PiCCO Technology

Hemodynamic monitoring at the highest level

This document is intended to provide information to an international audience outside of the US.

GETINGE

Simplify hemodynamics

Understand complex conditions with PiCCO

The life of your critically ill patient depends on the right decision for the next therapeutic step. Therapeutic conflicts often arise at the critical care bedside, where you need dependable information you can trust. A set of reliable hemodynamic parameters can help determine the best individual treatment for your patients.

The PiCCO Technology was introduced in 1997 by the Munich based company Pulsion Medical Systems. With more than 20 years of experience in the hemodynamic monitoring industry, Pulsion has developed PiCCO into the established standard for advanced hemodynamic monitoring, today.

As the evidence, PiCCO has achieved modular integration with world market leaders for patient-monitoring devices including Philips / Dixtal, Drager, GE, Mindray and Nihon Kohden.

Over the last 15 years, nearly 1,000 publications worldwide have confirmed the accuracy and clinical benefit of the PiCCO Technology.

Today, PiCCO is used more than 140,000 times per year, in over 60 countries globally.

since
1997

publications
1,000

annual PiCCO applications
140,000

Basics of hemodynamic monitoring

Monitoring cardiocirculatory function is of major importance in all intensive care patients.

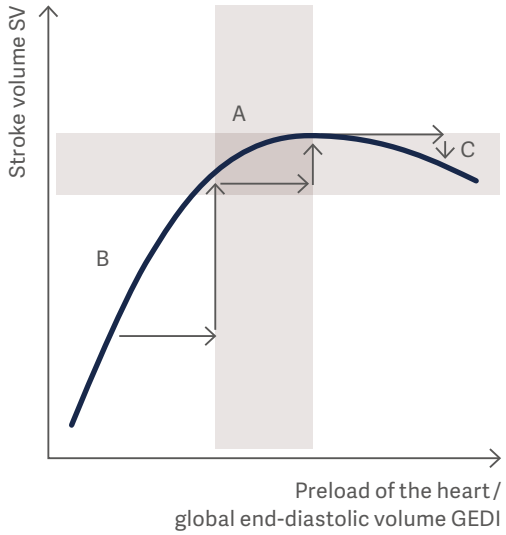
Monitoring with standard parameters: ECG non-invasive blood pressure and pulse oximetry provides insufficient information for deciding on the adequacy of treatments. Only advanced hemodynamic monitoring with minimally-invasive measurement of cardiac output and its determinants (preload, afterload, contractility) as well as the quantification of pulmonary edema allows a targeted treatment.

Frank-Starling mechanism

The Frank-Starling law states that the greater the volume of blood entering the ventricle during diastole (end-diastolic volume), the greater the volume of blood ejected during systolic contraction (stroke volume) and vice-versa. This is an adaptive mechanism of the organism to compensate for slight changes in the ventricular filling.

The power of the heart muscle depends on its initial load before the start of contraction.

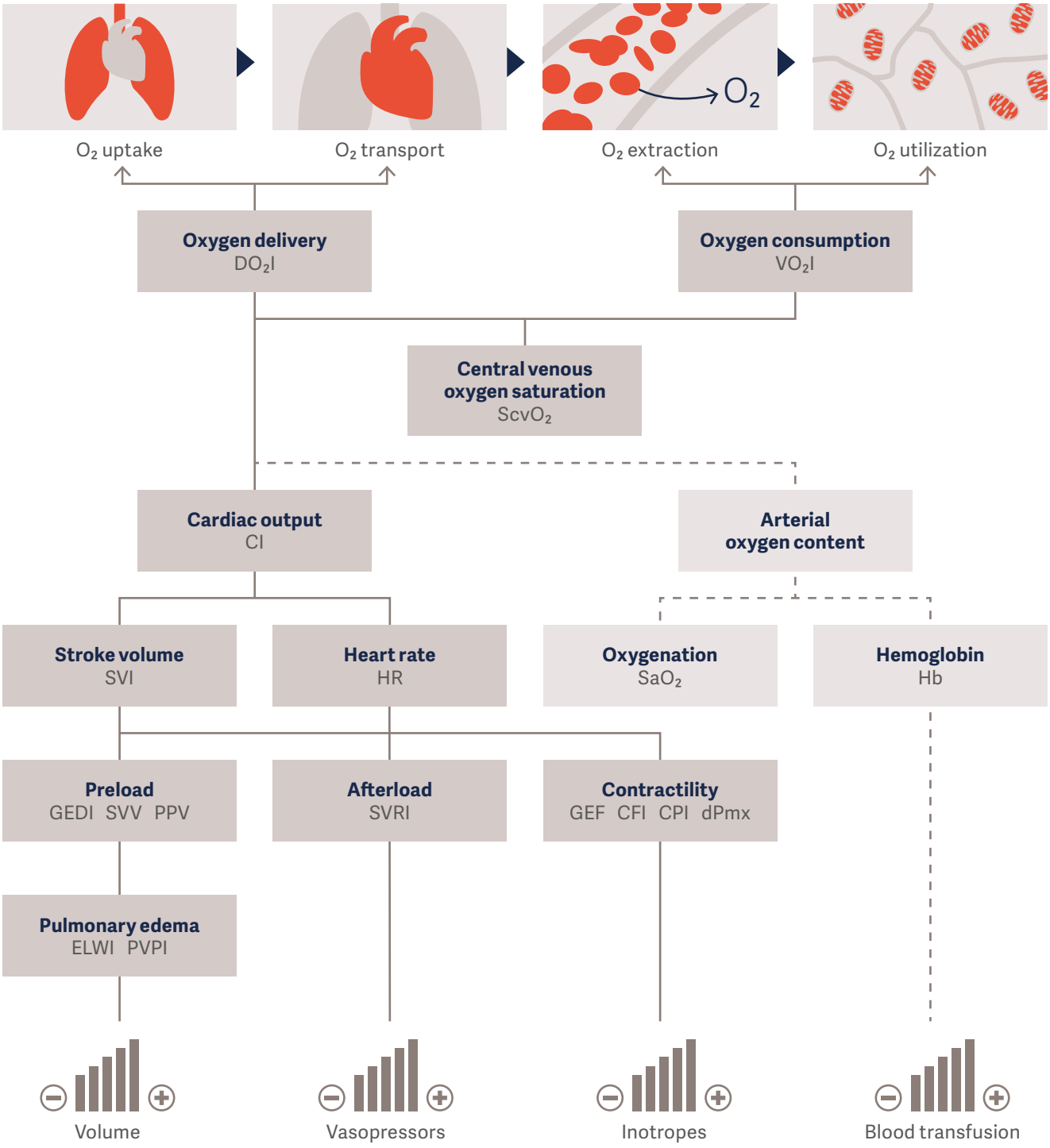
However, it can also be used to increase stroke volume by volume administration for therapeutic reasons. The force that any single cardiac muscle fibre generates is proportional to the initial sarcomere length (known as preload), and the stretch on the individual fibres is related to the end-diastolic volume of the ventricles.



