Haemodynamic monitoring

Central venous pressure monitoring – assessment of RV filling pressure

The central venous pressure (CVP) is measured on the tip of a catheter placed in the superior vena cava. Both the waveform and the mean value can be assessed.

CVP trace - normal pattern:

- a-wave occurs under atrial contraction
- c-wave occurs under isovolumetric ventricular contraction / bulging of the closed TV into the RA
- x-descent represents the RA pressure drop during atrial relaxation and early ventricular systole, when the TV is pulled away from RA
- v-wave occurs under late ventricular systole due to RA filling
- y-descent represents the TV opening / RA emptying into the RV

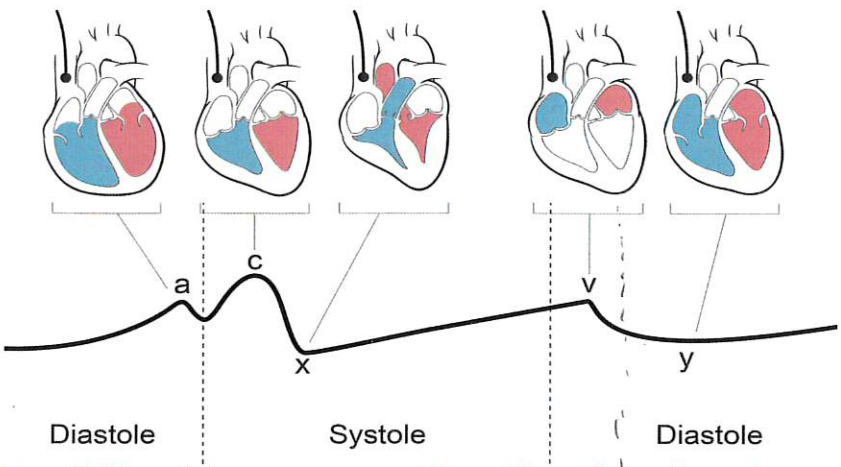

Figure 15: The central venous pressure waveform and associated cardiac events

The normal pattern may not always be obvious and absence does not necessarily equal pathology, though an altered CVP trace morphology may give a clue to underlying abnormalities, as for example:

- Cannon a-waves indicating right atrio-ventricular dyssynchrony. Cannon waves are caused by the right atrium (RA) contracting against a closed tricuspid valve, as in 3° AV block or pacemaker asynchrony.
- Giant v-waves are caused by tricuspid regurgitation during ventricular systole. Changes depend on the degree of regurgitation. In mild TR, the v-wave becomes more prominent. In severe TR, the x-descent obliterates resulting in one large systolic v-wave, called ventricularisation of the CVP trace.
- CVP values do not correlate well with circulating blood volume or fluid responsiveness. However, used in conjunction with a detailed examination and potentially echocardiography, CVP trends can be used to assess the patient's condition and response to therapies.

RA filling

The CVP transducer is zeroed with atmospheric pressure at the level of the RA so the pressure it measures is that which is relative to atmospheric pressure. CVP is conventionally measured at end expiration when the intrathoracic pressure should equal atmospheric

pressure, as there is no airflow with an open airway. The addition of PEEP or the presence of auto-PEEP will raise the intra-thoracic pressure and so raise the measured CVP value. However, the PEEP is not simply subtracted from the CVP value to obtain the filling pressure. The effect of airway pressure on the CVP also depends of the percentage transmission of PEEP to the intrapleural cavity - this is often approximated at 50%. In low- compliance lung pathology this pressure transmission may be much less. It is worth noting that intra-operative CVP values will be lower when the chest is open.

Traditionally, an elevated CVP was quoted as greater than 15 mmHg, though this will change from patient to patient and with the proportion of the transmitted positive end expiratory pressure (PEEP) in ventilated patients.

A high CVP may lead to examination for further evidence of:

- Cardiac tamponade
- RV failure
- Severe tricuspid regurgitation
- Extra-cardiac causes of high CVP: tension pneumothorax, pulmonary embolus, SVC compression / obstruction / thrombosis

Pulmonary artery catheters

Pulmonary artery catheters (Swan-Ganz Catheters, PACs) are still the gold standard of cardiac output monitoring to which new technology is tested against. They also give access to measurement of the pulmonary arterial pressure (PAP) and the pulmonary artery occlusion pressure (pulmonary capillary wedge pressure, PAOP, PCWP), an estimate of left atrial pressure (LAP). But clinical usefulness is debatable. There is a significant risk of serious complications (2-9%) and measurements should have impact on clinical management to justify its placement.

