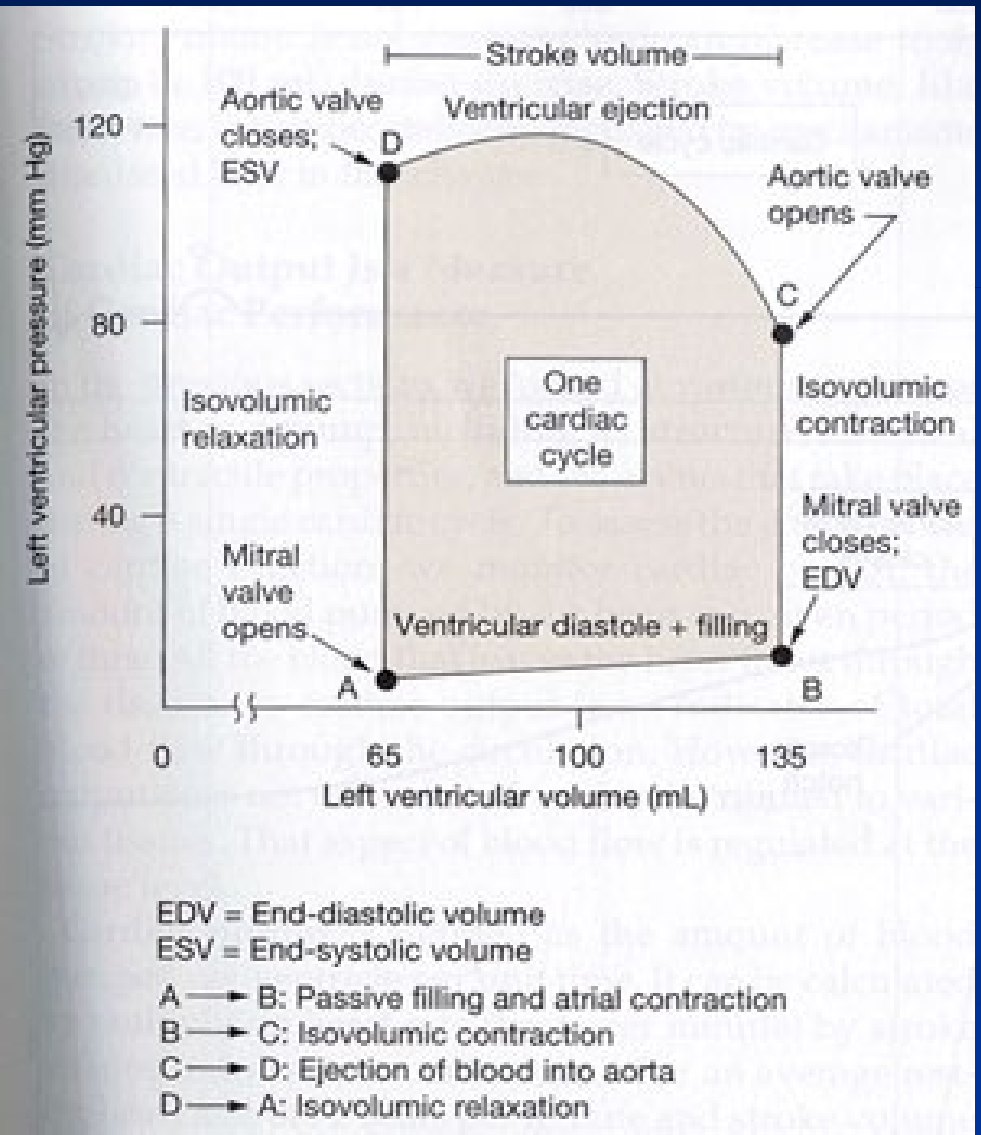


Starling Curve