Schematic Frank-Starling curve for verification of the preload status
A = Optimal preload, B = Volume reponsive, C = Volume overload

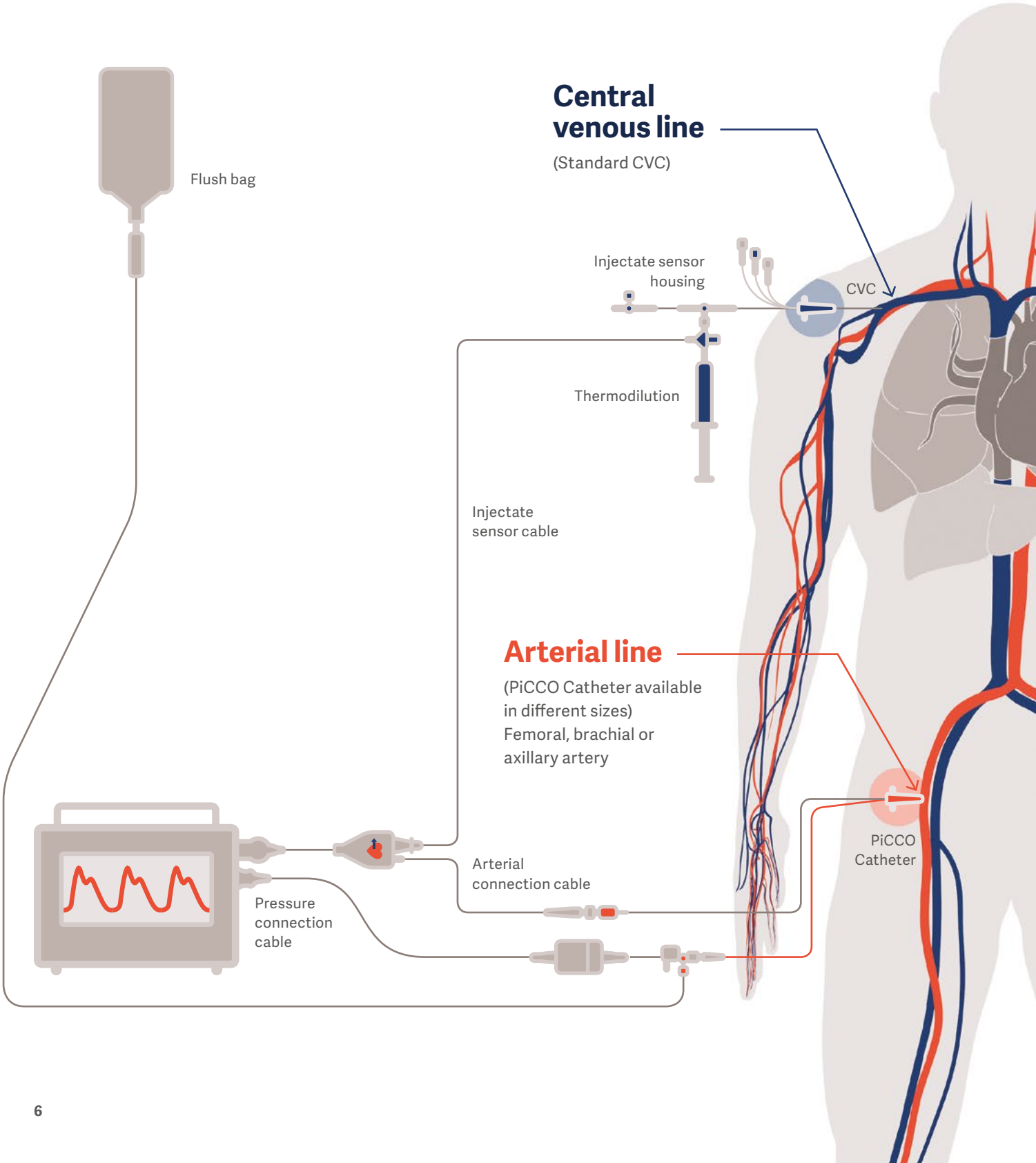
An increase in preload will, to a certain extent, lead to an increase in stroke volume (SV), based on optimal myocardial muscle fibre pre-stretching. Up to a certain limit, the more the sarcomeres of the muscle cells are stretched the greater the contraction. On the other hand, contractility may decrease in conditions of volume overload.

Hemodynamic parameters



Get the complete picture

How PiCCO Technology works



Two components of the PiCCO Technology

PiCCO Technology is based on two physical principles, namely, transpulmonary thermodilution and pulse contour analysis. Both principles allow the calculation of hemodynamic parameters and have been clinically tested and established for more than 20 years.[1, 2]

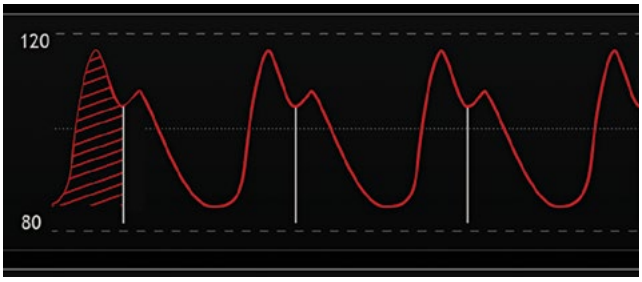
Arterial pulse contour analysis

The pulse contour analysis provides continuous information while transpulmonary thermodilution provides static measurements. Transpulmonary thermodilution is used to calibrate the continuous pulse contour parameters.