Insertion:

The PAC is inserted through an introducing sheath in a large vein and advanced under continuous pressure measurement at the tip of the catheter. Additional fluoroscopic guidance is helpful in complicated cases, especially in marked RA and RV dilatation. Preparations, consent issues, contraindications and the advantage of ultrasound-guided puncture are comparable to any other central line insertion. Usually at least two persons are required, one operator at the sterile insertion site, and one non-sterile assistant. Before introducing the catheter into the sheath the fiber-optic oximetry has to be calibrated, the integrity of the balloon checked, all lines flushed and capped, and both pressure transducers (CVP and PAP) connected, referenced and zeroed. The operator should have an unobstructed view of the haemodynamic monitor, and the PAP scale calibrated to 0-60 mmHg. Usually a sterile protective sleeve is placed on the PAC before insertion, which later can be connected to the introducer hub. Once the catheter tip reaches the superior vena cava, the balloon is fully inflated to aid floatation. Moderate resistance to inflation should be felt. The catheter is then advanced in a slow continuous motion. Even thought the catheter has a total length of 110 cm, it is rarely necessary to insert it more than 60 cm, otherwise there is an increased risk of loops and knots in the RA or RV resulting in failed placement and complicated removal. Placement is usually easiest from the right internal jugular vein with the preformed curve of the catheter pointing to the patients left side. While the

catheter is advanced through the central venous circulation and the heart, these characteristic pressure waveforms are recorded:

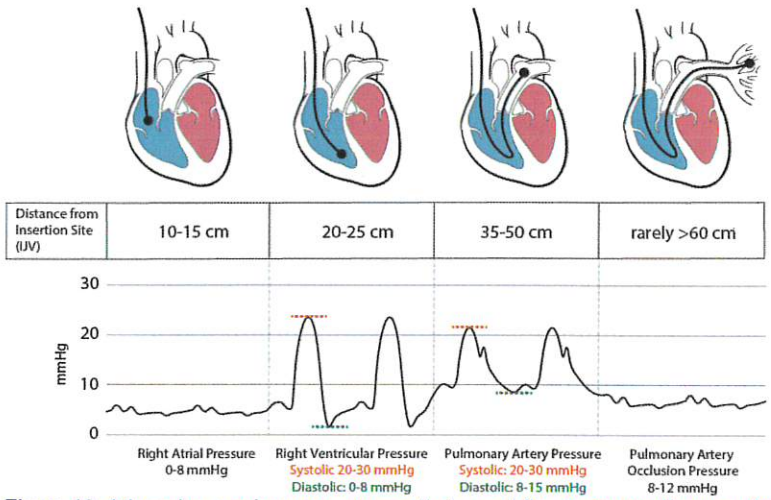

Figure 16: Advancing a pulmonary artery catheter and the corresponding waveforms

Flotation can be technically difficult in low cardiac output states, TV abnormalities, RV dilatation and pulmonary hypertension.

A correctly placed PAC should lie in West Zone 3 of the lung. This is the most dependent portion of the lung. Here the occlusion pressure most accurately reflects left atrial pressure rather than alveolar pressure.

Criteria for correct placement

- PAOP ≤ PADP, visible A and V-waves
- PaO2 from the tip of the wedged PAC slightly > peripheral arterial PaO2 (due to additional oxygen diffusion when blood flow is blocked by the balloon)
- Change of PAOP less than 50% change of PEEP (if the patients tolerates an increase in PEEP by at least 5cmH20 over a short period of time)
- CXR (two plane) after PAC insertion:
  - Front: rule out pneumothorax, and catheter loops within the RV. Catheter tip should not be above the pulmonary hilum
  - Lateral: Catheter tip below the level of the LA (=West Zone 3 in supine position)

Safety

- Never leave the balloon inflated!
- Never retract a PAC with the balloon inflated!
- When deflating the balloon the PAP curve should reoccur
- If less than 1 ml air is required to obtain the PAOP curve again, deflate the balloon immediately and withdraw the PAC at small increments (0.5 cm)
- The pressure trace should be continuously monitored to avoid inadvertent wedging (risk of infarction)