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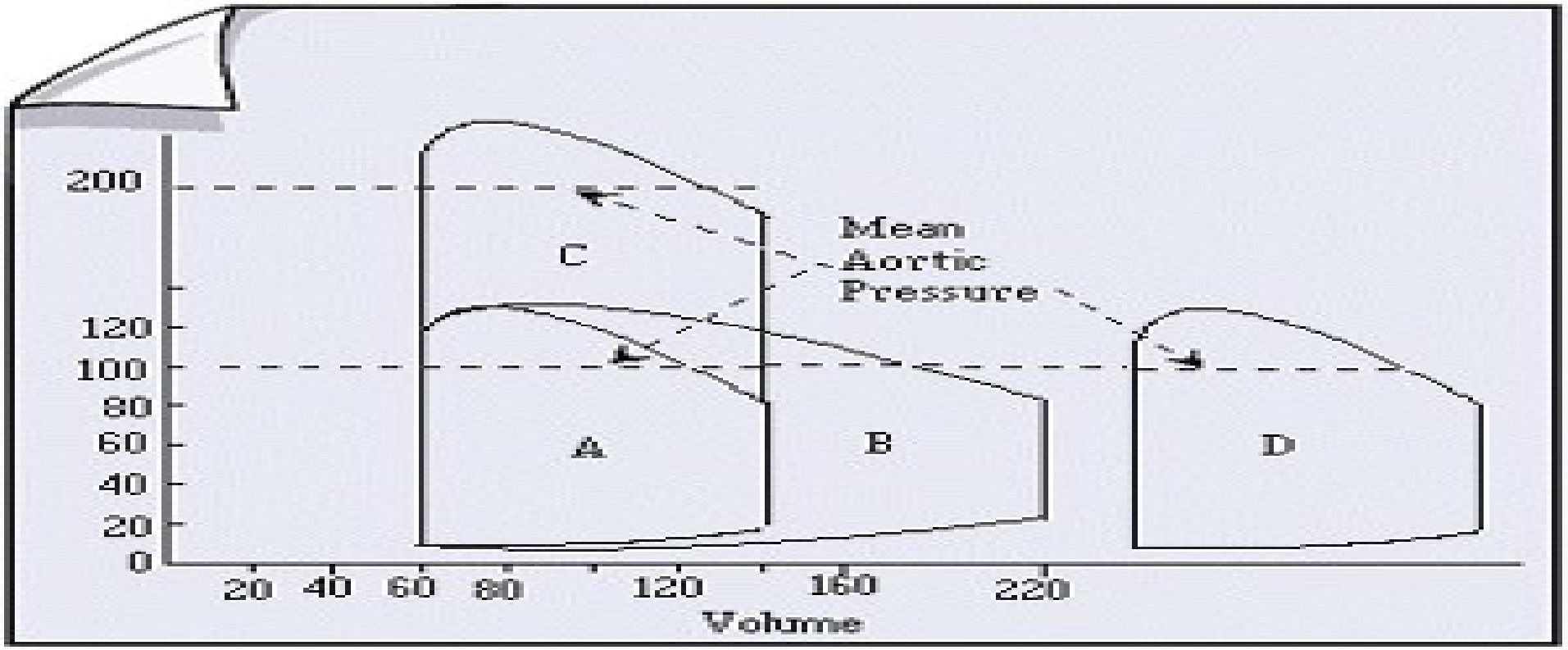
Cardiac Work