Transpulmonary thermodilution

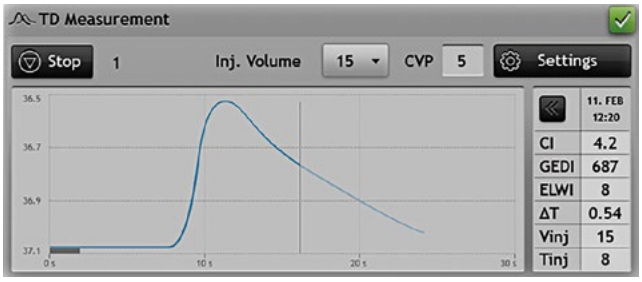
For the transpulmonary thermodilution measurement, a defined bolus (e. g. 15 ml cold normal saline) is injected via a central venous catheter.

The cold bolus passes through the right heart, the lungs and the left heart and is detected by the PiCCO Catheter, commonly placed in the femoral artery. This procedure should be repeated around three times in under 10 minutes to obtain an accurate average that is used to calibrate the device and to calculate the thermodilution parameters. These thermodilution parameters (i. e. they are updated only when the thermodilution procedure is performed) should be checked whenever there is a significant change in the patient's condition or therapy. It is recommended to calibrate the system at least 3 times a day.



Arterial pulse contour analysis

The shaded area below the systolic part of the pressure curve is proportional to the stroke volume.



Transpulmonary thermodilution

Pulse contour analysis

The theoretical basis of pulse contour analysis was established for the first time in 1899.[3]

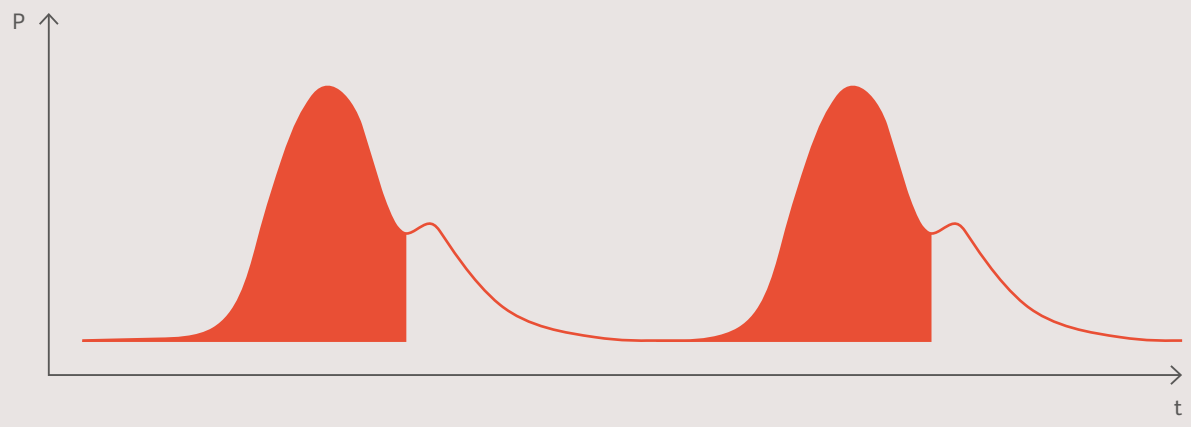
The basic idea was to use the analysis of the continuous arterial pressure signal to get more information than just the systolic, diastolic and mean value.

From a physiological perspective the arterial pressure curve provides information about the opening of the aortic valve (point of the increase of the systolic pressure) and when the aortic valve closes (incision in the pressure curve / the dicrotic notch). The time in between represents the duration of the systole and the area under the systolic part of the pressure curve directly reflects the stroke volume (SV), the amount of blood in milliliters which is ejected by the left ventricle with every heart beat.

However, the shape of the arterial pressure curve and the area under the curve is influenced not only by the stroke volume, but also by the individual compliance of the vascular system.

This is especially true of intensive care patients where a potentially rapid change in the vascular compliance occurs due to the disease process or medications. The individual calibration factor is determined with the initial calibration and needs to be updated regularly.[1, 4] In PiCCO Technology, this calculation factor is derived from the transpulmonary thermodilution measurement.

Analysis of the arterial pressure curve for the area under the systole



With the sophisticated algorithm, the stroke volume is calculated continuously and, by multiplying the stroke volume with the heart rate, a continuous cardiac output is derived, the pulse contour cardiac output (PCCO).[5]

$$PCCO = cal \times HR \times \int_{systole} \left(\frac{P(t)}{SVR} + C(p) \times \frac{dP}{dt} \right) dt$$

Patient-specific calibration factor (determined with thermodilution)

Area under the pressure curve

Shape of pressure curve

Heart rate

Compliance

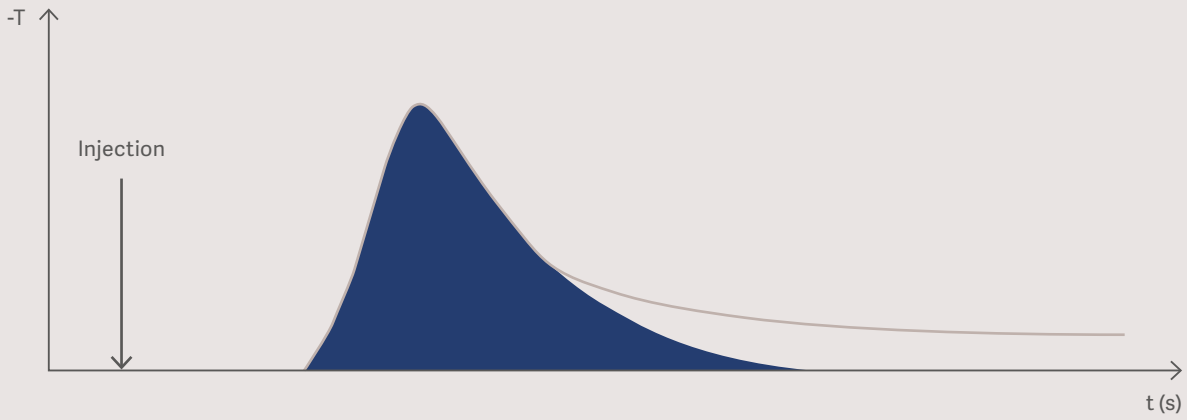
Basic formula to calculate pulse contour cardiac output (PCCO)

The PiCCO pulse contour algorithm has been extensively validated and has proved to be very reliable in daily critical care routine:

Overview of comparative studies on cardiac output measurement using PiCCO pulse contour and pulmonary arterial thermodilution [5–13]

