Hemodynamic Monitoring

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Edited by : Haya khader

- Hemodynamic monitoring plays a fundamental part in the initial assessment and the subsequent guidance of the treatment of critically ill patients suspected of or suffering from circulatory shock

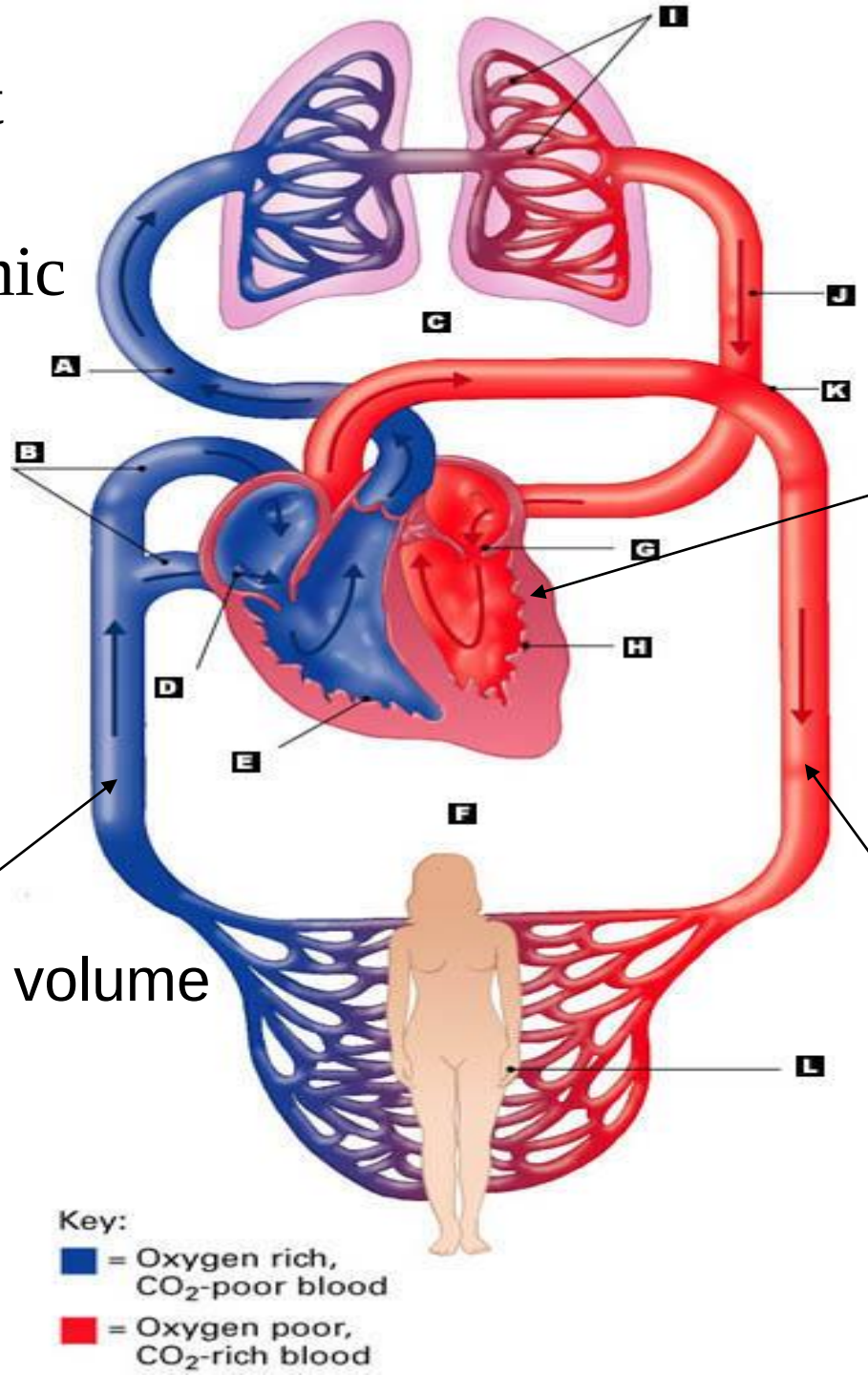
- About one-third of patients in the ICU eventually experience circulatory shock, and patients with circulatory shock have increased risks of multiorgan failure, long-term morbidity, and mortality

Introductions

- Hemodynamics is concerned with the forces generated by the heart and the resulting motion of blood through the cardiovascular system
- Hemodynamic monitoring is the intermittent or continuous observation of physiological parameters pertaining to the circulatory system with a view to early detection of need for therapeutic interventions

4 factors that affecting the haemodynamic conditions

preload contractility and afterload



Myocardial contraction and heart rate

Vasoactivity

Intravascular volume

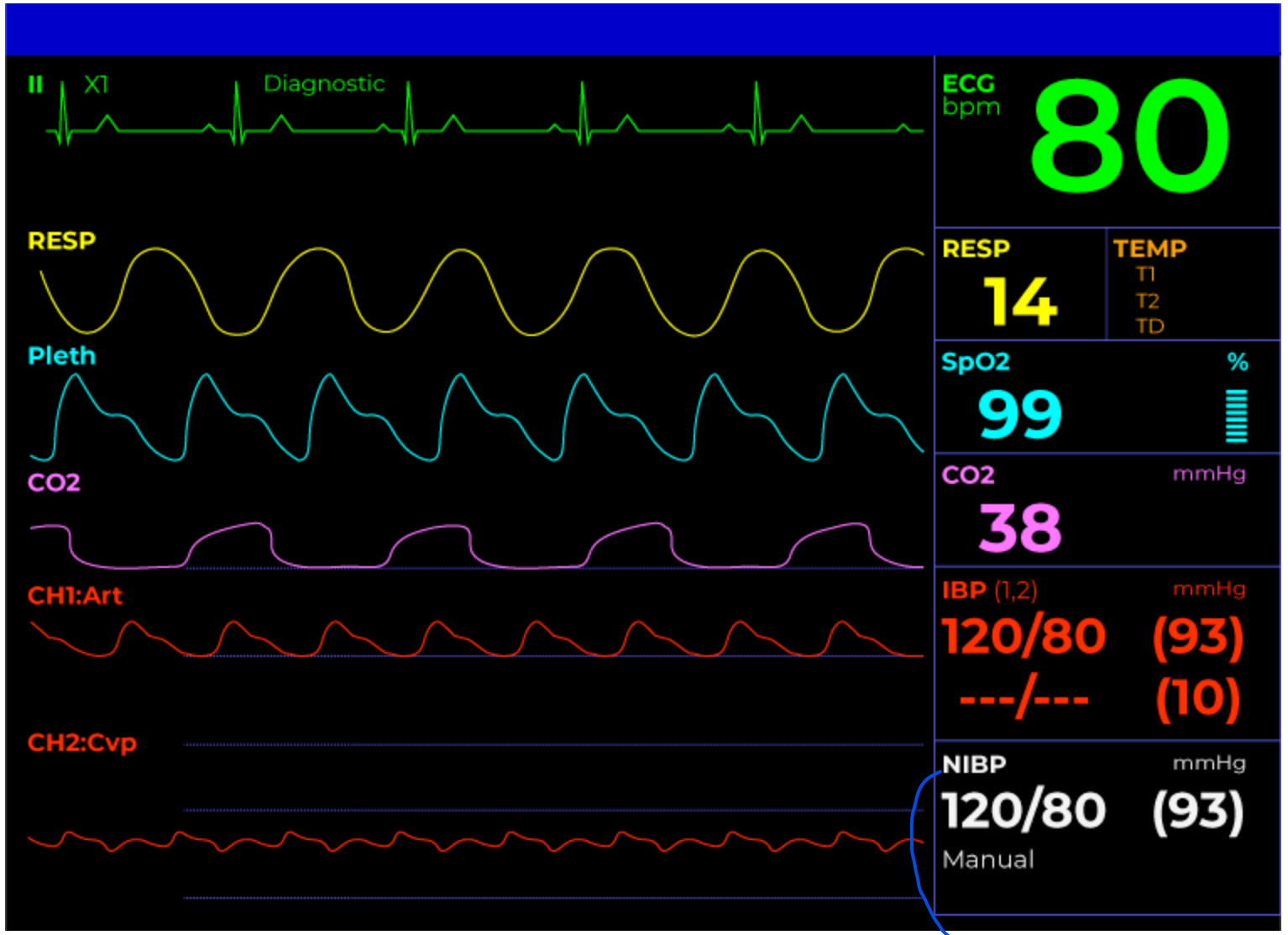
Clinical examination

- Clinical examination of the cardiovascular system can be used to assess perfusion or to estimate cardiac output (CO), and its daily application in critically ill patients makes it the first step of hemodynamic monitoring in suspected circulatory shock

- History
- Physical Examination

IN ICU

1. **Temperature** core temperature / surface temperature (hight temperature --> infection or hypodynamic circulation) (low temperature --> cold patient or severe vasoconstrictive or septic) so we have to measure the temperature as diagnostic tool and maintain the homeostasis of the body (by regulate the enzymes and proteins which work in normal temperature or hypothermia (like coagulation cascade)????
2. **Pulse** quantity of the pulse (large , small volume ....)
3. **Blood pressure**
4. **Respiratory rate** number , pattern (shallow , rapid ...)
5. **Pain**
6. **Oxygen saturation (SpO2)**
7. **Level of consciousness**
8. **Urine output**
9. **ECG**
10. **Lactate**



continuous ECG monitoring

RR AND TEMPERATURE

pulse oxymeter and saturated blood

in tidal CO2(measure CO2 in end inspiration

arterial line continuous

central venous pressure

non-invasive blood pressure --> from the cough

if the HR <60 --> the CO will decrease
if the HR > 120 --> the CO will decrease also (the filling time will decrease)

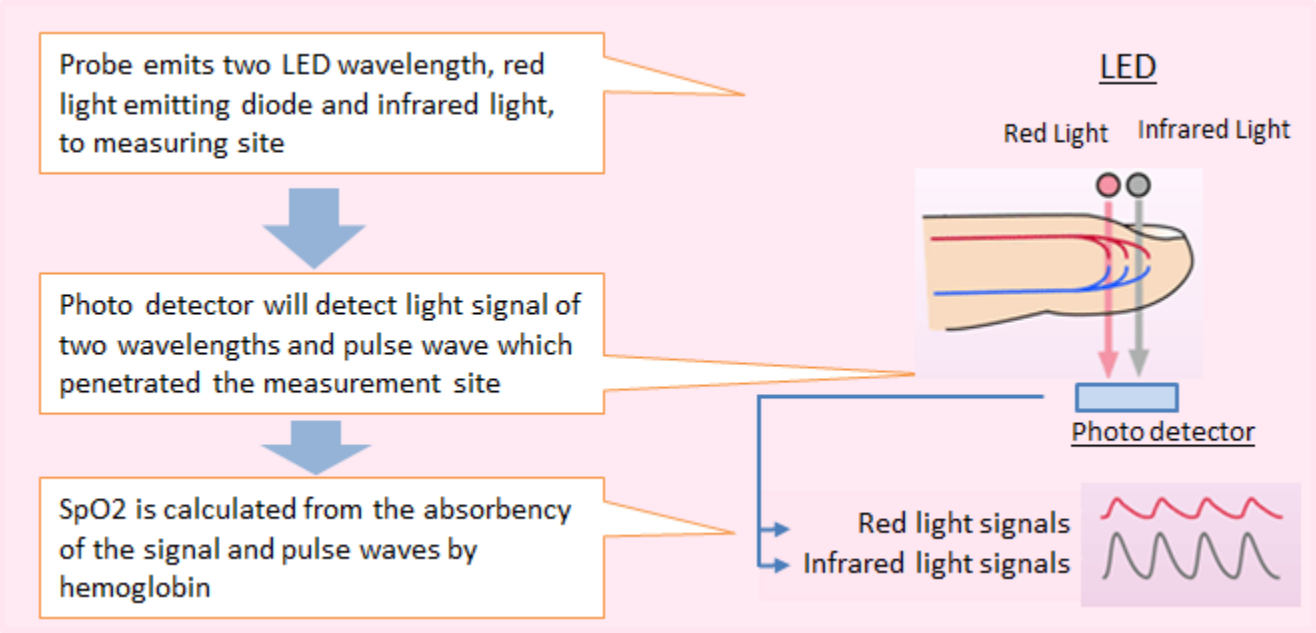
- HR 60-100 CO = stroke volume * HR
- Sinus Rhythm every P wave followed by QRS complex within 200 millisecond with equal RR interval
- Ischemic changes ST elevation / depression
  T wave inversion / elongation
- Heart Block first ,second , third block
- Arrhythmia supra ventricular , ventricular ...
- Electrolyte imbalance

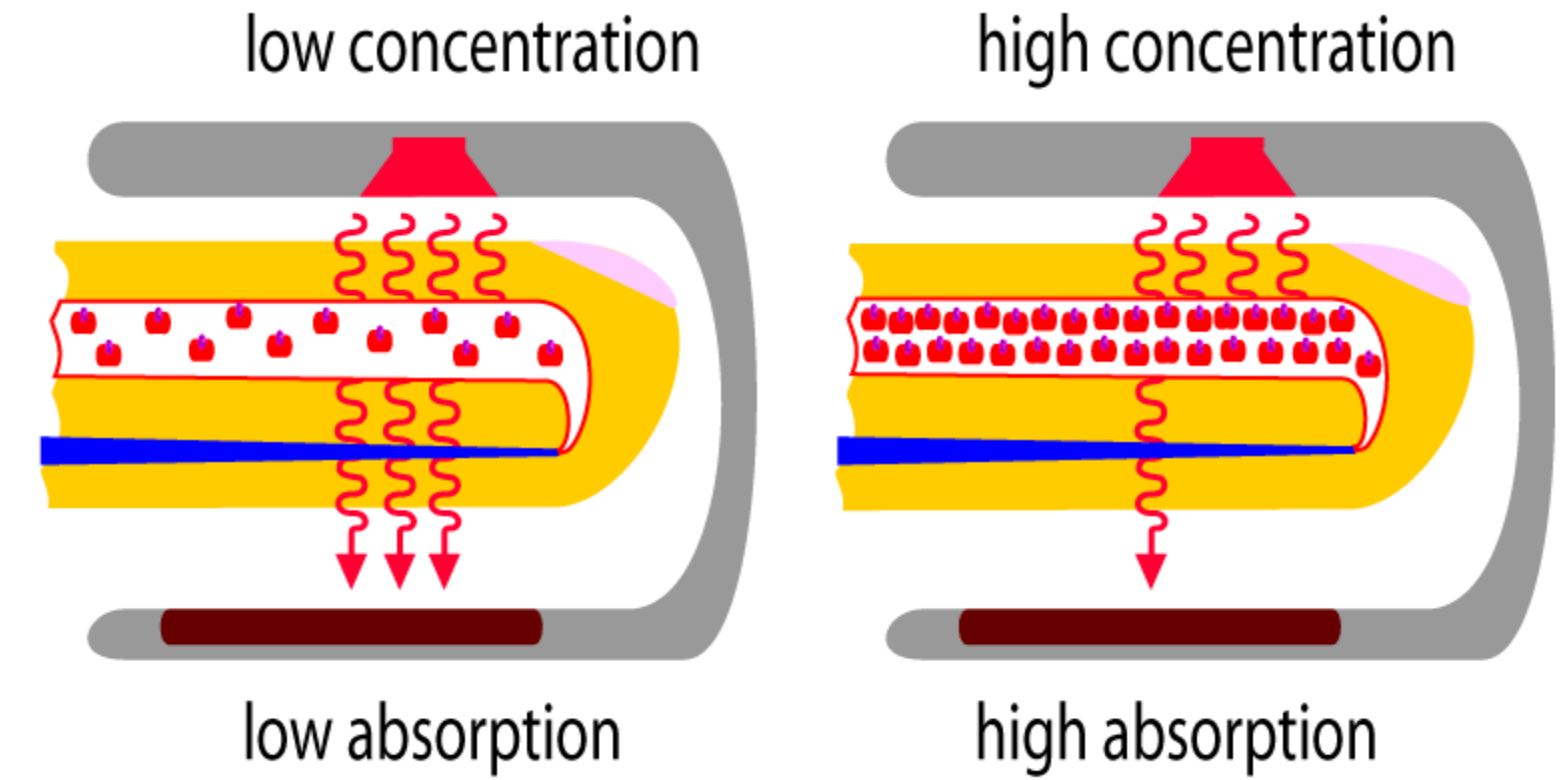
  beat T wave --> sinal hyperkalemia ??