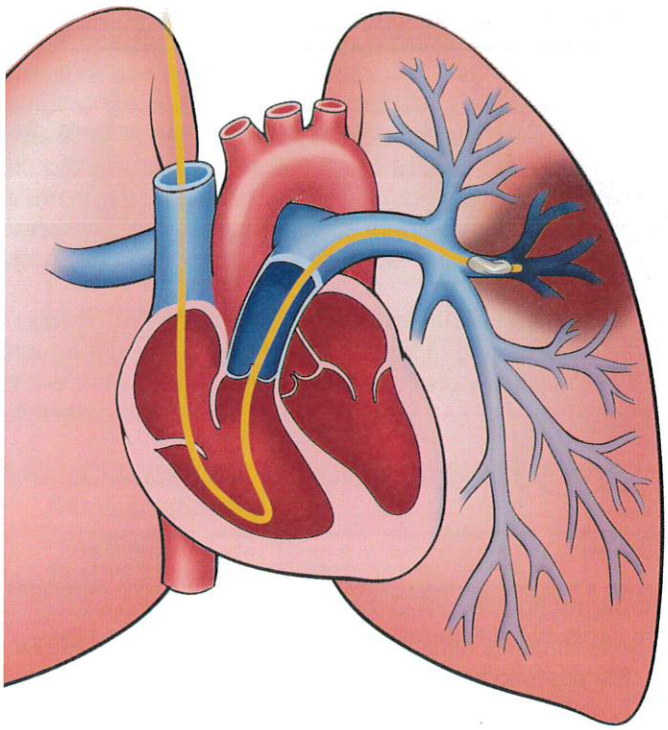

Figure 17 Inadvertently wedged PA catheter

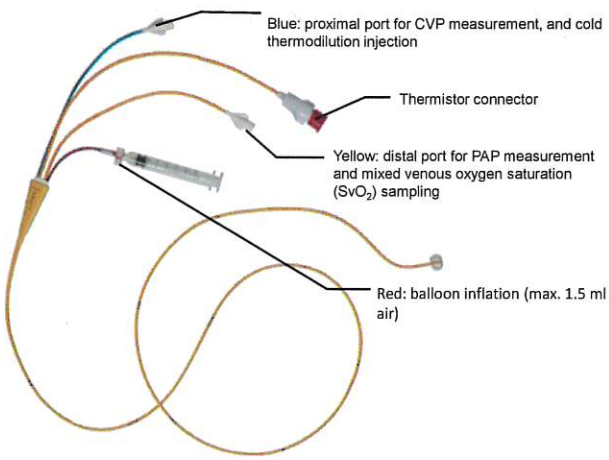

Figure 18. Standard pulmonary artery catheter

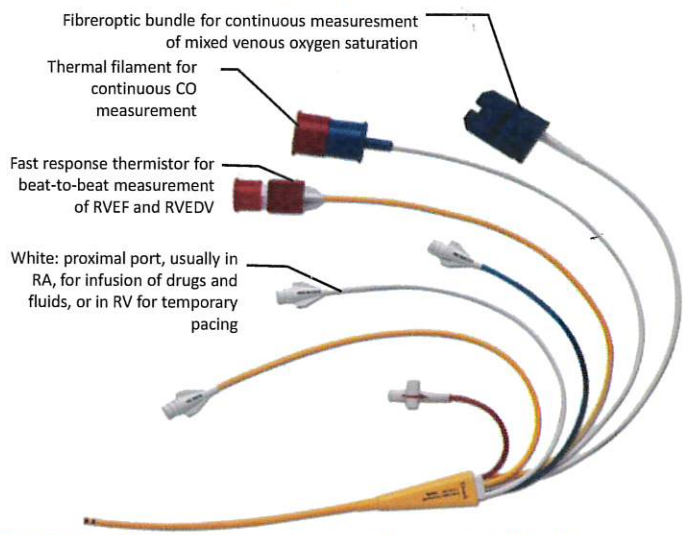

Figure 19: Pulmonary artery catheter with several additional lumen

Indications for PAC insertion:

- Shock with evidence of tissue hypoperfusion not responding to conventional therapy (especially cardiogenic, or combination of shock forms)
- RV infarct – acute RV failure
- Management of refractory pulmonary oedema
- Cardiac output monitoring with IABP in situ
- Patients with pulmonary hypertension (PHT) undergoing open heart surgery

Formulae for derived values

$$\text{Stroke volume (ml)} = \frac{\text{Cardiac output}}{\text{Heart rate} \times 1000}$$

$$\text{Cardiac index} = \frac{\text{Cardiac output}}{\text{Body surface area}}$$

$$\text{Systemic vascular resistance} = \frac{\text{MAP} - \text{RAP}}{\text{CO} \times 80}$$

$$\text{Pulmonary vascular resistance} = \frac{\text{Mean PAP} - \text{PAOP}}{\text{CO} \times 80}$$

$$\text{Left ventricular stroke work index} = \text{SV} \times \frac{\text{MAP} - \text{PAOP}}{\text{Body surface area}}$$