Reference	Accuracy (l/min)	Standard deviation (l/min)	Regression coefficient
Felbinger TW et al., J Clin Anesth 2005	0.220	0.26	0.92
Della Rocca G et al., Can J Anesth 2003	0.080	0.72	–
Mielck F et al., JCVA 2003	-0.400	1.30	–
Felbinger TW et al., J Clin Anesth 2002	-0.140	0.33	0.93
Della Rocca G et al., BJA 2002	0.040	–	0.86
Rauch H et al., Acta Anaesth Scand 2002	0.140	1.16	–
Godje O et al., Med Sci Monit 2001	-0.020	1.20	0.88
Zollner C et al., JCVA 2000	0.310	1.25	0.88
Buhre W et al., JCVA 1999	0.003	0.63	0.93

Transpulmonary thermodilution



The CO is calculated from the area under the thermodilution curve[14–15]

$$CO = \frac{(T_b - T_i) \times V_i \times K}{\int \Delta T_b \times dt}$$

Blood temperature

Injectate temperature

Injecate volume

Correction constant*

Area under the thermodilution curve

* comprises specific weight and specific heat of blood and injectate fluid

The cardiac output (CO) is determined from the trans-pulmonary thermodilution.

The thermodilution curves are analysed and the CO is determined by using a modified Stewart-Hamilton algorithm.[14, 15] This method of calculating the cardiac output is used similarly by the right heart (pulmonary artery) catheter.

Overview of comparative studies on cardiac output measurement using transpulmonary and pulmonary arterial thermodilution

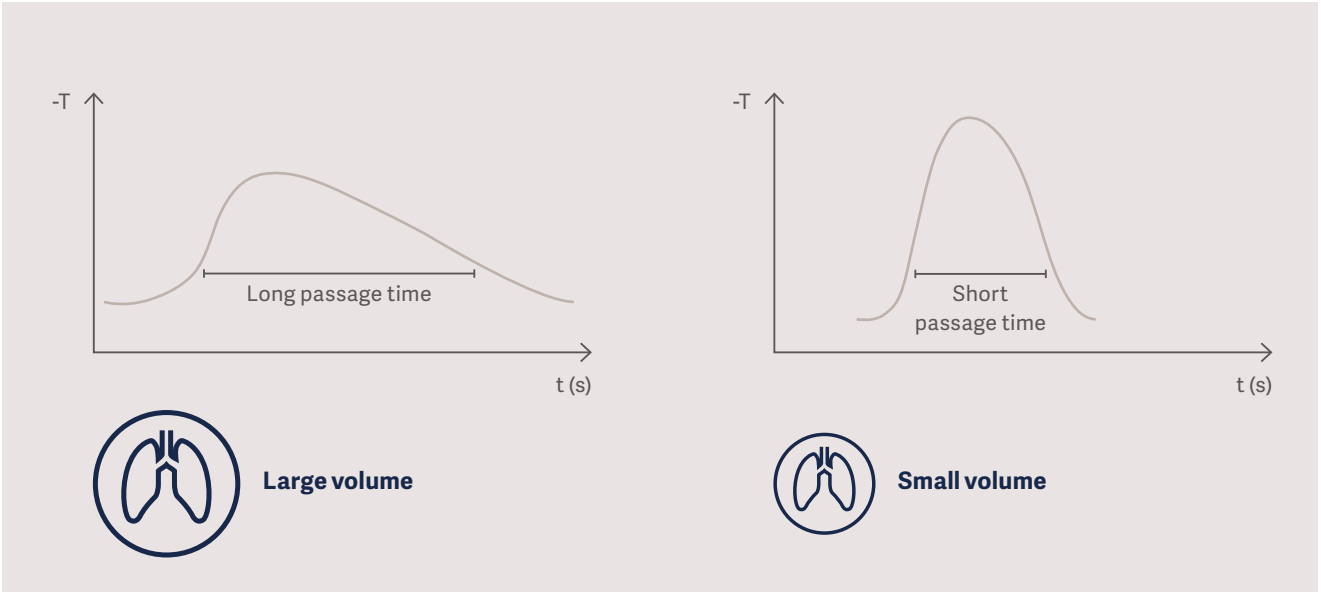
Clinical studies confirm the accuracy of the cardiac output values measured with transpulmonary thermodilution.[16]

Author	Patient group	Age	N	n	r	Bias (%)	Precision (%)
Della Rocca et al., 2002	Liver transplant	24–66	62	186	0.93	+1.90	11.0
Friesecke et al., 2009	Severe heart failure n. a.	29	325		ni	10.30	27.3 (PE*)
Goedje et al., 1999	Cardiac surgery	41–81	24	216	0.93	-4.90	11.0
Holm et al., 2001	Burns	19–78	23	109	0.97	-8.00	7.3
Kuntscher, 2002	Burns	21–61	14	113	0.81	ni	ni
Mc Luckie et al., 1996	Pediatrics	1–8	10	60	ni	+4.30	4.8
Segal et al., 2002	Intensive care	27–29	20	190	0.91	-4.10	10.0
von Spiegel et al., 1996	Cardiology	0.5–25	21	48	0.97	-4.70	12.0
Wiesenack et al., 2001	Cardiac surgery	43–73	18	36	0.96	+7.40	7.6
Zöllner et al., 1999	ARDS	19–25	18	160	0.91	-0.33	12.0

N = number of patients; n = number of measurements; r = regression coefficient; ni = not indicated
* PE= percentage error according to Critchley

An advantage of transpulmonary thermodilution is that it is independent from breathing or ventilatory cycles. Additionally, because the indicator passes through the heart and lungs, this allows the determination of intravascular and extravascular volumes inside the chest area, in particular, the preload volume and lung water.