ECG

1lead --> give continuous information
if you want more information about ischemic changes you have to 12-15 leads

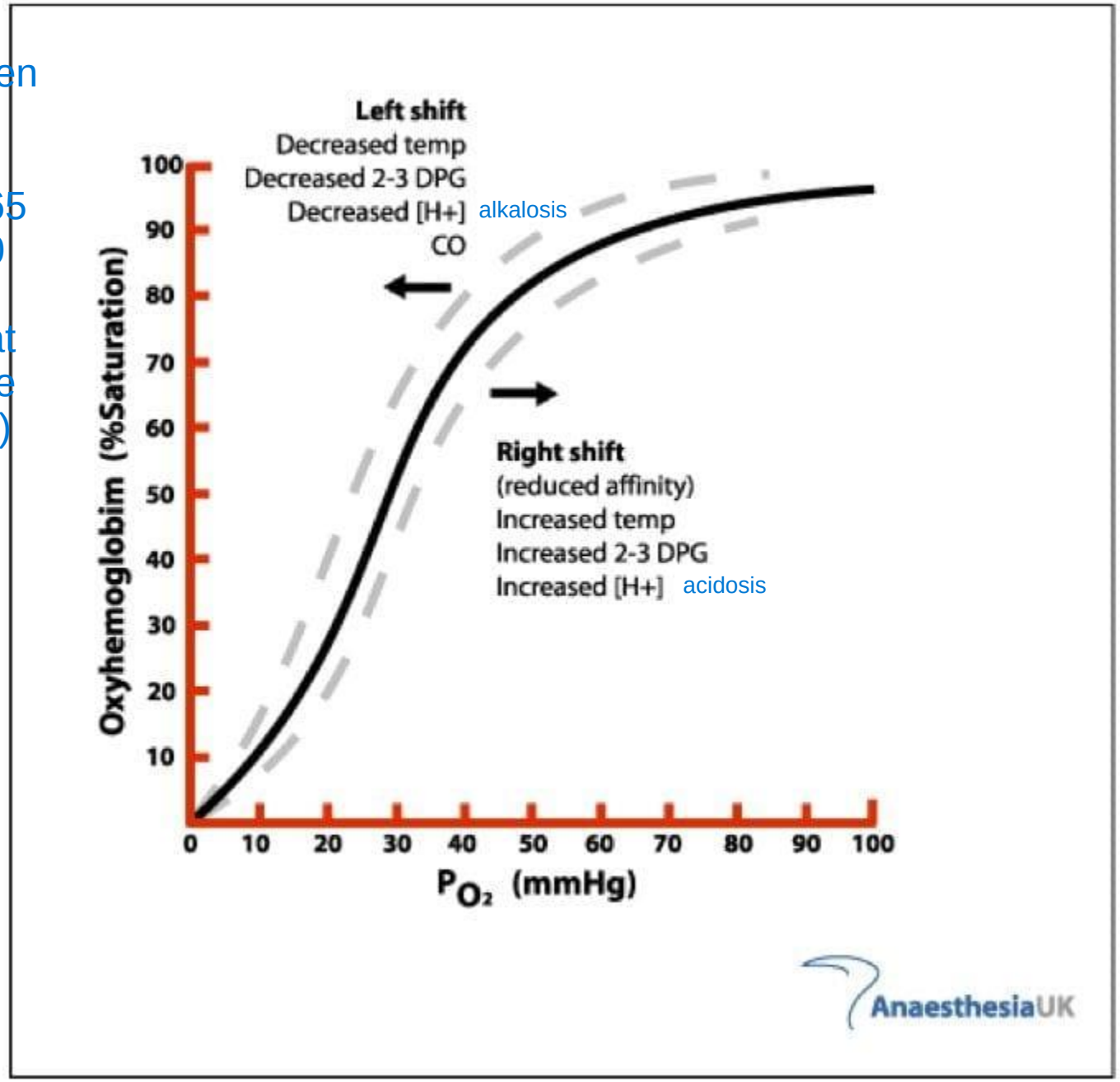
pulse oximeter --> we put probe (one side accept information and the other side send information) and give red light and measure how much the red light absorbed (the red blood take the red light so it measure the saturated hemoglobin




low concentration
high concentration
low absorption
high absorption
how equipment works .com

saturated-oxyhemoglobin :
-the relation between oxygen saturation and PO2 is sigmoidal (not linear)
saturation = 93 the PO2 =65
sat =70 PO2 = 40

as we go to the right the sat will remain near 100 but the PO2 increase (till 300-400)



PaO2 --> dissolved oxygen in serum (400-500 max)

the anesthesia done preoxygenation to elongate the plateaue till PO2 reach 300 to fill the serum with dissolved oxygen for 5-6 minutes

the PO2 need 5-6 min to decrease from 300--> 90
but if it reach 90 then it will decrease very very very quick

urine output : the minimal accepted UO --> 0.5ml/kg/h less than then --> oligouria then anuria

UO very sensitive for perfusion

lactate level -->
lactate arise from anaerobic metabolism (in hypoxic or low perfusion patient)
if the patient has liver problems --> increase the lactate level (the lactate metabolism happen in the liver)

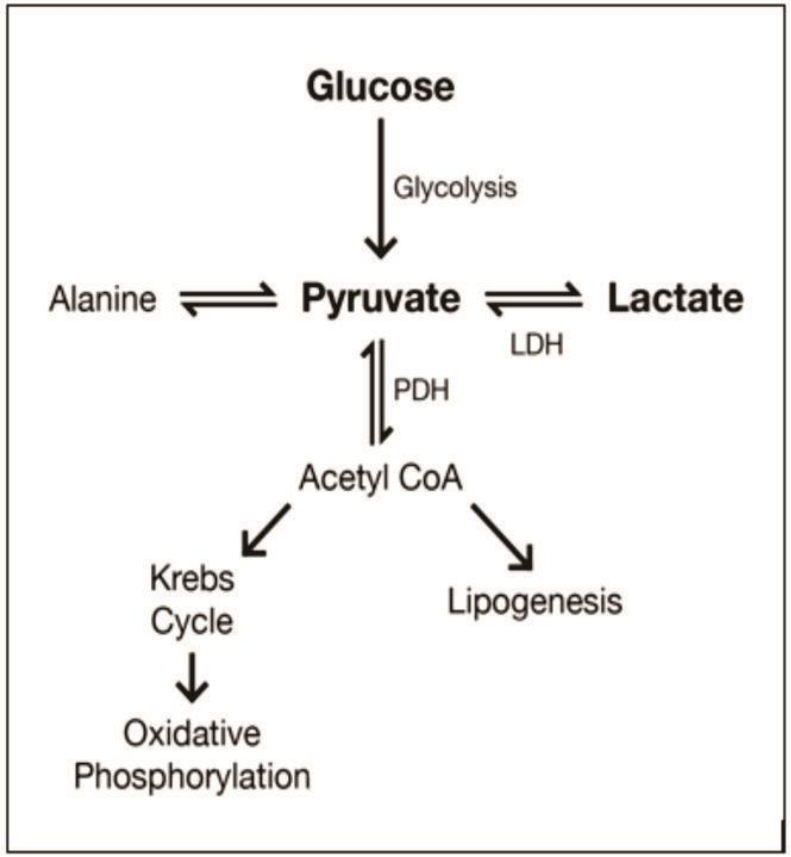


Figure 1: Pathway of pyruvate and lactate metabolism

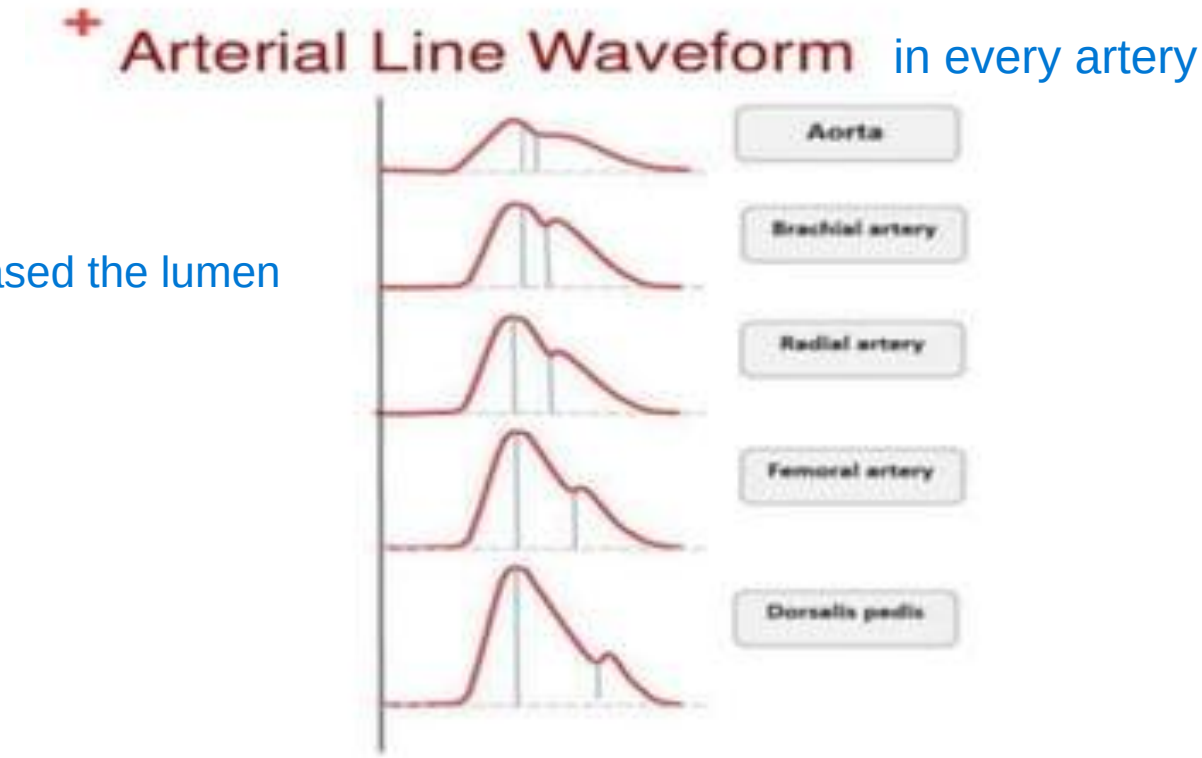
Table 2 : Diagnoses to Consider in Septic Patients with Elevated Lactate

Causes of Elevated Lactate	Consider
Hypovolaemic shock	Ensure volume resuscitation prior to or along with commencement of vasopressors
Cardiogenic shock	Consider cardiac dysfunction as a co-morbid cause of hyperlactataemia
Drugs	Consider metformin, NRTIs, linezolid increase the lactate level
Toxins	Consider the lactate gap (ethylene glycol poisoning), cyanide poisoning, paracetamol overdose
Adrenaline / salbutamol use	Interferes with interpretation of lactate levels as markers of resuscitation
Co-existent hepatic dysfunction	Routinely measure liver function and measures of synthetic function.
Seizures	Consider as a differential diagnosis in patients with reduced conscious state
Thiamine deficiency	Consider thiamine replacement in all septic patients