Interpretation

Parameter	Normal	Cardiogenic shock	RV infarct or failure	Septic shock	Cardiac tamponade
Directly measured:					
RAP [mmHg]	0-8	↑↑	↑↑↑	N	Equalization of RAP, RVEDP, PA diastolic and PAOP: 12-18 mmHg
RV systolic diastolic [mmHg]	15-28 0-12	↑↑	EDP ↑↑↑	N or ↓	
PA systolic diastolic mean [mmHg]	15-28 5-15 10-22	↑↑	N or ↑	N or ↓	
PAOP [mmHg]	5-12	↓↓	N	N	
CO [L/min]	4-6	↓↓	↓↓	↑↑	↓↓↓
Derived :					
Stroke volume [ml/beat]	70-130	↓↓	↓↓	N or ↑	↓↓↓
Cardiac index [l/min/m^2]	2.5-4.2	↓↓	↓↓	↑↑	↓↓↓
Systemic vascular resistance [dynes.s/cm^5]	900-1500	↑↑	N or ↑	↓↓↓	N
Pulmonary vascular resistance [dynes.s/cm^5]	120-250	↑↑	N or ↑	N or ↓	N
Left ventricular stroke work index [g/m/beat/m^2]	45-60	↓↓↓	↓↓	↓↓	↓↓↓

Table 3: Haemodynamic parameters available from a PAC – normal values in recumbent adults, and alterations to expect in common CTS pathologies

- Systolic RVP and PAP rarely exceed 40-50 mmHg in acute conditions. Higher values are indicative of an underlying chronic pulmonary hypertension.

- A significant systolic pressure gradient between the RV and the PA is found in pulmonary stenosis.

Measuring and interpreting the PAOP

Estimating LV filling pressure:
When occluding a tapering branch of one of the pulmonary arteries, phasic blood flow and arterial pressure variation ceases. The pressure measured at the tip of the catheter reflects the LA pressure transmitted by a static column of blood. PAOP should be measured at end-expiration (both in spontaneously breathing and mechanically ventilated patients), when pleural pressure can be assumed to be closest to zero. Usually at least three measurements are performed and the mean value calculated.

Both applied PEEP and intrinsic PEEP increase the intrapleural pressure, causing the measured PAOP to overestimate the actual LV filling pressure. With normal lung- and chest wall compliance approximately 50% of the PEEP is transmitted to the pleural space, and PAOP will rise by less than 50%. If lung compliance is decreased (for example in severe ARDS) PEEP will have less effect on PAOP measurements. Therefore any increase in PAOP > 50% should not be attributed to the application of PEEP.

Conditions where PAOP does not accurately reflect LV filling pressure:

PAOP < LVEDP:

- Decreased LV compliance (hypertrophy, ischaemia) probably due to premature MV closure
- Severe AR
- Reduction of the pulmonary vascular tree (pneumectomy, massive pulmonary embolism)

PAOP > LVEDP:

- Elevated V-waves (quite frequent) due to MR
- Obstruction in pulmonary veins (tumor, fibrosis, thrombosis)
- LA mass (myxoma, thrombus)
- Tachycardia > 130 bpm
- Increased pleural pressure
- Catheter placement in West Zone 1 or 2(if PEEP applied)

Even in the absence of these caveats, it is important to remember that PAOP is only a surrogate measure of the LV filling volume / LV preload.

Measurement of cardiac output

The thermodilution method by a PAC is well validated and remains the gold standard to which other systems are tested against. It is based on the injection of an indicator substance (usually 10 ml of cold dextrose or saline) into the bloodstream at the proximal port of the PAC and the measurement of its dilution in the blood downstream at the distal port of the PAC in the PA. The lowering of the blood temperature over time is recorded as a temperature-time curve. The area under the curve is inversely proportional to the flow rate, and hence an estimate of CO, as long as there is no intra-cardiac shunt (overestimation of the CO) or TR (underestimation of the CO).