Physiological principles



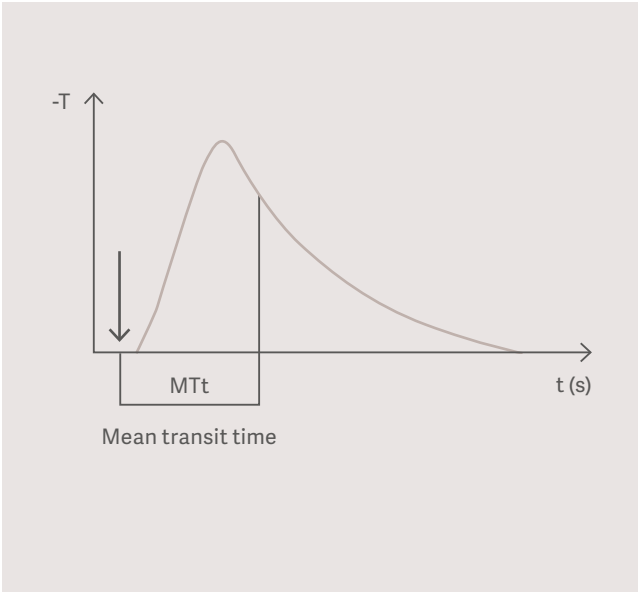
Large volume of intravascular and extravascular volumes

Small volume of intravascular and extravascular volumes

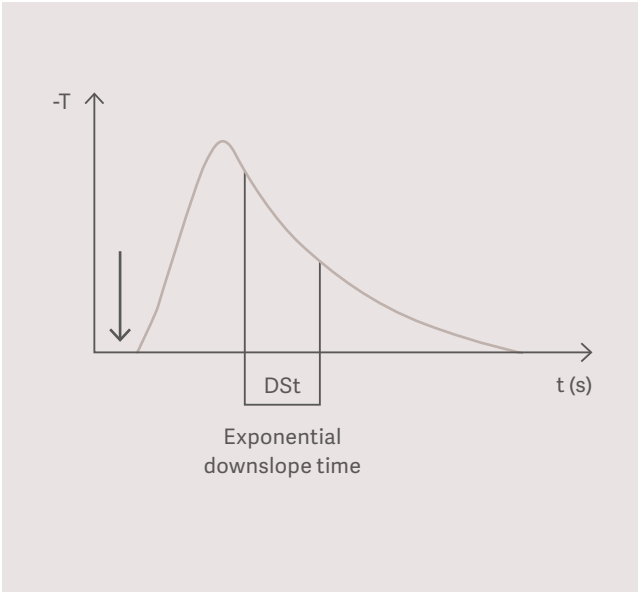
Assessment of volumes from transpulmonary thermodilution

The shape of the transpulmonary thermodilution curve is strongly influenced by the amount of intravascular and extravascular volume between the injection point (central venous) and detection point (central arterial). This means that the larger the volume amount in the chest, the longer the passage time of the indicator and vice versa. Determination of specific transit times of the thermal indicator thus enables quantification of specific volumes in the chest.

This analysis and calculation is based on a publication by Newman et al.[17] and has also been described by other authors.[18–24]



Determination of mean transit time



Determination of exponential downslope time

Mean transit time (MTt)

Mean transit time represents the time when half of the indicator passes the detection point (central artery). It is determined from the bisector of the area under the curve.

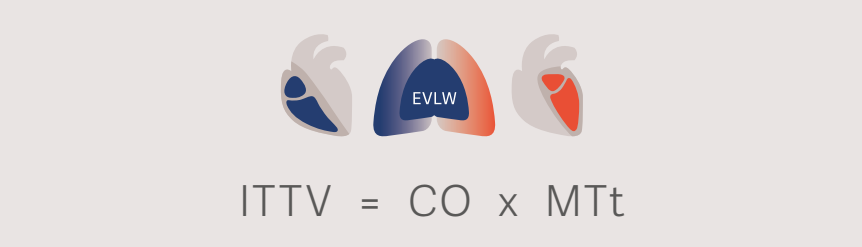
Exponential downslope time (DSt)

The exponential downslope time represents the wash-out function of the indicator.
It is calculated from the downslope part of the thermodilution curve.

Both mean transit time and exponential downslope time serve as the basis for calculation of the following volumes.

Intrathoracic thermal volume

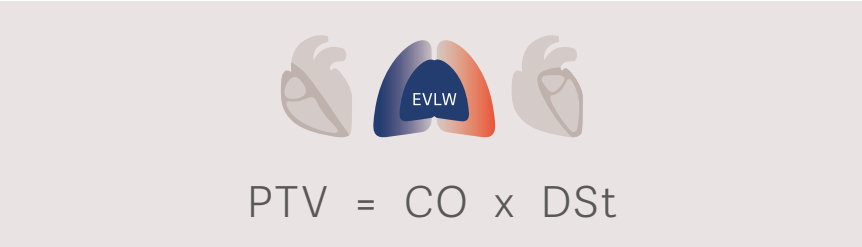
The multiplication of the mean transit time (MTt) with cardiac output (CO) represents the intrathoracic thermal volume (ITTV).



Scheme and calculation of the intrathoracic thermal volume (ITTV)

Pulmonary thermal volume

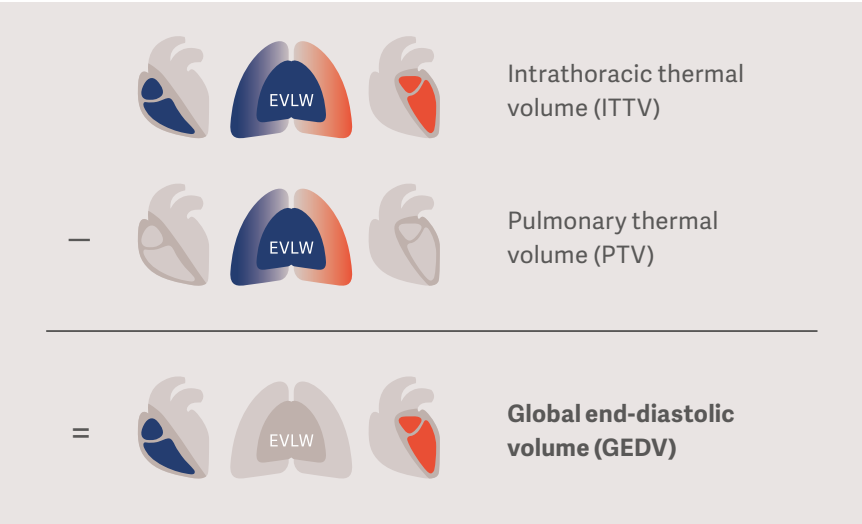
The exponential downslope time always characterises the volume of the largest mixing chamber in a row of mixing chambers. In the cardiopulmonary systems this is the lung. Thus the multiplication of the exponential downslope time (DSt) with the cardiac output (CO) represents the pulmonary thermal volume (PTV).