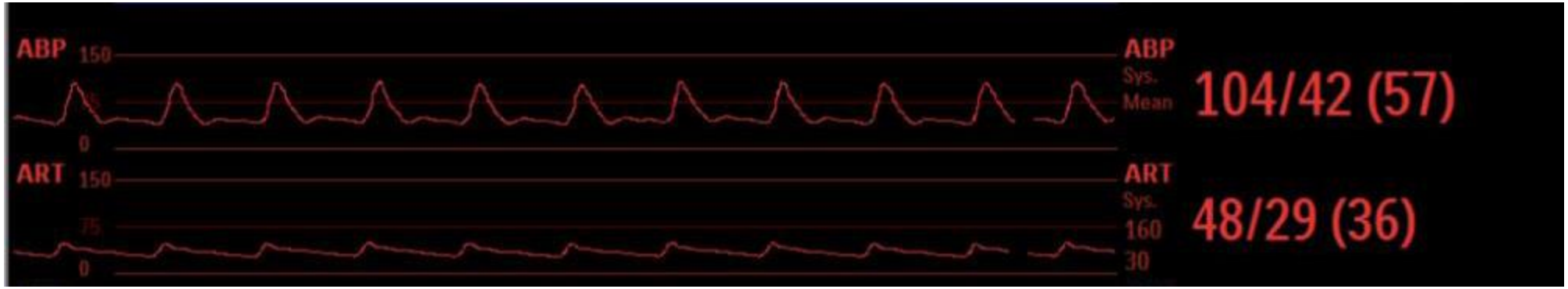
Old equipments

- Systolic
- Diastolic
- Pulse Pressure

as we go distally
the more narrow wave (due to decreased the lumen
more proximally (more wide)



in every artery



continuous arterial pressure(hace characteristic waves

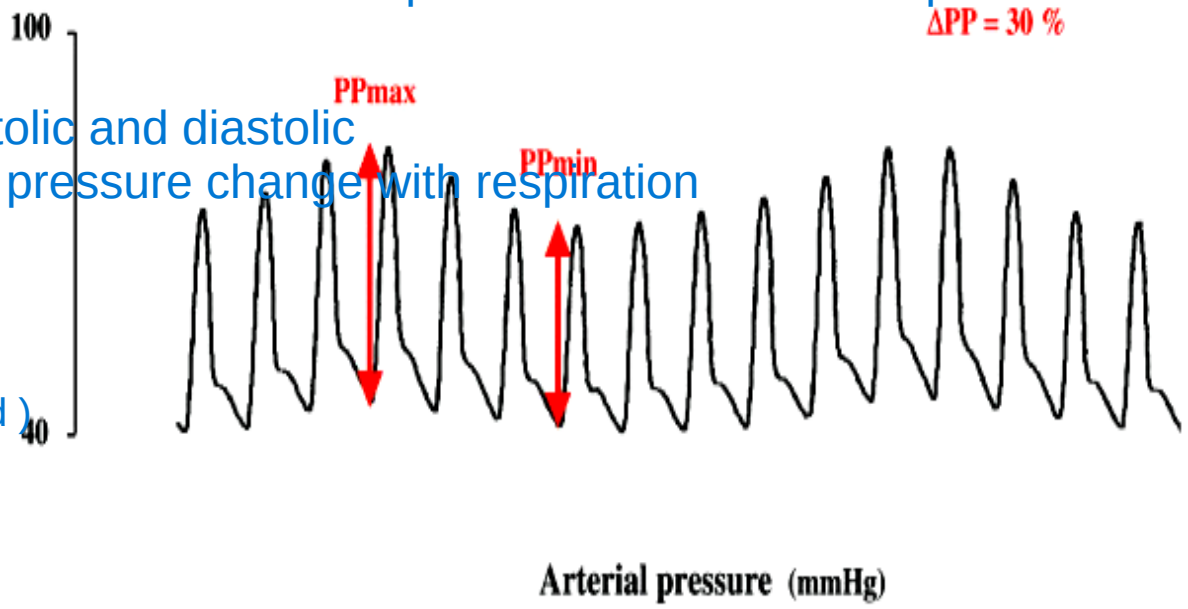
Old equipments

- Arterial line
  - Real time SBP, DBP, MAP
  - Pulse pressure variation (ΔPP)

systolic / diastolic blood pressure / mean arterial pressure

pulse pressure =the difference between the systolic and diastolic
pulse pressure variation --> howmuch the pulse pressure change with respiration

pulse pressure variability:
>=13 --> fluid responder (need fluid)
<13 --> fluid nonresponder (doesnt need fluid)



- ΔPP (%) = 100 × (PPmax - PPmin)/([PPmax + PPmin]/2)
- >= 13% (in septic pts,) discriminate between fluid responder and non respondaer (sensitivity 94%, specificity 96%)

Am J Respir Crit Care Med 2000, **162:**134-138

Arterial line

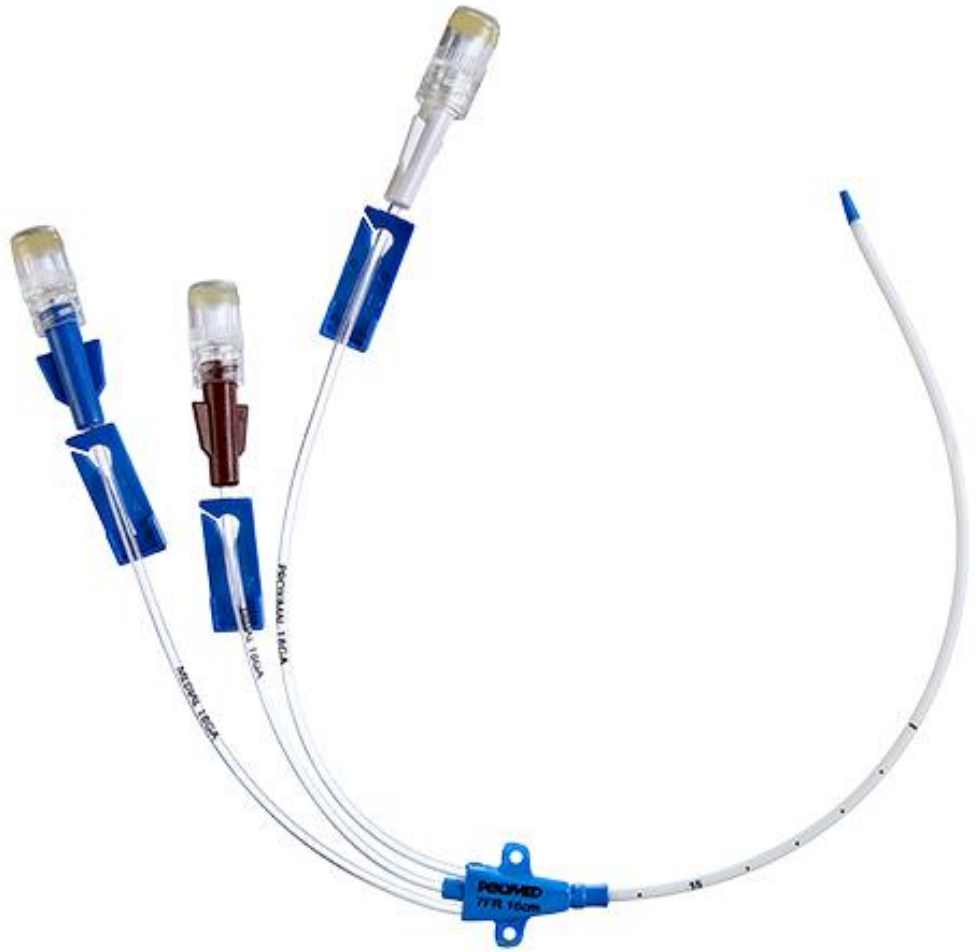
- Advantages
  - Easy setup
  - Real time BP monitoring
  - Beat to beat waveform display
  - Allow regular sampling of blood for lab tests
- Disadvantages
  - Invasive penetrate the artery (30% of radial artery undergo thrombosis after putting arterial line)
  - Risk of haematoma, distal ischemia, especially in femoral artery pseudoaneurysm formation and infection

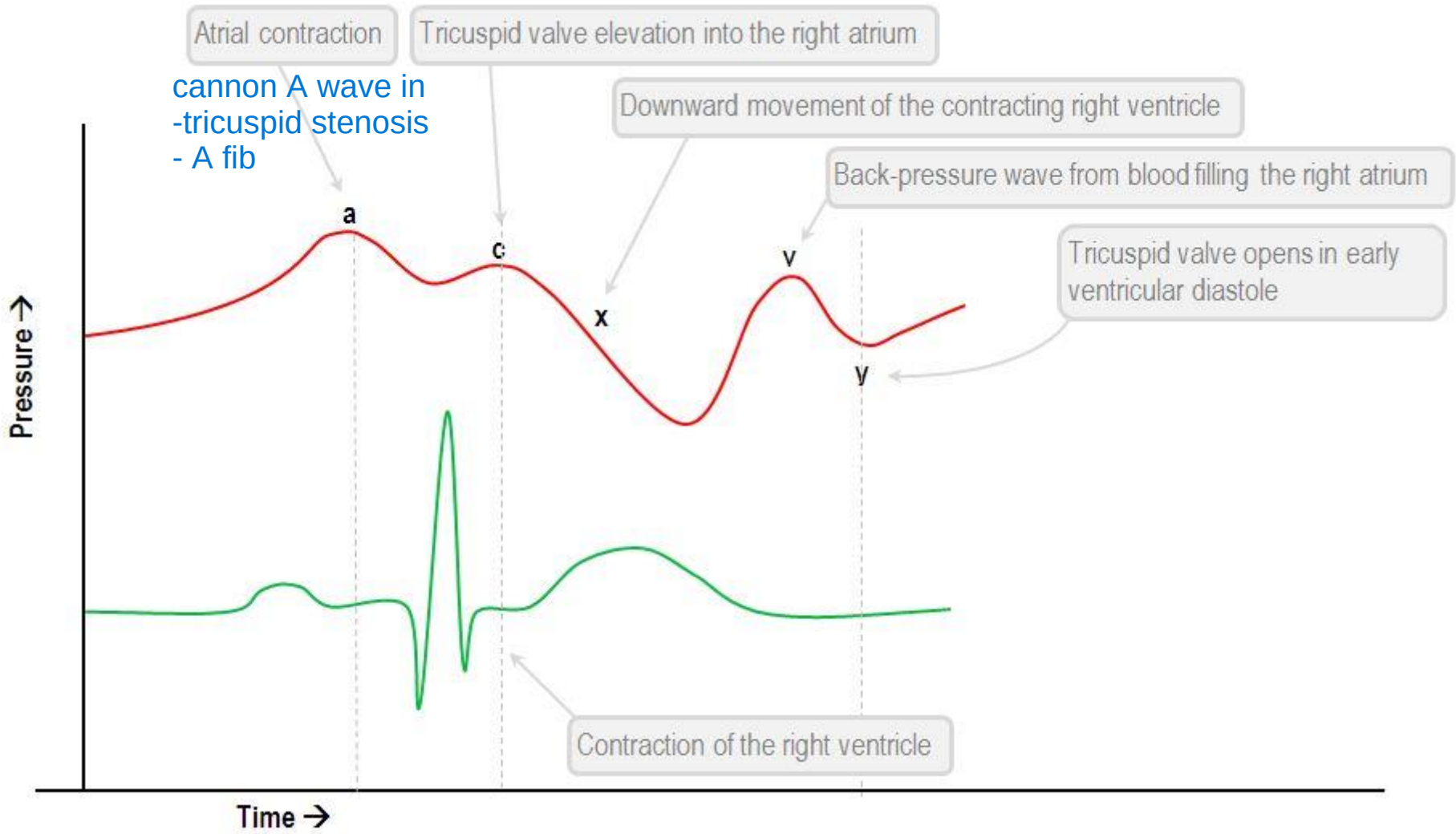
we use radial / ulnar (less complication) / brachial (more complications)

Old equipments

- Central venous catheter
  - Measurement of CVP, medications infusion, SvO2

central venous line :
-1-3-5 lumens (classicaly 3 lumens)
-put in internal jugular and measure central venous pressure and we can take sample to measure central venous saturation
we can use it to give some drugs (when we cant give it in peripheral veins) --> LIKE total parental nutrition / catecholamines / hard peripheral access




Atrial contraction
Tricuspid valve elevation into the right atrium
cannon A wave in
-tricuspid stenosis
- A fib
Downward movement of the contracting right ventricle
Back-pressure wave from blood filling the right atrium
Tricuspid valve opens in early ventricular diastole
a
c
x
v
y
Pressure →
Time →
Contraction of the right ventricle

Central venous catheter

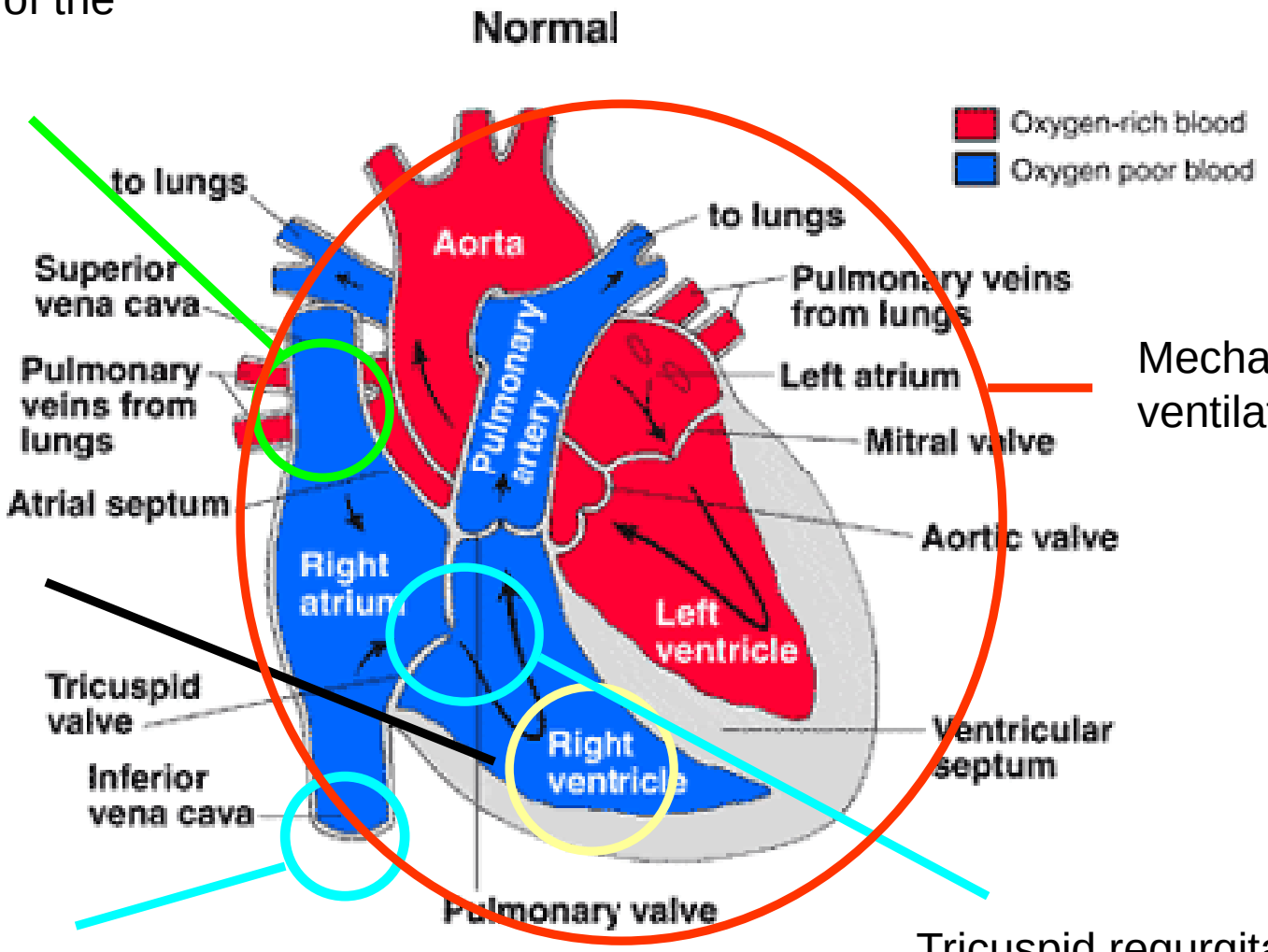
give an idea of preload of the right ventricle

- Advantages
  - Easy setup with US
  - Good for medications infusion
- Disadvantages
  - Cannot reflect actual RAP in most situations
  - Multiple complications
    - Infections, thrombosis, complications on insertion, vascular erosion and electrical shock