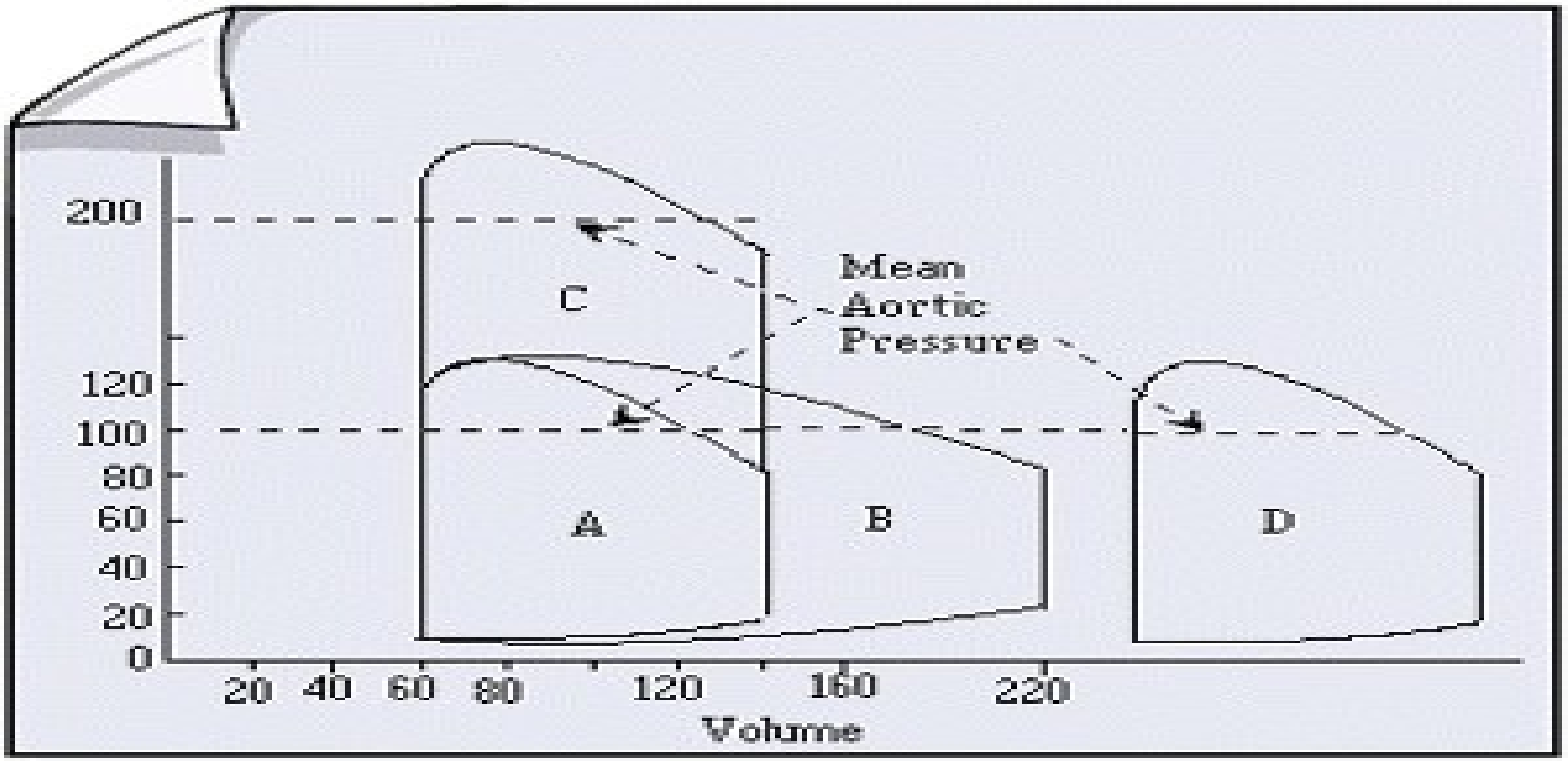
- *The area enclosed by the loop is an approximation of the ventricular stroke work.*
- *SW = SV x MAP.*
- *LVSW = (140 ml - 60 ml) x 100 mmHg, or 8000 ergs;*
- *RVSW = (140ml-60ml) x 18 mmHg = 1440 ergs, or about one-sixth that of the left ventricle.*
- *It follows that since the stroke volume of the two ventricles is equal, the "work" is a function of the afterload, which is about five to six times higher in the aorta than in the pulmonary artery.*