Scheme and calculation of the pulmonary thermal volume (PTV)

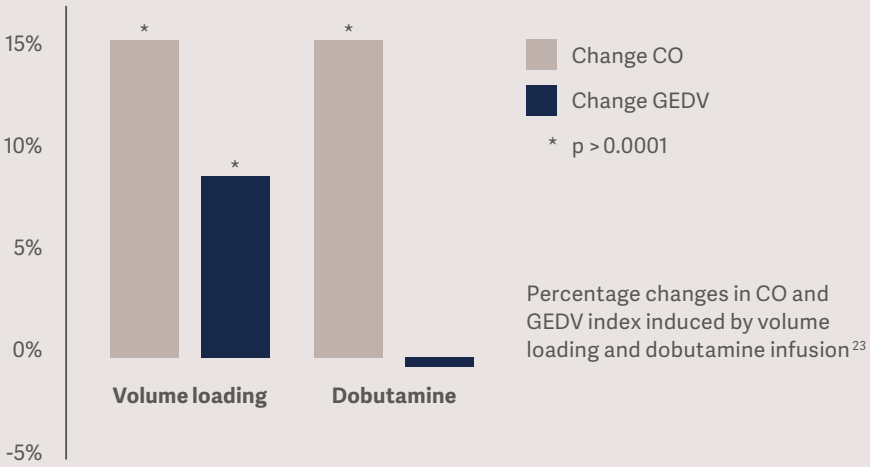
Quantification of the preload volume

By simply subtracting the pulmonary thermal volume from the intrathoracic thermal volume, the global end-diastolic volume (GEDV) is derived. GEDV indicates the level of preload volume.



Calculation of global end-diastolic volume (GEDV)

Cardiac output and transit times are derived from the same thermodilution signal. This raises the question of mathematical coupling, which has been investigated several times,[23] concluding that CO increases without a corresponding increase in GEDV.

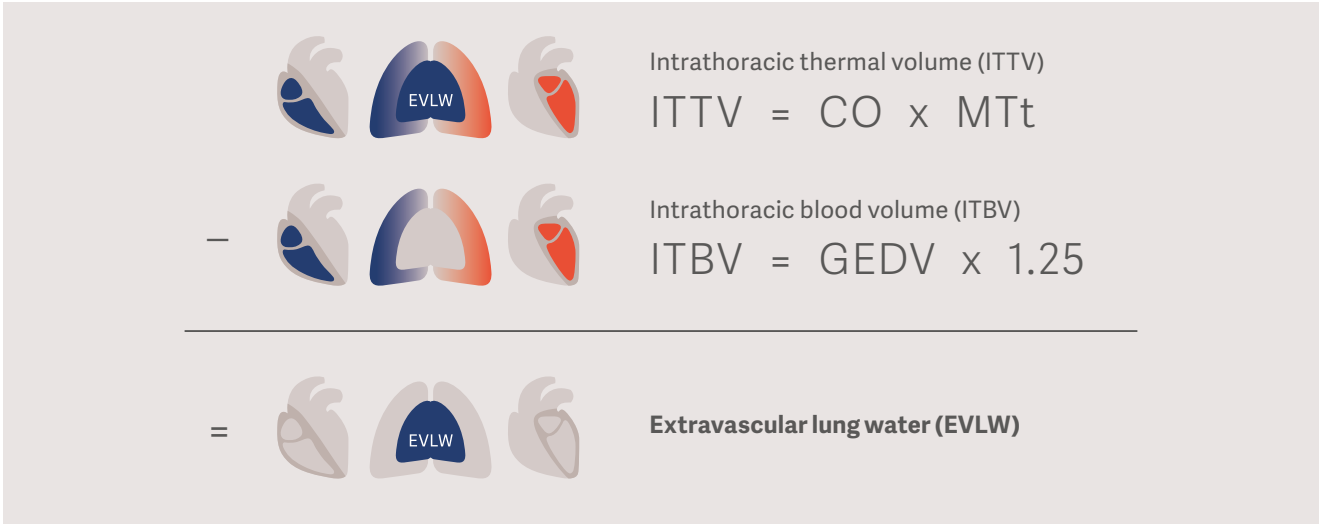


Percentage changes in CO and GEDV index induced by volume loading and dobutamine infusion[23]

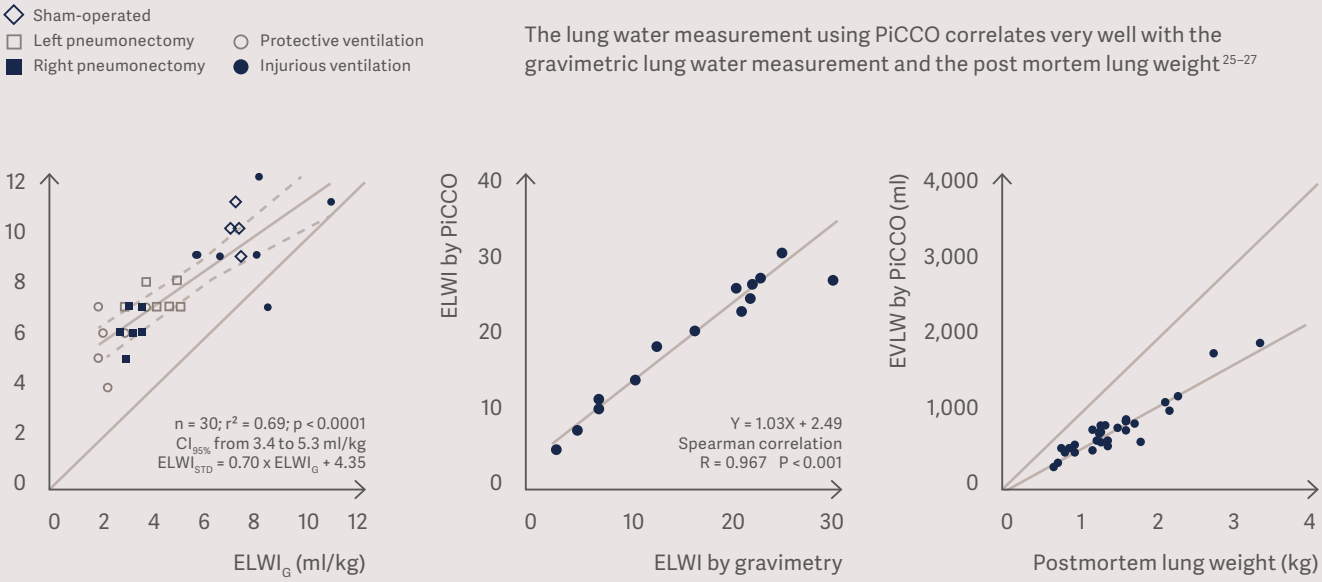
Quantification of a pulmonary edema

Using further calculations, the PiCCO Technology also provides quantification of the amount of pulmonary edema, expressed as extravascular lung water (EVLW). The only additional information required for this calculation is the amount of intravascular volume (ITBV). A clinical study using double-indicator dilution technology to measure ITBV and EVLW[24] found that intrathoracic blood volume is consistently 25% higher than the global end-diastolic volume. Thus, the intrathoracic blood volume can simply be calculated by multiplying the global end-diastolic volume with the factor 1.25. The calculated intrathoracic blood volume (ITBV) is then subtracted from the intrathoracic thermal volume (ITTV) to derive the extravascular lung water (EVLW).