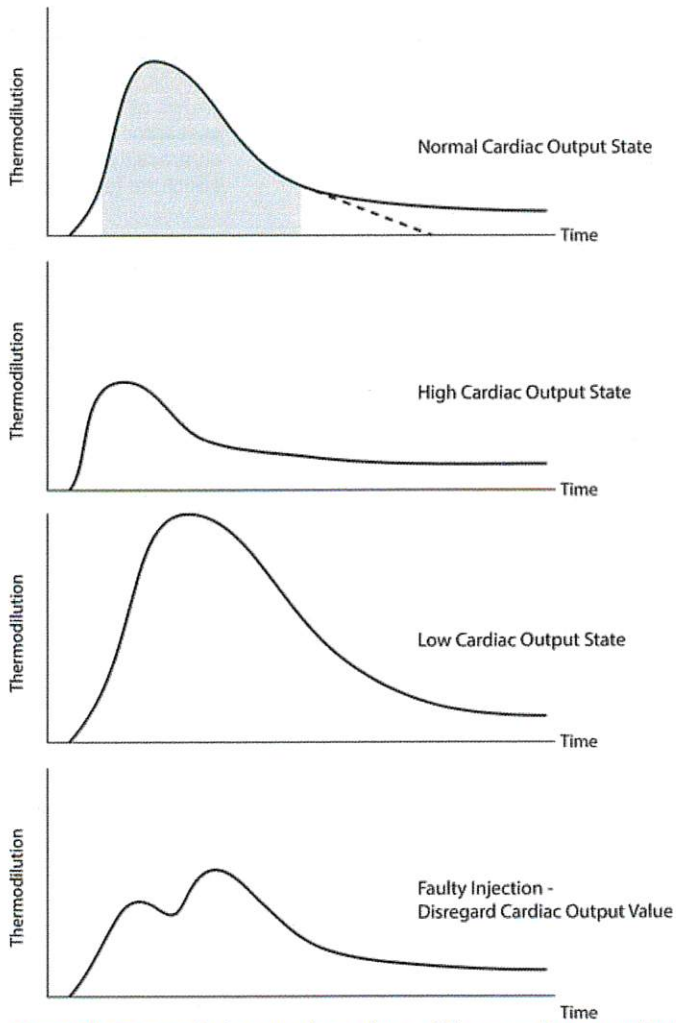

Figure 20. Thermodilution over time, shape of the normal curve and common alterations

Usually the mean of at least three thermodilution measurements is calculated. The measurements should be done in expiration, and the variation has to be less than 10%. There are several newer methods available to measure cardiac output. Modern PACs have an integrated heating filament that warms the blood flowing past it. A thermistor near the tip of the catheter measures temperature changes of the blood and uses the data to average CO over time, providing continuous readings.

Several non-invasive CO measurement technologies have been developed and they may be categorized into calibrated and non-calibrated systems. Calibrated systems use intermittent dilutional CO (cold saline or lithium dilution) data to calibrate the pulse waveform data. Non-calibrated systems use patient data such as sex, height and weight to derive CO from measurement data alone. Calibrated non-invasive CO monitoring systems are more reliable in patients on vasopressor/inotrope therapy than the non-calibrated pulse contour systems or the oesophageal Doppler systems.

Furthermore, the non-calibrated systems rely on trending responses to fluid challenges that may be detrimental to cardiac surgical patients with significant pre-existing impairment of cardiac function.

Method	Device	Precision	Features
Pulmonary thermodilution	PAC	+/- 20%	Gold standard, but invasive
Echo	TTE / TOE	operator dependent	Allows additional assessment of contractility and structural features
Transpulmonary thermodilution	PiCCO™, VolumeView™	good agreement with PAC	Calibrated, less invasive (CVC + arterial cannula)
Transpulmonary indicator dilution	LiDCO™	good agreement with PAC	Calibrated, less invasive (PVC and arterial cannula)
Arterial pressure waveform derived	PiCCO™, LiDCO (*rapide*)™ FloTrac/Vigileo™ Finapres™, Nexfin™	variable, depending on reliable waveform	Non-calibrated, continuous, measurement, does not work with irregular HR or IABP
Oesophageal doppler	CardioQ™	variable, depending on probe position	Non-calibrated, continuous measurement, Minimally invasive, but requires some sedation

Table 4: Methods of measuring or estimating the cardiac output

Assessment of fluid responsiveness

Fluid responsiveness is asking the question whether a patient's cardiac output will increase on fluid administration. But it is important to remember, that in healthy individuals the heart usually still is fluid responsive; therefore fluid therapy should only be given to the patient if there is evidence of fluid responsiveness ***and*** organ hypoperfusion at the same time.