Pressure-volume loops are less than perfect in estimating cardiac work because

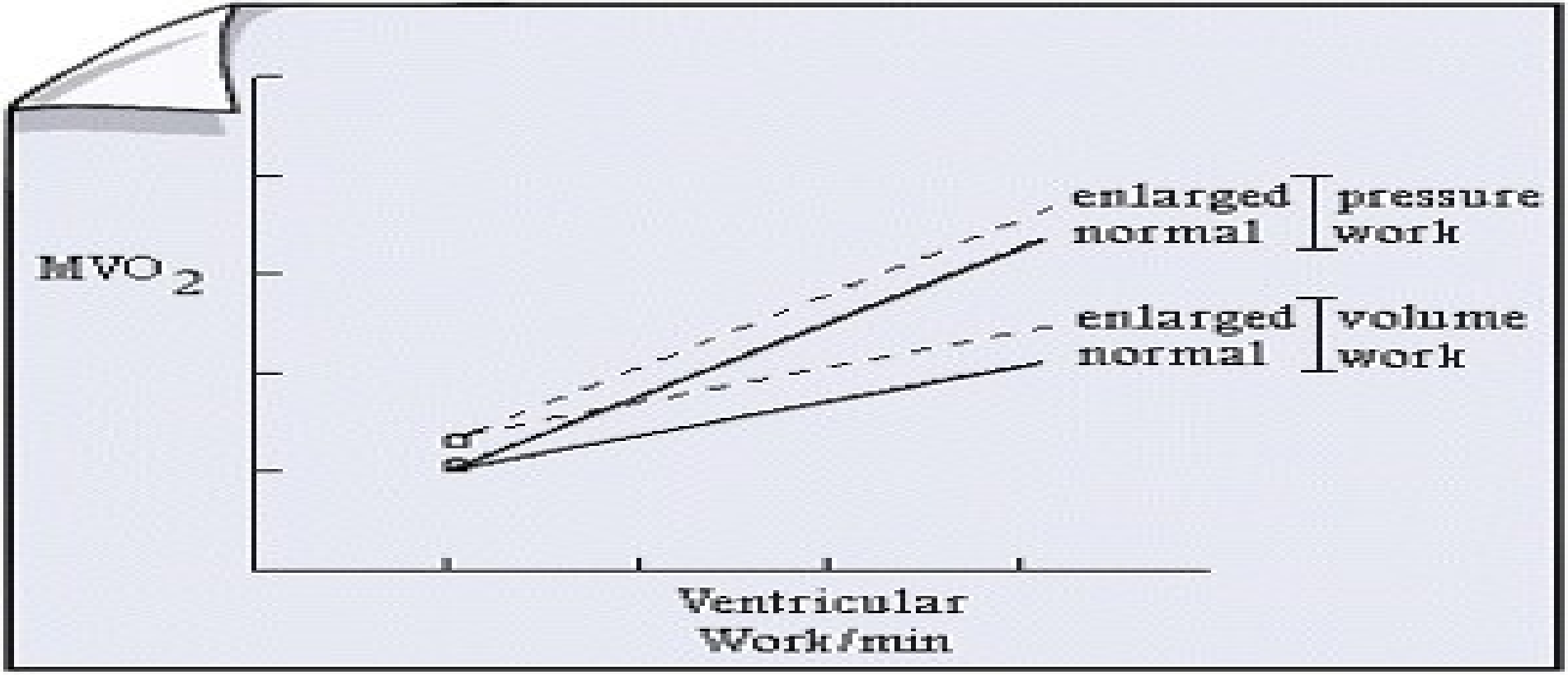
1. *They fail to take into account HR*
2. *They fail to account for changes in resting wall tension, which may also alter oxygen use.*