Several validation studies comparing gravimetry and lung weight show that both this method and the introduction of the fixed factor for calculation of extravascular lung water show high accuracy.[25–27]



Calculation of extravascular lung water (EVLW)



PiCCO parameters

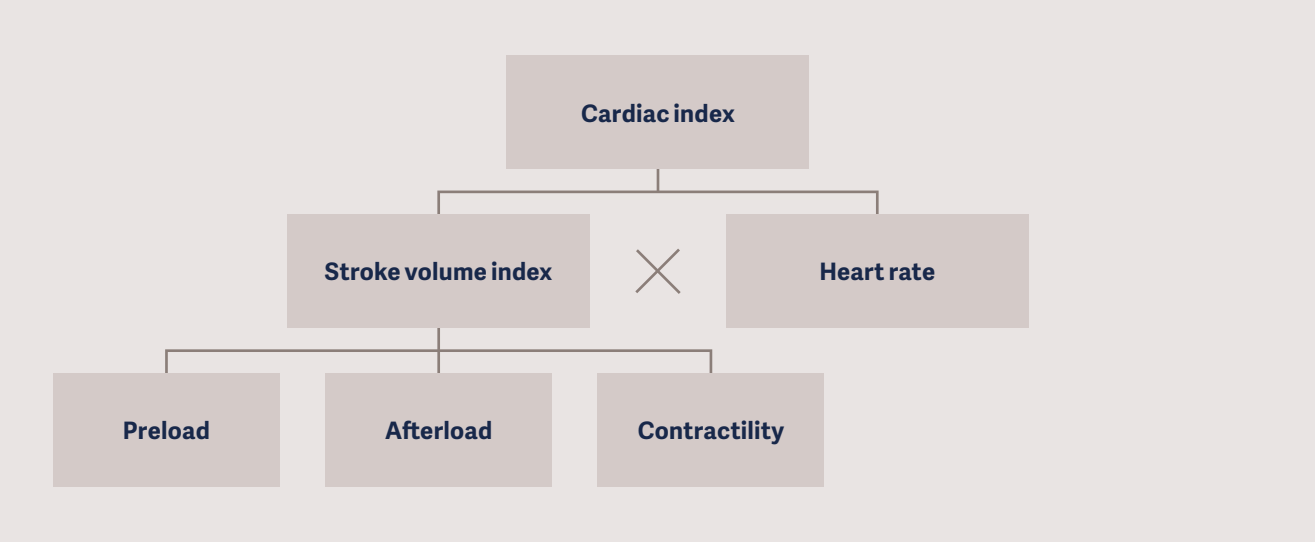
Cardiac index (CI), stroke volume index (SVI)

Cardiac index is the amount of blood pumped by the heart per minute indexed to 1 m² of the body surface area (BSA); the cardiac index represents the global blood flow. The PiCCO Technology provides discontinuous (transpulmonary thermodilution) and continuous (pulse contour analysis) parameters.

A decrease in cardiac index is a clear alarm signal and requires appropriate measures to manage the situation.

But knowledge about cardiac index alone is not enough to make a therapeutic decision, as the cardiac index is influenced by several factors. First of all it is the product of stroke volume and heart rate. Stroke volume is dependent on preload, afterload and contractility.

Thus, in addition to the cardiac index, further information on its determinants is required for appropriate treatment.



Cardiac index and its determinants

CI
3–5 l/min/m²

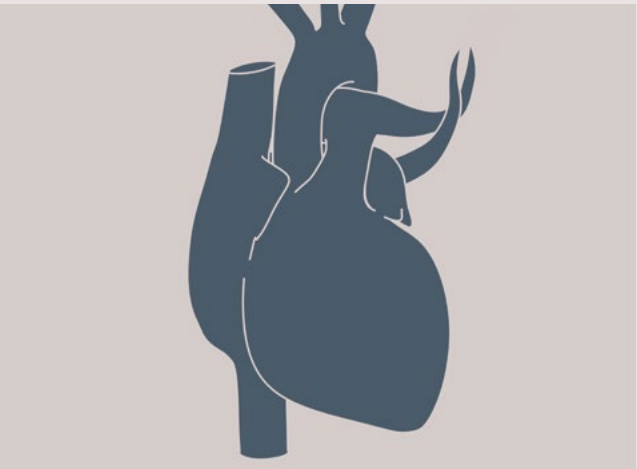
SVI
40–60 ml/m²

Preload

Global end-diastolic volume index (GEDI)
The preload is, along with afterload and contractility, one of the determinants of stroke volume and therefore cardiac output. Theoretically, it can be best described as the initial stretching of a single muscle cell of the heart prior to contraction, which means at the end of diastole. As this cannot be measured in vivo, other measurements have therefore to be substituted as estimates. In the clinical setting, preload is referred to as the end-diastolic pressure or (more precisely) end-diastolic volume.
A higher end-diastolic volume implies higher preload.

A higher central venous pressure (CVP) and / or a higher pulmonary capillary wedge pressure (PCWP) is still often regarded as an indicator of higher preload (CVP for the right heart, PCWP for the left heart). However, many studies have shown that CVP and PCWP are not reliable indicators for this purpose. This is mainly due to the limitation that pressure cannot be directly transferred into volume. So any volumetric parameter assessing the filling of the ventricle at the end of diastole reflects the actual preload more precisely.

In the clinical setting, preload is referred to as the end-diastolic pressure or (more precisely) end-diastolic volume.