Limitation of CVP

central venous line :
put in the rt atrium
-represent the correlation of preload and ventricular function of rt ventricle

Obstruction of the great veins



Mechanical ventilation

also
the lumen of the Central vein line is abnormal
-lung problem (COPD / ventilated) so intrathoracic pressure will affect the intraatrial pressure

Decrease right ventricular compliance

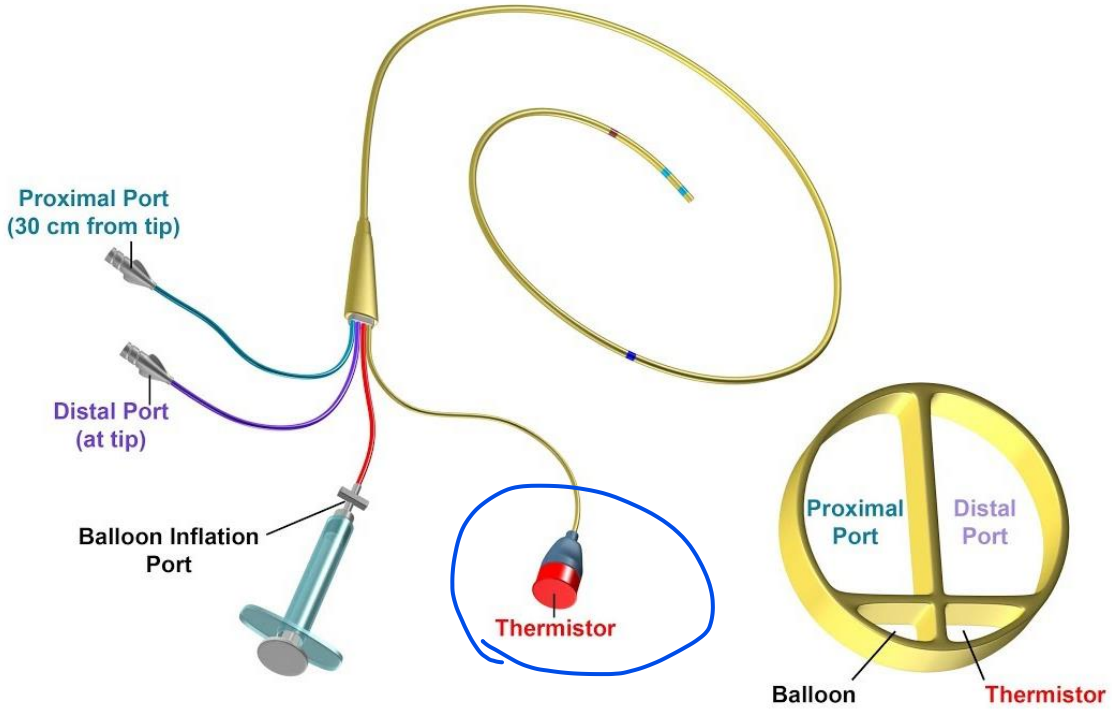
Tricuspid regurgitation

Systemic venoconstriction

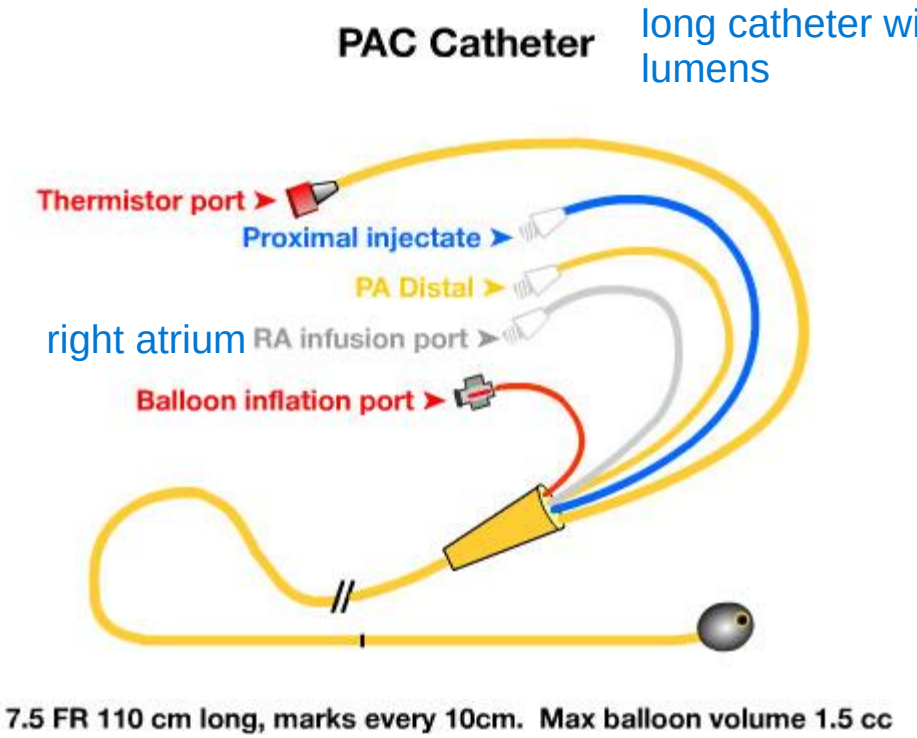
in the large vein so dont measure the real filling of the right atrium

PAC

pulmonary artery catheter / swan-gaz
used for hemodynamic monitoring and obtain values called (measured (like right atrium pressure CVP / right ventricular pressure / pulmonary artery pressure) or calculated



long catheter with many lumens

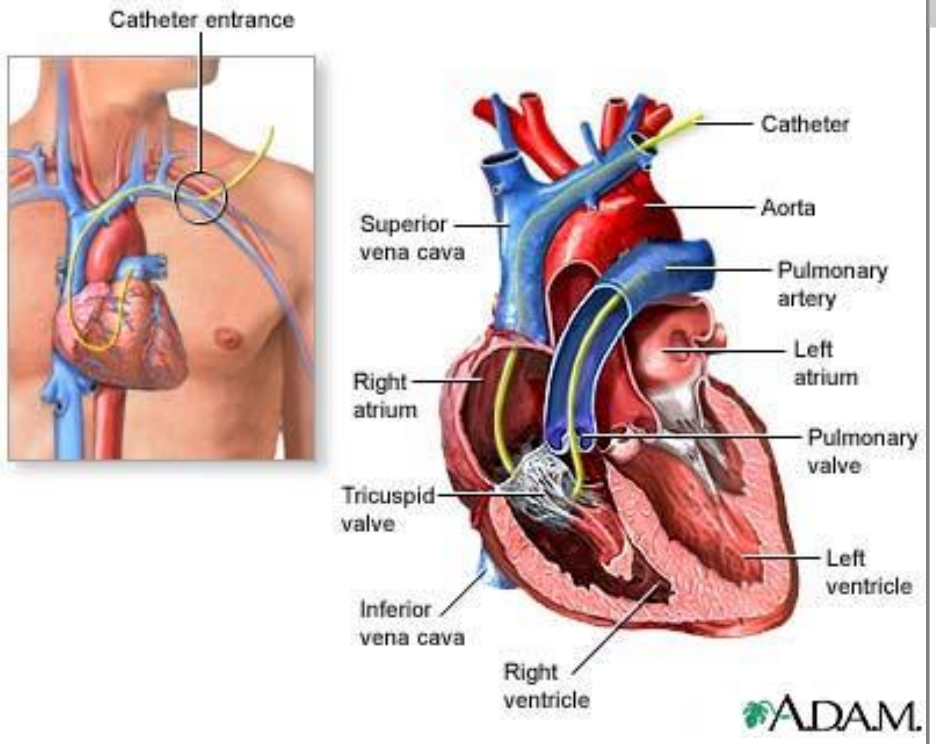


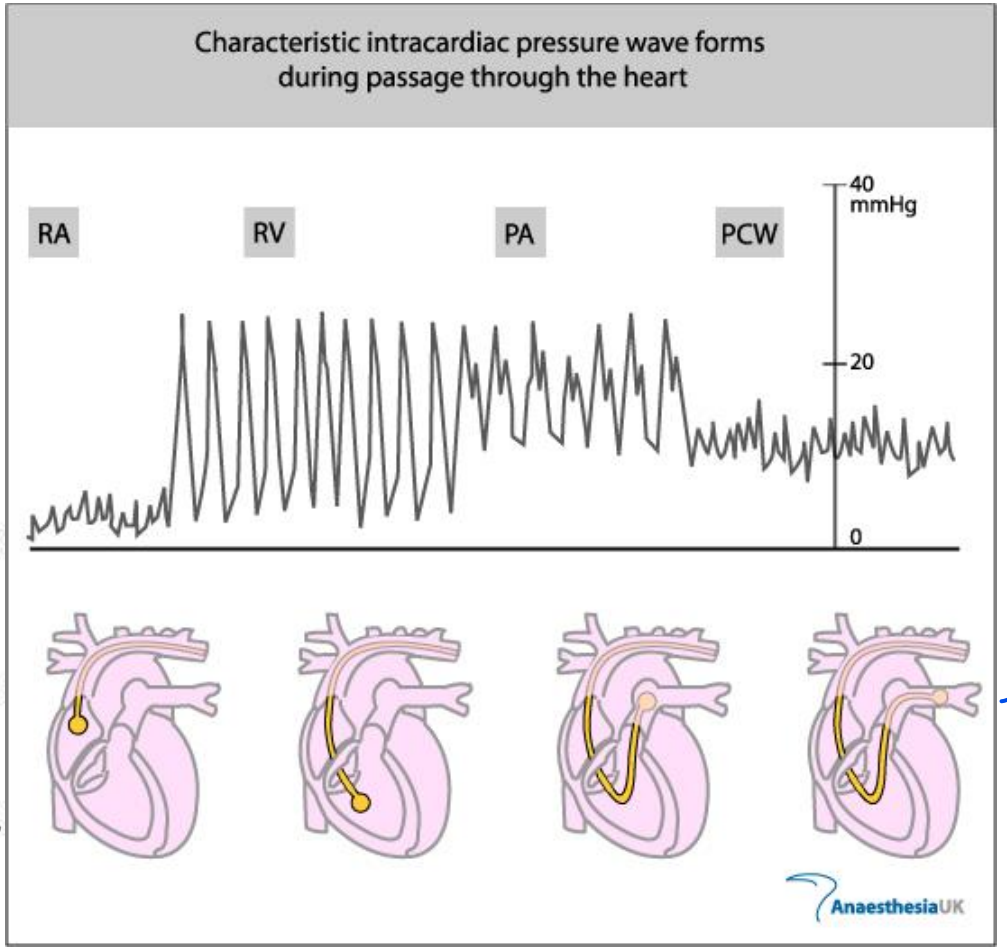
right atrium

Old equipment

put in the internal jugular --> right atrium (take CVP value) --> right ventricle (read the waves of the Rt ventricle) --> pulmonary artery (read PA waves) --> pulmonary capillaries wedge pressure PCW --> left atrium (pressure after static blood reflect the Lt atrial pressure so measure the pulmonary-capillary occlusive pressure which reflect left atrial pressure or the preload of the left ventricle)

- Pulmonary arterial catheter





this triangular called wedge

Indications for PAP monitoring

- Shock of all types
- Assessment of cardiovascular function and response to therapy
- Assessment of pulmonary status
- Assessment of fluid requirement
- Perioperative monitoring

Clinical applications of PAC

PAC can generate large numbers of haemodynamic variables

- Central venous pressure (CVP)
- Pulmonary arterial occlusion pressure (PAOP) =LAP = LVEDP
  left atrial pressure
  left ventricualr end diastolic pressure
- Cardiac output / cardiac index (CO / CI) ⇒ By thermodilution
- Stroke volume (SV)
- R ventricle ejection fraction/ end diatolic volume (RVEF / RVEDV)
- Systemic vascular resistance index (SVRI)
- Pulmonary vascular resistance index (PVRI)
- Oxygen delivery / uptake (DO2 / VO2)

the idea of theromdilution :
we have proximal and distal port so it give cold water in the proximal and then measure the water pressure in the distal port (temperature /time)
-if The CO was good /quick --> the time will be less and less change of temperature
-if the CO was slow (the temperature change will be high in low / slow time

Area under curve is inversely proportion to rate of blood flow in PA (= CO)

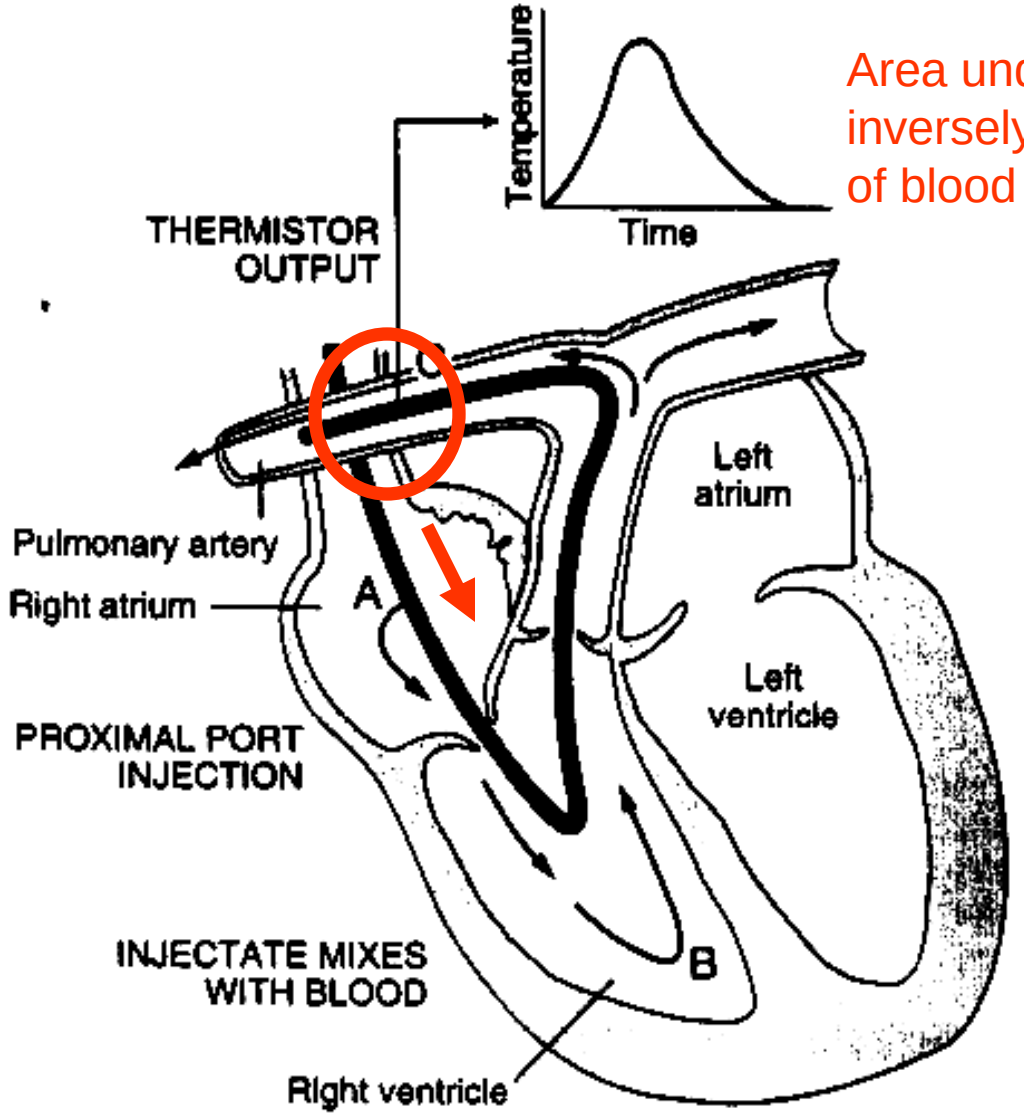


FIGURE 10–1. Schematic illustration of the thermodilution method utilizing the pulmonary artery catheter. The cold injectate is introduced into the right atrium **(A)** and mixes completely with blood within the right ventricular chamber **(B)**; the cooled blood flows into the pulmonary arterial circulation and past the thermistor bead near the tip of the pulmonary artery catheter **(C)**. (Adapted from Marino PL: Thermodilution cardiac output. In The ICU Book. Philadelphia: Lea & Febiger, 1991, p. 124.)