Fluid challenge: The easiest way of answering this question is to give a fluid bolus – in patients with impaired systolic or diastolic function smaller fluid boluses should be given over a longer period of time (for example 100 ml crystalloid over 15 minutes).

A subsequent 15% increase of cardiac output from baseline is usually considered proof of fluid responsiveness. Bear in mind that in cardiac patients already the first fluid challenge might be harmful, when hypotension is not attributable to hypovolaemia, and the heart already is operating on the horizontal limb of the Frank Starling curve.

Leg raise test: A proper conducted leg raise test resulting in "auto-transfusion" from the patient's lower limbs has the advantage of reversibility compared to the fluid challenge. To obtain reliable test results, several factors are important:

- The patient should be informed, if awake, and not be in any apparent distress.
- The patient's bed is moved from semi recumbent position to leg raise position.
- Preferably cardiac output should be assessed for at least one minute.
- If no CO measure available, a 10-15% increase in arterial pulse pressure can be regarded a sign of fluid responsiveness.

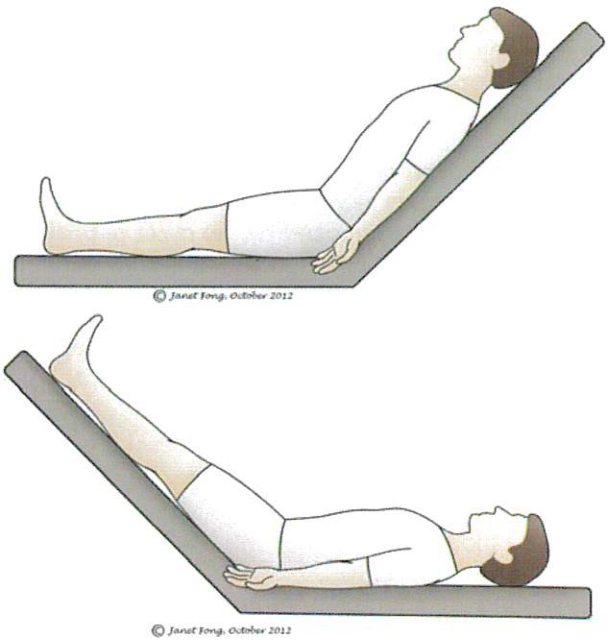

Figure 21: Performing a passive leg raise

There are also several other surrogate measures of fluid responsiveness. They all have specific limitations, but may give additional information to enhance the clinical picture.

		Measure	Method / Device
Static measures	Pressure	Jugular vein pressure (JVP)	Visualization
		Central venous pressure (CVP)	Central venous catheter
		Pulmonary artery occlusion pressure (PAOP)	Pulmonary artery catheter
	Volume	Global end-diastolic volume (GEDV)	Transpulmonary thermodilution (PiCCO™, VolumeView™)
		Left-ventricular end-diastolic volume	Echocardiography
Dynamic measures		Pulse pressure variation (PPV)	(PiCCO™, LiDCO plus™, Most Care™)
		Stroke volume variation (SVV)	Arterial pulse contour analysis (PiCCO™, LiDCO plus™, Most Care™, FloTrac/Vigileo™) Volume clamp method (Finapres™, Nexfin™) Assessment of superior vena cava (SVC) and inferior vena cava (IVC) Echo Doppler

Table 5: Static and dynamic measures of fluid responsiveness

Measuring venous pressures and end-diastolic volumes of the heart chambers result in static surrogate estimates of RV preload and LV preload. They all are poorly correlated to fluid responsiveness since the relationship of preload and stroke volume depends on ventricular contractility and the compliance of the venous system.

There are also limitations of dynamic measures in the mechanically ventilated patients. These methods rely on the concept that the variations in intrathoracic pressure imposed by the cycle of positive pressure ventilation affect venous return and subsequently cardiac output. These variations are exaggerated in hypovolaemia indicating that the heart currently is operating on the ascending limb of the Frank-Starling curve. The major drawback with all respiratory cycle related measurements estimating LV preload is that they are only validated in paralyzed patients receiving a tidal volume of at least 8-10 ml/kg, who also have a regular heart rate. In postoperative patients with a regular heart rate, these tests still can be performed, as long as they are sedated and paralyzed, by transiently increasing the TV to 10 ml/kg, even though in general a more lung protective ventilator setting is preferred. Under these circumstances a pulse pressure- or stroke volume variation of more than 10-15% is regarded a reliable predictor of fluid responsiveness.