- *The area of loops A and D are equal, as are afterload and SV. Stroke work (SW) is therefore the same. Determinations of oxygen consumption reveal that heart D will consume more O2 than heart A because it is operating at a larger size (end diastolic volume is increased) and resting wall tension is higher (EDV=130 ml vs >220 ml).*

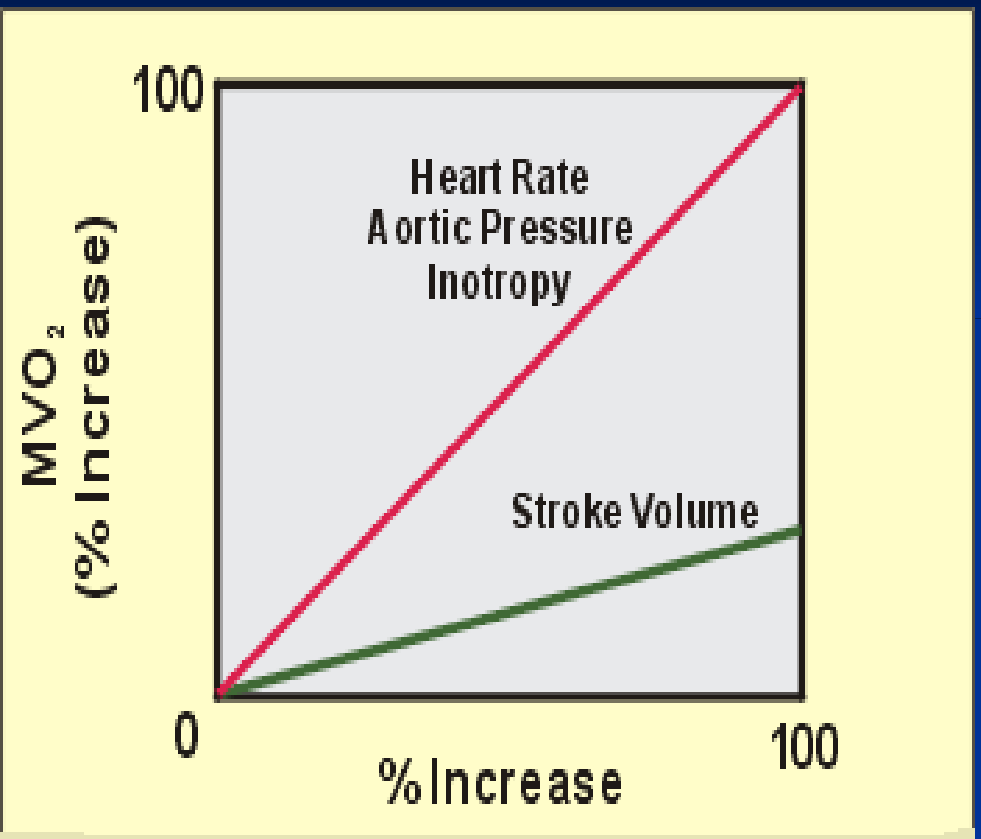


- *The area of loop B is exactly two times that of loop A and the stroke work of B is also two times that of A (SWA=100mmHg X 80ml=8000; SWB=100mmHg X 160ml=16000). Oxygen consumption (MVO2) is approximately doubled. In C the SV is 80ml and the afterload is increased by two times (to 200mmHg). The SW = 80 X 200=16000, or the same SW as in B. There is a MUCH greater increase in MVO2, however.*



- *a) the energy requirement (MVO2) to do pressure work is much greater than is the requirement needed to do an equal amount of volume work, and*
- *b) large hearts require more oxygen than smaller hearts to do the same amount of work.*

Determinants of MV02



- *Tension*
- *Heart rate*
  - *Preload*
  - *Afterload*
  - *Contractility*

Aortic stenosis vs regurgitation

- *Pressure overload vs volume overload*
- *MVO2 aortic stenosis >>> aortic incompetence*
- *Therefore, **anginal pain** is more likely to occur in **pressure overload** conditions.*