Patient with hypotension

Hypovolemia
- Low CVP
- Low CI cardiac index
- High SVRI systemic vascular resistance

⇒ Consider fluid challenge

Cardiogenic
- High CVP
- Low CI
- High SVRI

⇒ Consider inotopic / IABP

Vasogenic /distriputive shock
- Low CVP
- High CI
- Low SVRI

⇒ Consider vasopressor

Mixed Venous Saturation SvO2

blood extracted from the central line --> called central venous saturation
blood extracted from the pulmonary artery --> called mixed venous saturation
the difference between them only 5mmHg (the coronary sinus saturation 35mmHg and mixed with right atrial blood and become less

- Measured in pulmonary artery blood
- Marker of the balance between whole body O2 delivery (DO2) and O2 consumption (VO2)

in arterial

- $VO2 = DO2 * (SaO2 – SvO2)$

oxygen consumption = oxygen delivery *(arterial saturation - (central / mixed veins) venous saturation)

- In fact, DO2 determinate by CO, Hb and SaO2. Therefore, SvO2 affected by
  - CO
  - Hb
  - Arterial oxygen saturation
  - Tissue oxygen consumption

all affect the central venous saturation

Mixed Venous Saturation SvO2

- Normal SvO2 70-75%

Decreased SvO2
- increased consumption
    - pain, hyperthermia
- decreased delivery
    - low CO
    - anemia
    - hypoxia

Increased SvO2
- Increased delivery
    - high CO in distributaive shock / shunt
    - hyperbaric O2
- Low consumption
    - sedation
    - paralysis
    - cyanide toxicity

PAC

- Advantages
  - Provide lot of important haemodynamic parameters
  - Sampling site for SvO2
- Disadvantages
  - Costly
  - Invasive
  - Multiple complications (eg arrhythmia, catheter looping, balloon rupture, PA injury, pulmonary infarction etc) die from rupture of pulmonary artery because we iflate a balloon in the PA in the wedge and leave it inflated and cause infarction / PE
  - **Mortality not reduced and can be even higher**

Crit Care Med 2003;31: 2734-2741

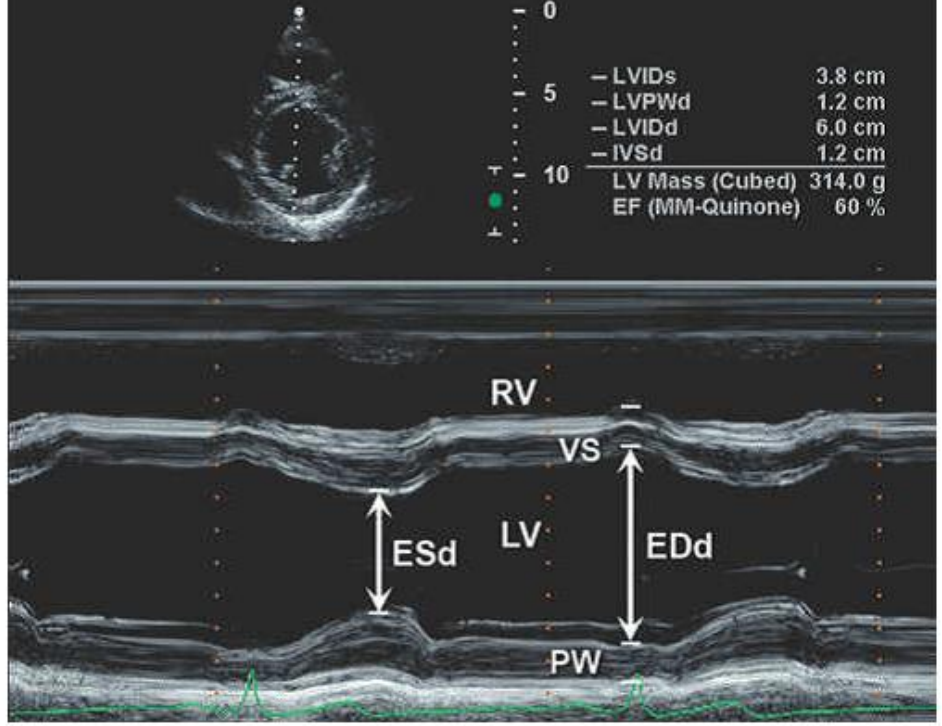
JAMA 1996;276 889-897

Advance in haemodynamic assessment

- Modification of old equipment
- Echocardiogram and esophageal doppler
- Pulse contour analysis and transpulmonary thermodilution
- Partial carbon dioxide rebreathing with application of Fick principle
- Electrical bioimpedance

Transthoracic echo

- Assessment of cardiac structure, ejection fraction and cardiac output + cardiac function
- Based on 2D and doppler flow technique



we can use it to measure
end systolic diameter
end diastoilc diameters
ejection fraction

Figure 7-1 Two-dimensionalâ€"guided M-mode echocardiogram of the left ventricle (*LV*) at the papillary muscle level. The LV end-diastolic internal dimension (*EDd*) measured at the onset of QRS is 60 mm, and the LV end-systolic internal dimension (*ESd*) is 38 mm. Therefore,

$$\text{LV ejection fraction (LVEF)} = \frac{60^2 - 38^2}{60^2} \times 100$$
$$= 60\% \text{ (uncorrected)}$$

If apical contractility is normal,

$$\text{Corrected LVEF} = 60\% + [(100 - 60) \times 15\%]$$
$$= 60\% + 6\%$$
$$= 66\%$$

LV mass is also calculated from LV dimensions, posterior wall (*PW*) thickness, and ventricular septal (*VS*) thickness. *RV*, right ventricle.

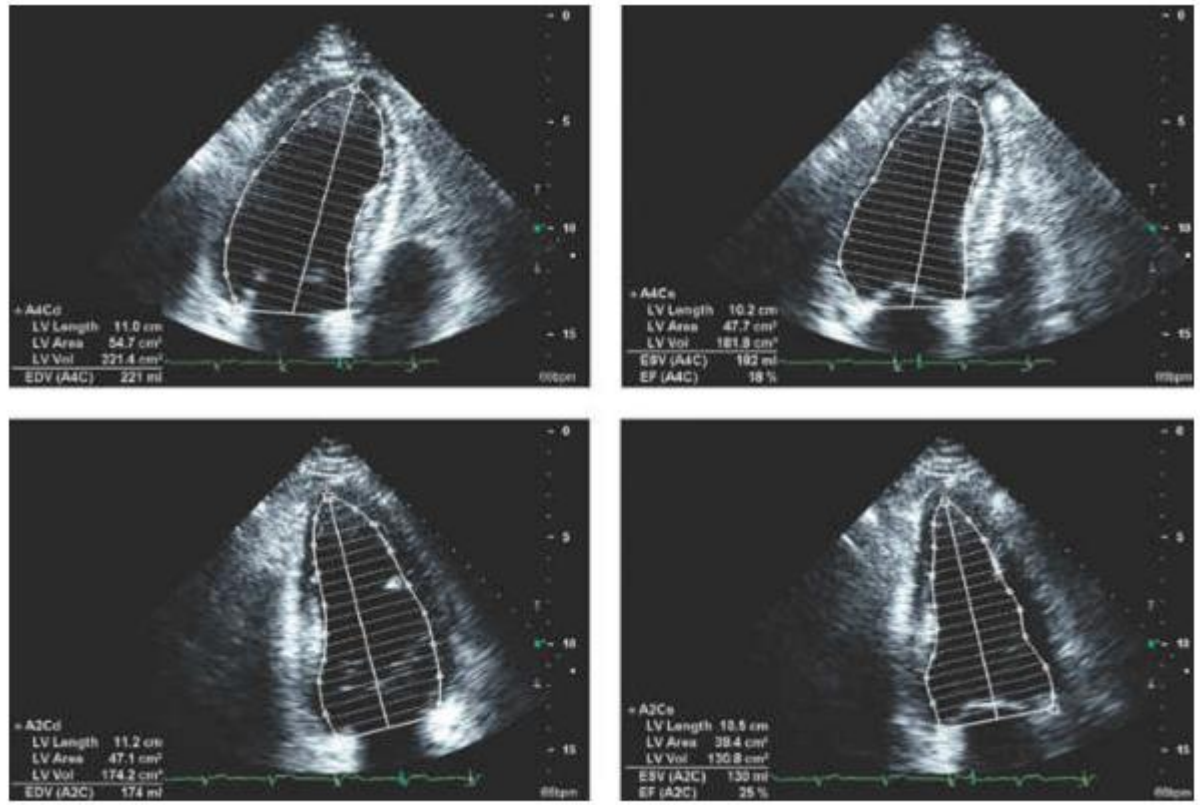


Figure 7-10 Still frames of two orthogonal views (apical four-chamber [*top*] and apical two-chamber [*bottom*] views) to calculate the left ventricular (*LV*) volume and ejection fraction using a modified Simpson method. End-diastolic (*EDV*) and end-systolic (*ESV*) frames illustrate 20 cylinders (disks) of equal height. When the endocardial border and long axis (vertical line to the short-axis lines) are identified, a fixed number of cylinders are created, and the volumes of the cylinders are summed to estimate ventricular volume (*Vol*). *EF*, ejection fraction.

sepson formula :
--> measure the volume in systole / diastole and ejection fraction

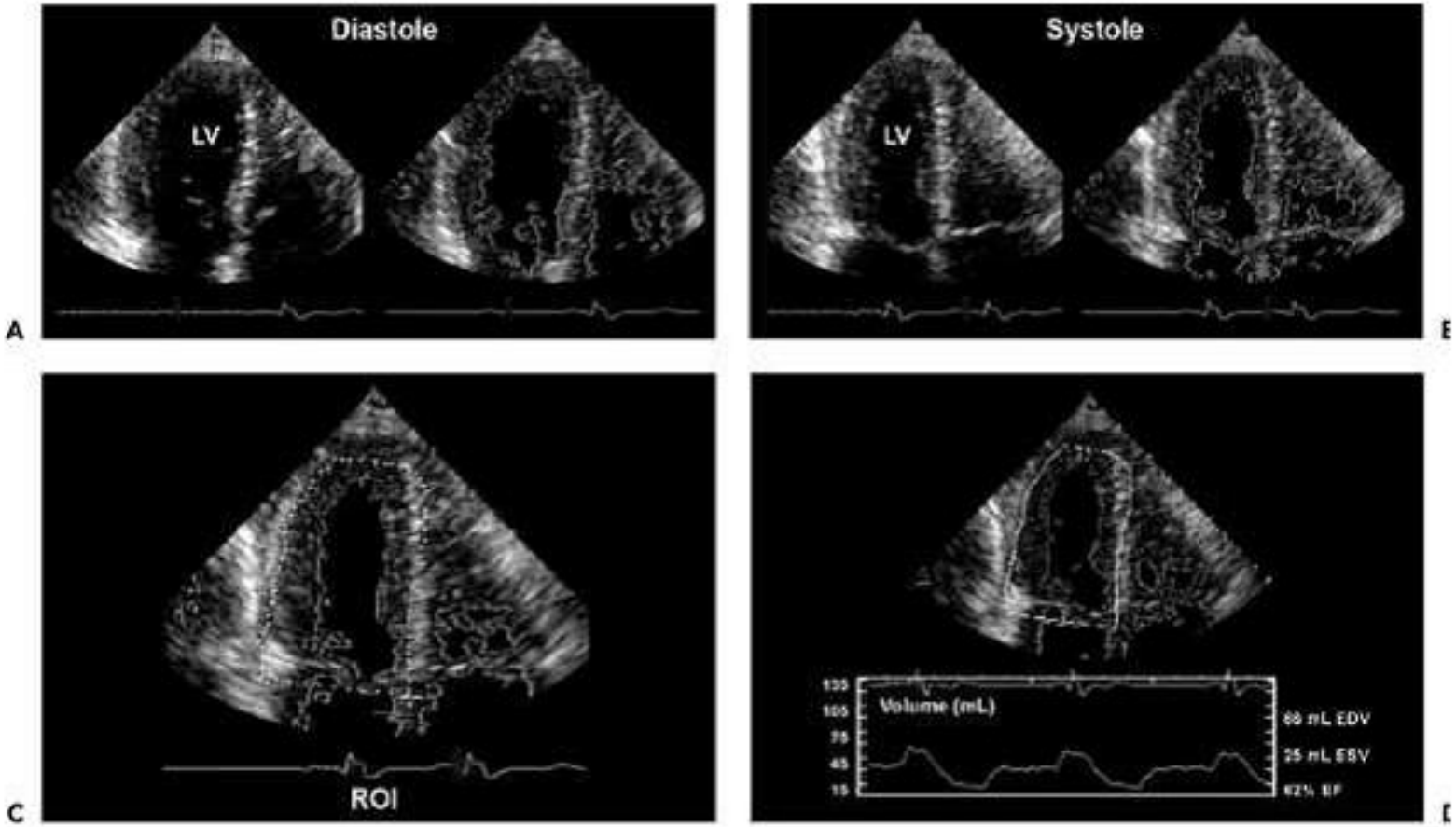
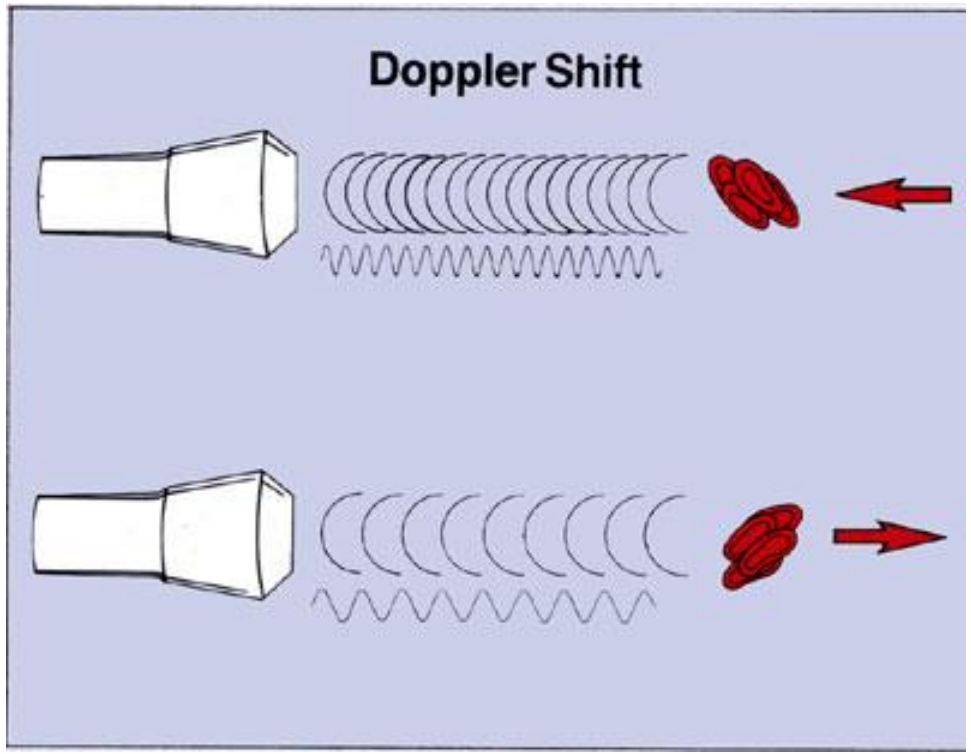


Figure 7-12 A and **B:** Ultrasound backscatter differs markedly between the myocardium and intracavitary blood pool. Therefore, the endocardial border is defined where a greater- than- preestablished threshold difference in backscatter is identified. The borders are identified and dotted. Connecting the endocardial dots creates an endocardial border in real time throughout the cardiac cycle. *LV*, left ventricle. **C:** An area of interest is identified by selecting the region. Because we are interested in the volumetric change of the LV, the LV cavity was chosen as the region of interest (*ROI*). **D:** From the real-time automatic border detection of the LV endocardium, LV volume changes are instantaneously determined to provide end-diastolic volume (*EDV*), end-systolic volume (*ESV*), and ejection fraction (*EF*).

Echo doppler ultrasound

we put doppler in the tip of the probe and it gives US waves and measure the velocity of blood then we can measure the flow rate

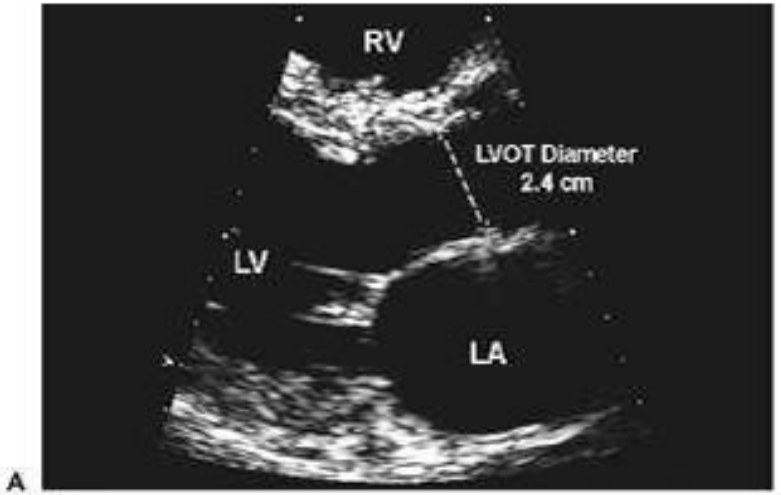
- Measure blood flow velocity in heart and great vessels
- Based on Doppler effect ⇒ “ Sound freq. increases as a sound source moves toward the observer and decreases as the soure moves away”

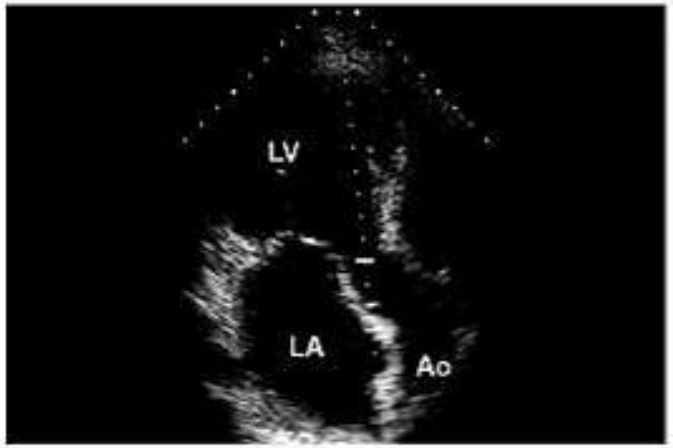


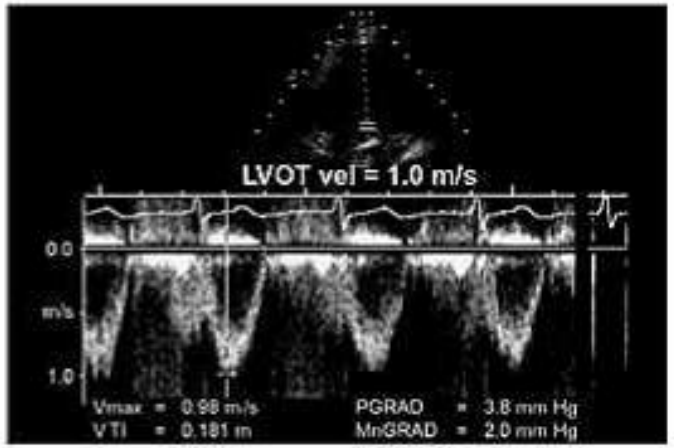
For transthoracic echo

- Haemodynamic assessment for SV and CO
  - Flow rate = CSA x flow velocity
    cross-section surface area
  - Because flow velocity varies during ejection, individual velocities of the doppler spectrum need to be summed
  - Sum of velocities called velocity time integral (VTI)
  - SV = CSA x VTI
  - CSA =(LVOT Diameter /2)^2 * π
  - Therefore SV = D^2 * 0.785 * VTI
  - CO = SV * HR

we can measure the area of aortic / mitral valve







$$\text{LVOT area (cm}^2\text{)} = \left(\frac{D}{2}\right)^2 \times \pi$$

$$= D^2 \times 0.785$$

$$\text{SV(mL)} = \text{Area (cm}^2\text{)} \times \text{TVI}$$

$$= D^2 \times 0.785 \times \text{TVI}$$

$$= 4.5\ \text{cm}^2 \times 18\ \text{cm} = 81\ \text{mL}$$

Transthoracic echo

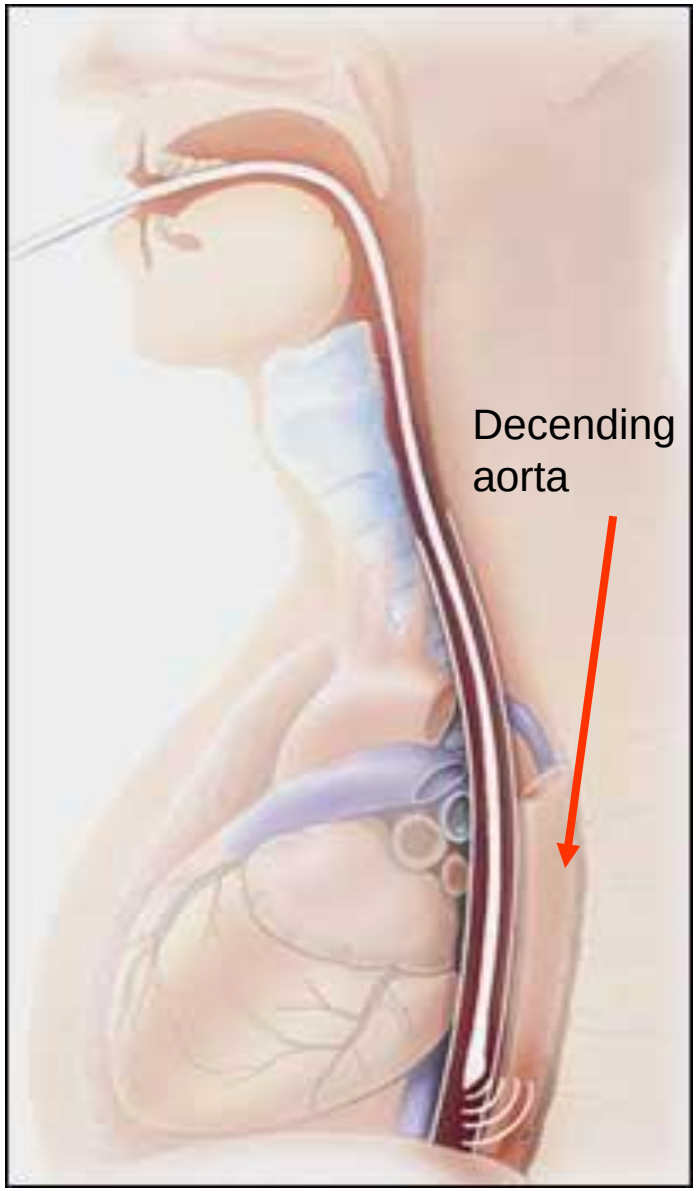
- Advantages
  - Fast to perform
  - Non invasive
  - Can assess valvular structure and myocardial function
  - No added equipment needed
- Disadvantages
  - Difficult to get good view (esp whose on ventilator / obese)
  - Cannot provide continuous monitoring

Transesophageal echo

we put the probe in the esophagus so we are nearer to the heart so the image will be better

- CO assessment by Simpson or doppler flow technique as mentioned before
- Better view and more accurate than TTE
- Time consuming and require a high level of operator skills and knowledge

Esophageal aortic doppler US



- Doppler assessment of decending aortic flow
- CO determinate by measuring aortic blood flow and aortic CSA
- Assuming a constant partition between caudal and cephalic blood supply areas
- CSA obtain either from nomograms or by M-mode US
- Probe is smaller than that for TEE
- Correlate well with CO measured by thermodilution

Crit Care Med 1998 Dec;26(12):2066-72

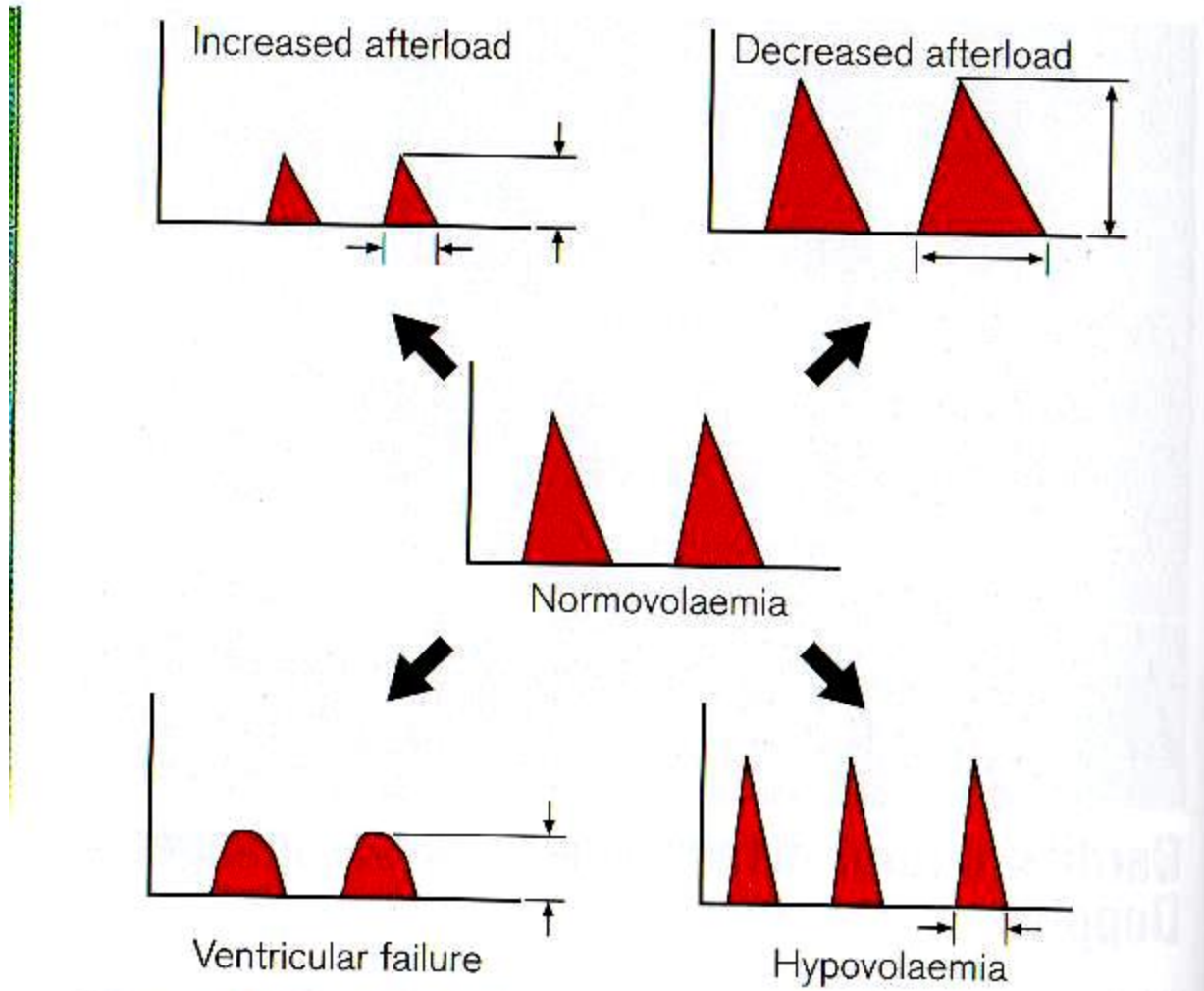


Figure 18.8 Predominant changes observed in the aortic waveform due to alterations in preload, afterload and contractility.

Esophageal aortic doppler US

- Advantages
  - Easy placement, minimal training needed (~ 12 cases)
  - provide continuous, real-time monitoring
  - Low incidence of iatrogenic complications
  - Minimal infective risk
- Disadvantages
  - High cost
  - Poor tolerance at awake patient, so for those intubated
  - Probe displacement can occur during prolonged monitoring and patient's turning
  - High interobserver variability when measuring changes in SV in response to fluid challenges

Pulse contour analysis

do analysis for the area under the pulse contour and measure the systemic vascular resistance and CO

- Arterial pressure waveform determinate by interaction of stroke volume and SVR

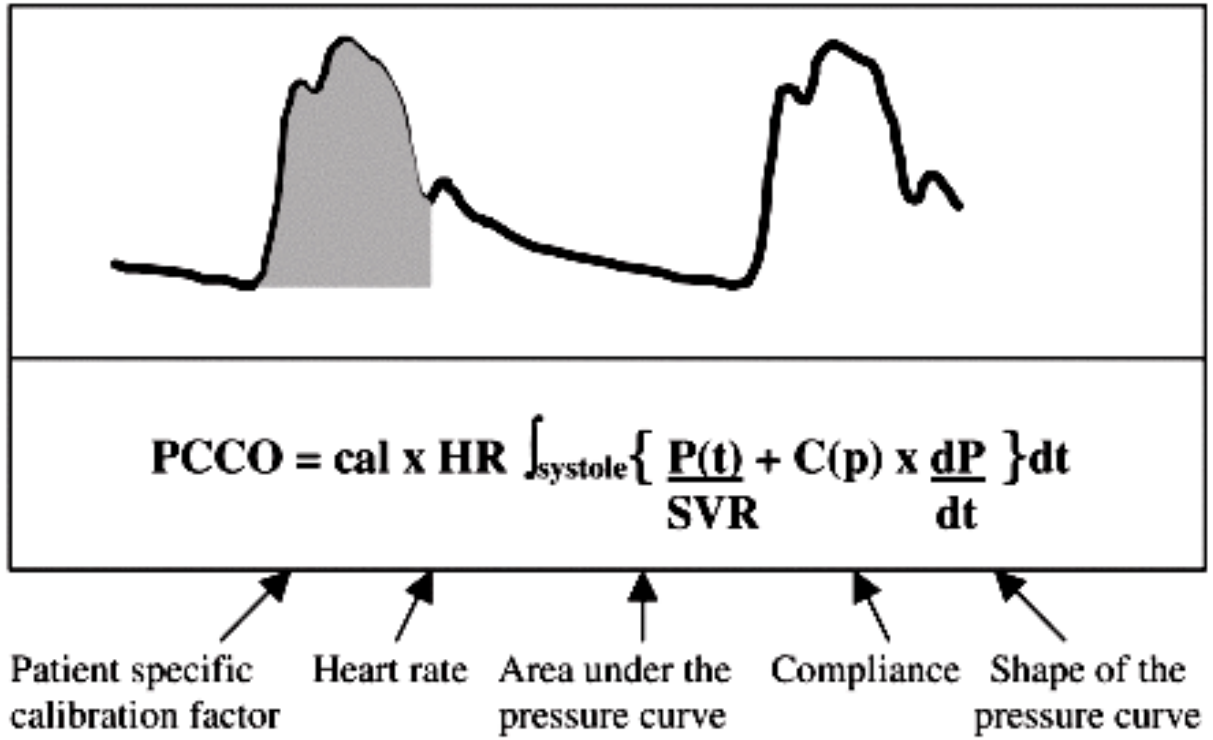


Figure 2. Calculation used by the PiCCO for measurement of continuous cardiac output.

Pulse contour analysis

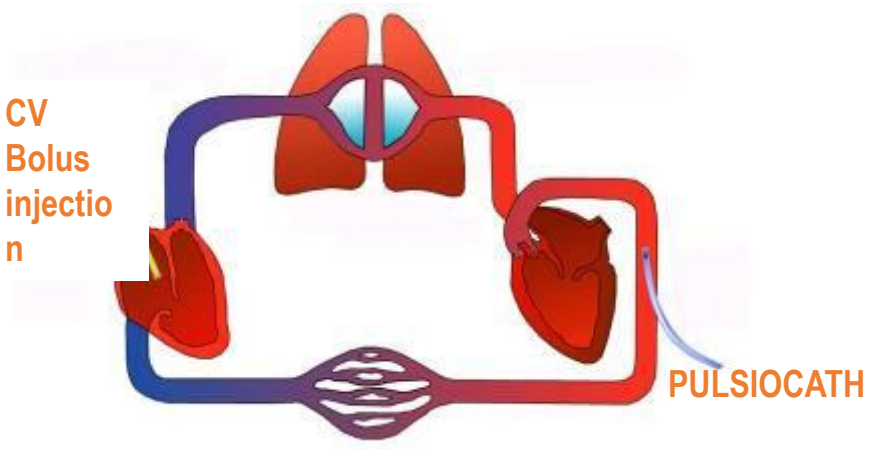
- Because vascular impedance varies between patients, it had to be measured using another modality to initially calibrate the PCA system
- The calibration method usually employed was arterial thermodilution or dye dilution technique
- PCA involves the use of an arterially placed catheter with a pressure transducer, which can measure pressure tracings on a beat-to-beat basis
- PiCCO and LiDCO are the two commonly used model

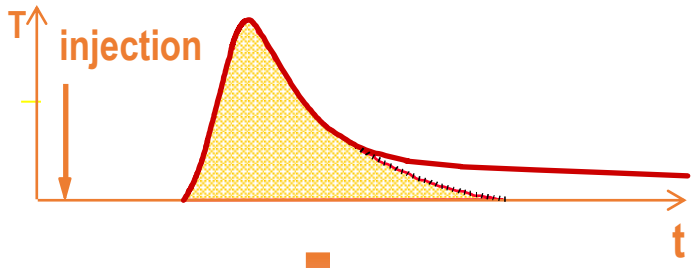
What is the PiCCO-Technology?

depend on 1- trans-pulmonary thermodilution but without pulmonary artery catheter 2- pulse contour analysis

- The PiCCO-Technology is a unique combination of 2 techniques for advanced hemodynamic and volumetric management without the necessity of a right heart catheter in most patients:

- Transpulmonary Thermodilution





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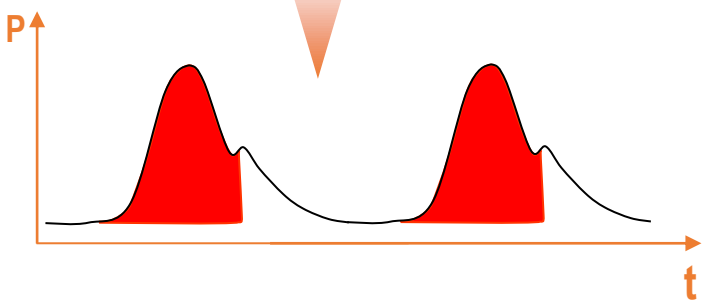


- Pulse Contour Analysis

How does the PiCCO-Technology work?

so the patient need central line and arterial line and ECHO

- Most of hemodynamic unstable and/or severely hypoxemic patients are instrumented with:

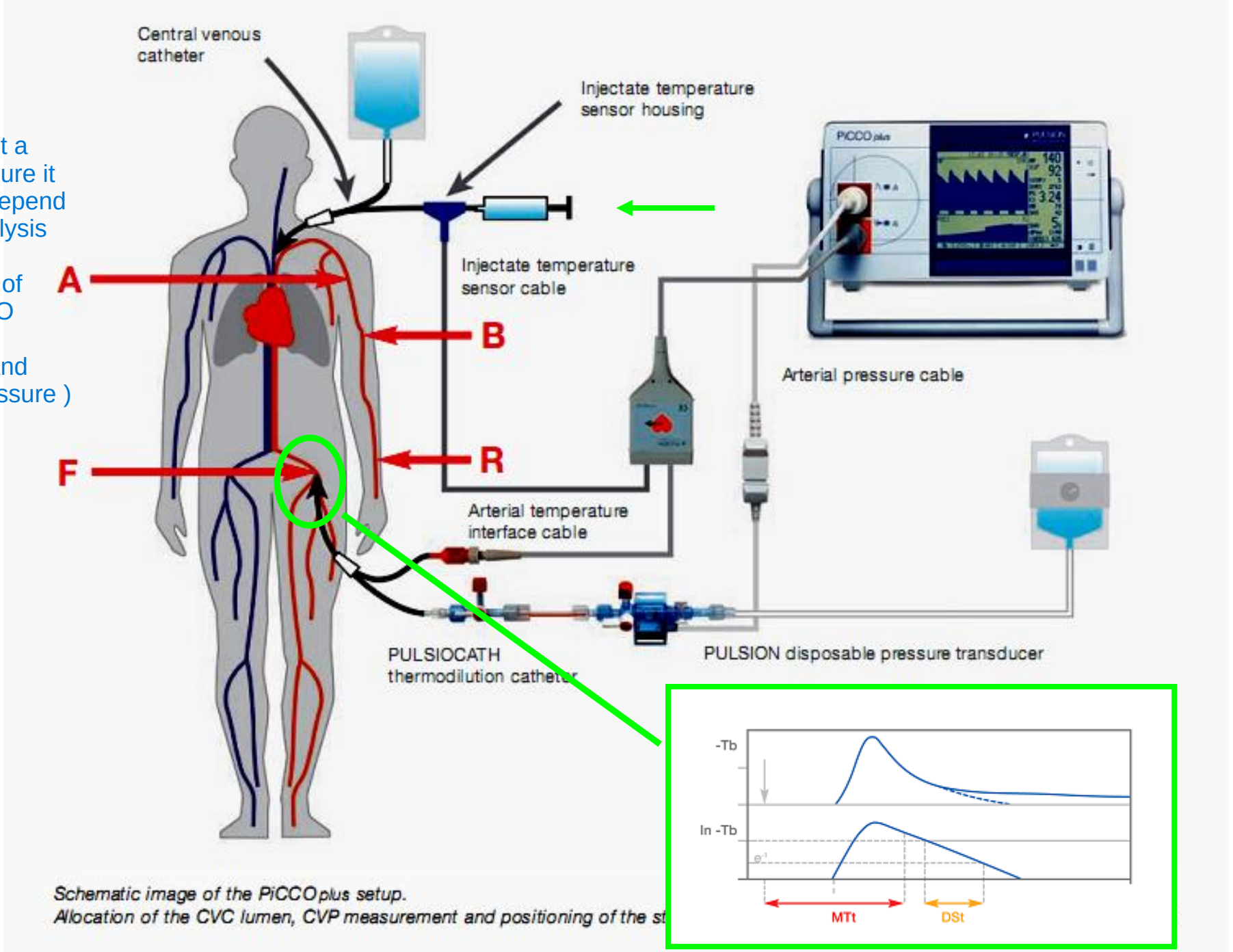
Central venous line (e.g. for vasoactive agents administration…)

Arterial line (accurate monitoring of arterial pressure, blood samples…)

- The PiCCO-Technology uses any standard CV-line and a thermistor-tipped arterial PiCCO-catheter instead of the standard arterial line.

so we give the patient a cold saline and measure it from the artery and depend on pulse contour analysis

we can obstruct a lot of information from PICO except (pulmonary vascular resistance and pulmonary artery pressure)



Schematic image of the PiCCOplus setup.
Allocation of the CVC lumen, CVP measurement and positioning of the st

Fig 1

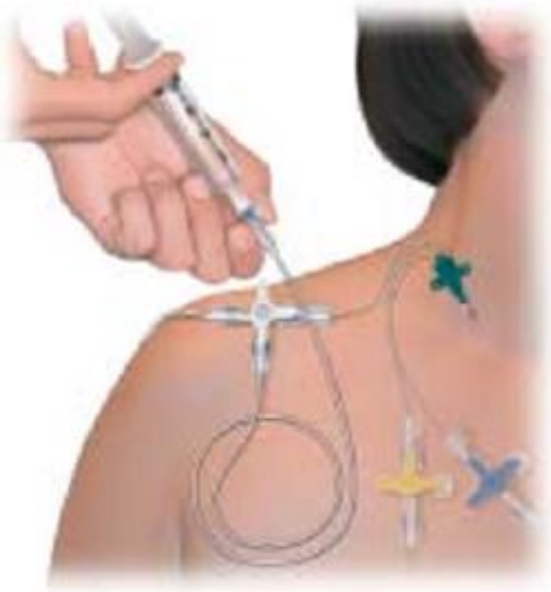
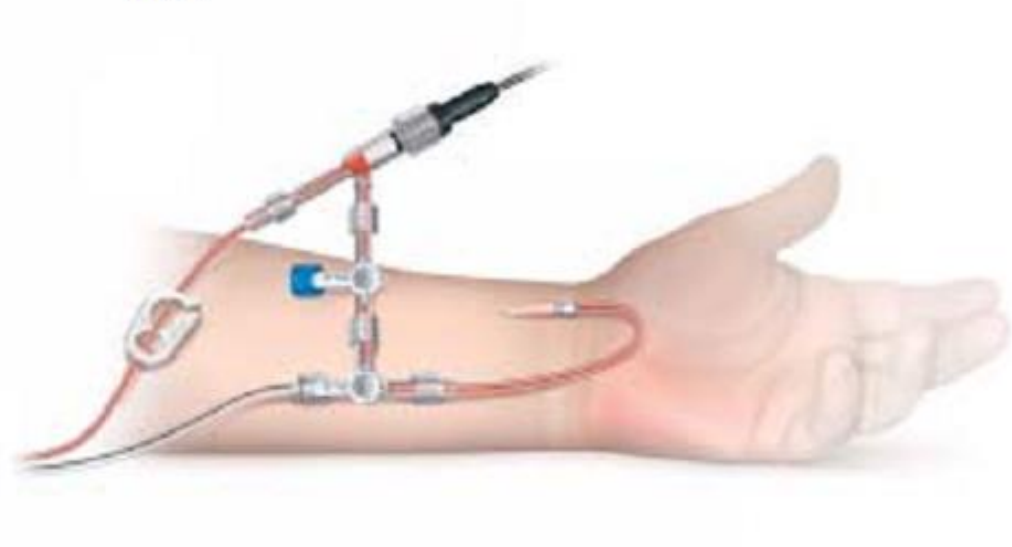
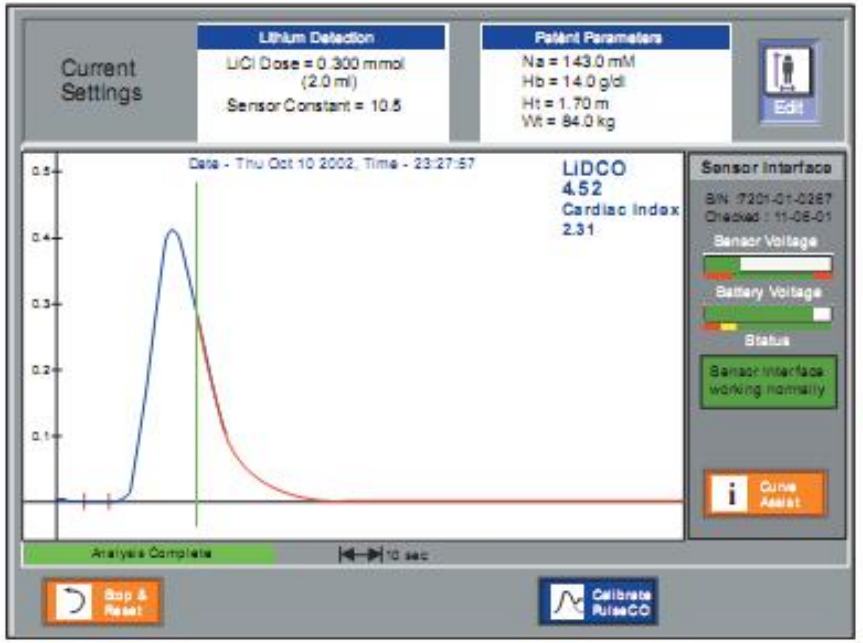


Fig 2



depend in pulse contour analysis
-dont contain thermodilution so the PICO is more accurate

Fig 3



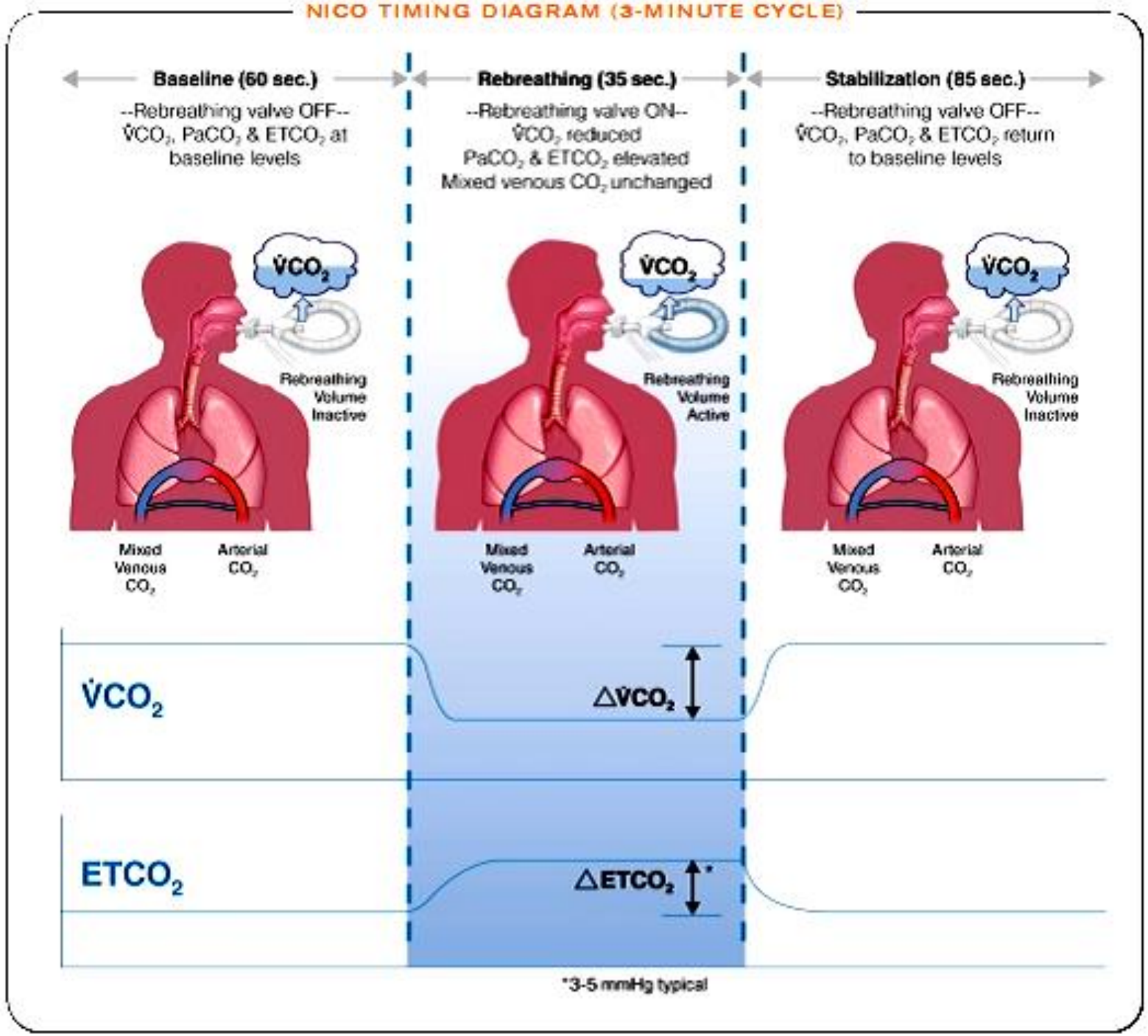
LiDCO system

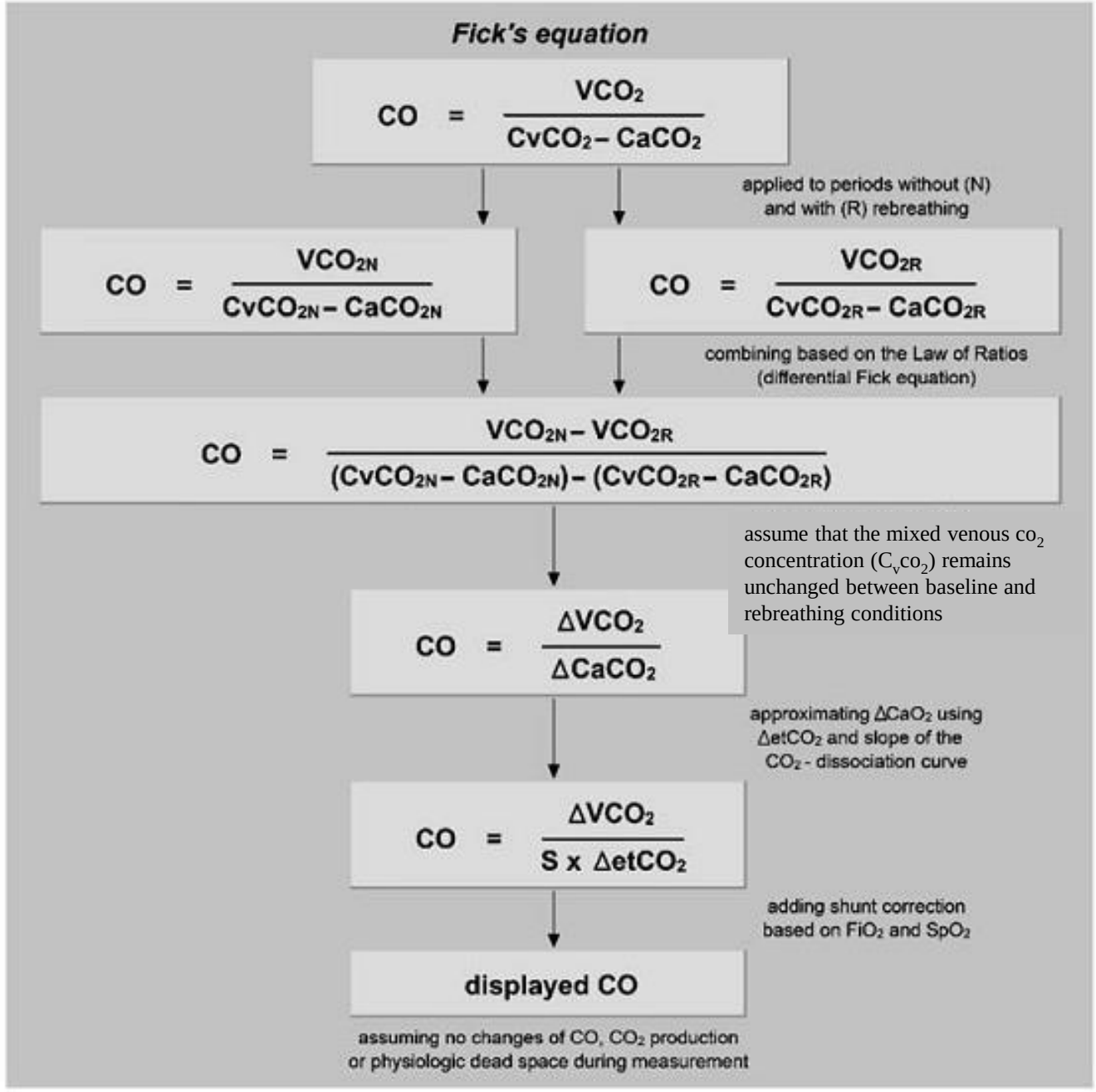
Pulse contour analysis

- Advantages
  - Almost continuous data of CO / SV / SV variation
  - Provide information of preload and EVLW
- Disadvantages
  - Minimal invasive
  - Optimal arterial pulse signal required
    - Arrhythmia
    - Damping
    - Use of IABP

Partial carbon dioxide rebreathing with application of Fick principle

- Fick principle is used for CO measurement
- $CO = VO_2 / (CaO_2 – CvO_2) = VCO_2 / (CvCO_2 – CaCO_2)$
- Based on the assumption that blood flow through the pulmonary circulation kept constant and absence of shunt
- Proportional to change of CO_2 elimination divided by change of $ETCO_2$ resulting from a brief rebreathing period
- The change was measured by NICO sensor


NICO TIMING DIAGRAM (3-MINUTE CYCLE)
Baseline (60 sec.)
--Rebreathing valve OFF--
V̇CO2, PaCO2 & ETCO2 at baseline levels
Rebreathing (35 sec.)
--Rebreathing valve ON--
V̇CO2 reduced
PaCO2 & ETCO2 elevated
Mixed venous CO2 unchanged
Stabilization (85 sec.)
--Rebreathing valve OFF--
V̇CO2, PaCO2 & ETCO2 return to baseline levels
V̇CO2
Rebreathing Volume Inactive
Rebreathing Volume Active
Rebreathing Volume Inactive
Mixed Venous CO2
Arterial CO2
V̇CO2
△V̇CO2
ETCO2
△ETCO2
*
*3-5 mmHg typical



S = slope of CO2 dissociation curve

Partial carbon dioxide rebreathing with application of Fick principle

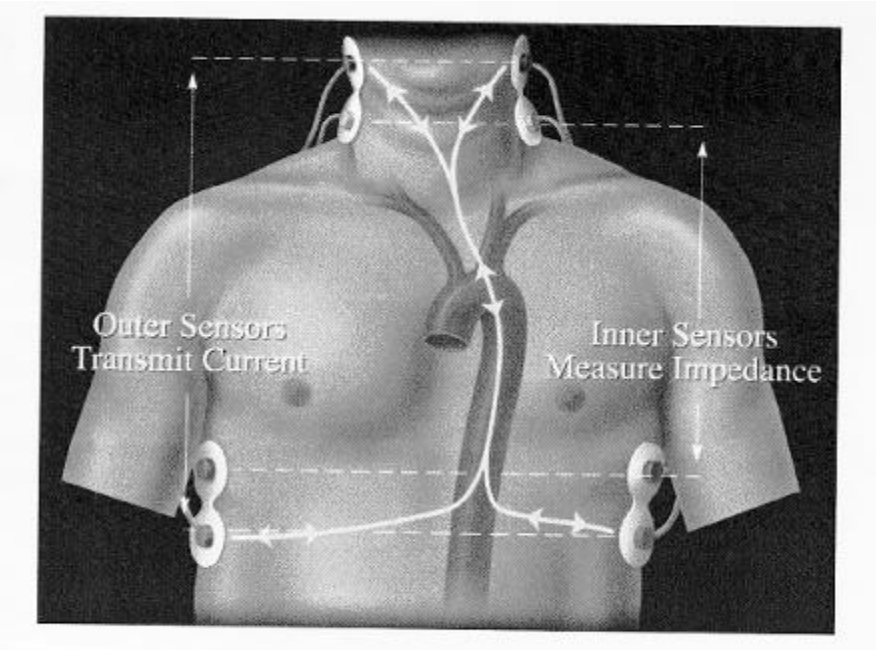
- Advantages
  - Non invasive but the patient must be mechanically ventilated
- Disadvantages
  - Only for those mechanically ventilated patient
  - Variation of ventilation modality and presence of significantly diseased lung affect the CO reading
  - Not continuous monitoring

Electrical bioimpedance

- Made uses of constant electrical current stimulation for identification of thoracic or body impedance variations induced by vascular blood flow

Electrical bioimpedance

we put sensors and give electrics and measure the time of the transmit of the electrical wave from place to another
- the more the water in the body (like in congested HF patients --> the electrical waves transmit quicker
-measure CO



- Electrodes are placed in specific areas on the neck and thorax
- A low-grade electrical current, from 2 - 4 mA is emitted, and received by the adjacent electrodes
- Impedance to the current flow produces a waveform
- Through electronic evaluation of these waveforms, the timing of aortic opening and closing can be used to calculate the left ventricular ejection time and stroke volume

Electrical bioimpedance

- Some report same clinical accuracy as thermodilution technique

Crit Care Med 22: 1907-1912

Chest 111: 333-337

Crit Care Med 14: 933-935

- Other report poor agreement in those haemodynamically unstable and post cardiac surgery

Crit Care Med 21:1139-1142

Crit Care Med 23: 1667-1673

- Newly generation EB device using upgraded computer technology and refined algorithms to calculate CO and get better results

Curr Opin Cardio 19:229-237

Int Care Med 32:2053-2058

Electrical bioimpedance

- Advantage
  - Non invasive
- Disadvantage
  - Reliability in critically ill patients still not very clear

we aim to do preload / cardiac function / after load for right and left ventricle to know the type of shock

Classic Hemodynamic Changes Associated with Shock States

Shock States	SVR	PVR	CI	SvO2	RAP	RVP	RAP	PAOP
	systemic vascular resistance	pulmonary vascular resistance	cardiac output / index	central venous sat	RT atrial pressure	RT ventricualr pressure		pylmonary artery - occlusive pressure
Cardiogenic	↑	N	↓	↓	↑	↑	↑	↑
Distributive	↓	N	N/↑	N/↑	N/↓	N/↓	N/↓	N/↓
Hypovolemic	↑	N	↓	↓	↓	↓	↓	↓
Obstructive	N/	↑	↓	N/↑	↑	↑	↑	N/↓

Table 3: Expected changes to classic hemodynamic parameters in shock states.

In conclusion

- Haemodynamic monitoring enable early detection of change in patient’s conditions
- New techniques provide reasonably good results and less invasive
- Always correlate the readings / findings with clinical pictures in order to provide the best treatment options

Thank you for your Attention