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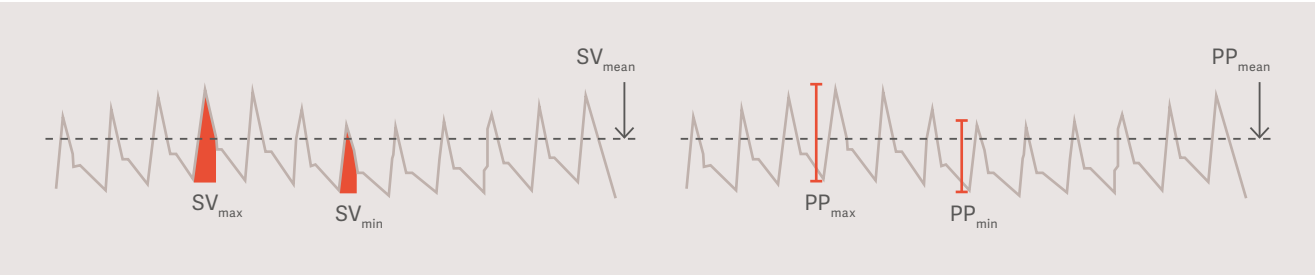
GEDI
680–800 ml/m²

Volume responsiveness



Stroke volume variation (SVV)

Pulse pressure variation (PPV)

Stroke volume variation (SVV) and pulse pressure variation (PPV)

The stroke volume variation (SVV) or pulse pressure variation (PPV) give – provided there is a continuously ventilated patient with a stable heart rhythm – information as to whether an increase in preload will also lead to an increase in stroke volume.

Mechanical ventilation induces cyclic changes in vena cava blood flow, pulmonary artery blood flow and aortic blood flow. At the bedside, changes in the aortic blood flow are reflected by swings in the blood pressure curve (and thus variations in stroke volume and blood pressure). The magnitude of these variations is highly dependent on the volume responsiveness of the patient. With controlled ventilation, the rise in intrathoracic pressure during early inspiration leads to a squeezing of the pulmonary blood into the left ventricle. This process in turn increases the left ventricular preload. With a volume responsive patient, this results in an increased stroke volume or pulse pressure.

An increase in intrathoracic pressure also results in reduced right ventricular filling. With a volume responsive right heart, this will reduce the volume ejected. Thus, during late inspiration a couple of heartbeats later, the left ventricular preload will decrease as will the stroke volume or pulse pressure. The variations in stroke volume and pulse pressure can be analysed over a 30 second time frame by the following formula:

$$SVV = \frac{(SV_{max} - SV_{min})}{SV_{mean}}$$

$$PPV = \frac{(PP_{max} - PP_{min})}{PP_{mean}}$$

The higher the variation the more likely the patient is to be volume responsive. For proper use of the parameters, the following preconditions must be fulfilled:

- Fully controlled mechanical ventilation with a tidal volume ≥ 8 ml/kg PBW (predicted body weight)
- Sinus rhythm
- Pressure curves free of artifacts

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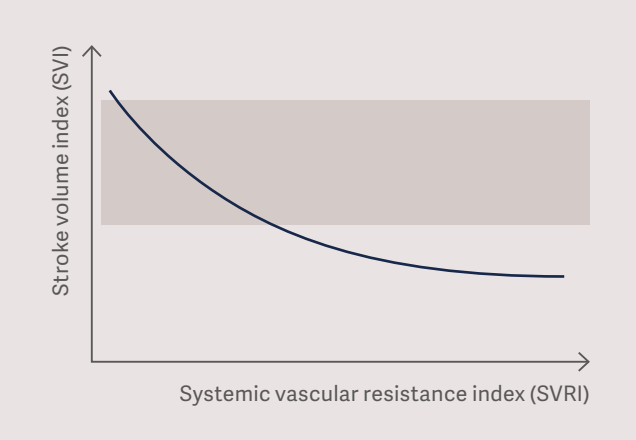
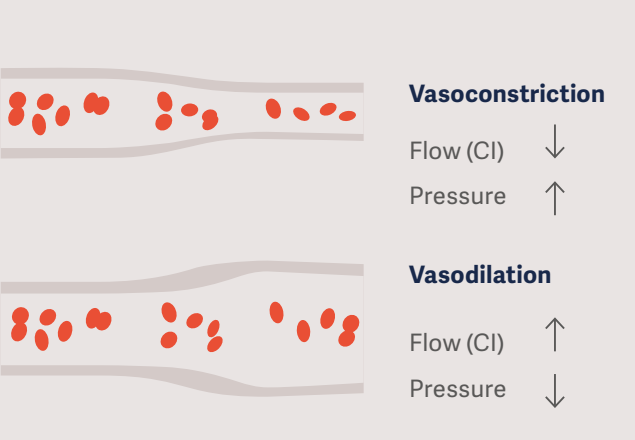
SVV/PPV

< 10%

Afterload





Systemic vascular resistance index (SVRI)

The afterload is another determinant of stroke volume / cardiac output. The physiological meaning of SVRI is the tension or pressure that builds up in the wall of the left ventricle during ejection. Following Laplace's law, the tension upon the muscle fibers in the heart wall is the product of the pressure within the ventricle and the ventricle radius, divided by the ventricle wall thickness.

In the clinical context things are often simplified and so the afterload is seen as the resistance the heart has to pump against; the systemic vascular resistance index (SVRI) is the parameter that represents this.

- If the afterload (SVRI) is increased, the heart must pump with more power to eject the same amount of blood as before.
- The higher the afterload, the less the cardiac output.
- The lower the afterload, the higher the cardiac output.

If the afterload exceeds the performance of the myocardium, the heart may decompensate.

$$SVRI = \left[\frac{(MAP - CVP)}{CI} \right] \times 80$$

OK

SVRI

1,700–2,400 dyn*s*cm^{-5}*m^2

Contractility

Contractility influences cardiac output.

Contractility of the myocardium represents the ability of the heart to contract independent of the influence of preload or afterload. Substances that cause an increase in intracellular calcium ions lead to an increase in contractility. Different concentrations of calcium ions in the cell lead to different degrees binding between the actin (thin) and myosin (thick) filaments of the heart muscle. Direct determination of cardiac contractility is not possible in the clinical setting. Therefore, surrogate parameters are used to evaluate or estimate the contractility.

Global ejection fraction (GEF)

Ejection fraction represents the percentage of volume in a heart chamber which is ejected with a single contraction. The measurement of the global ejection fraction offers a complete picture of the overall cardiac contractility.

$$GEF = \frac{4 \times SV}{GEDV}$$

Cardiac function index (CFI)

The cardiac function index can be used to estimate cardiac contractility. It represents the relation of the flow (cardiac output) and the preload volume (GEDV). Thus, cardiac function index is a preload related cardiac performance parameter.

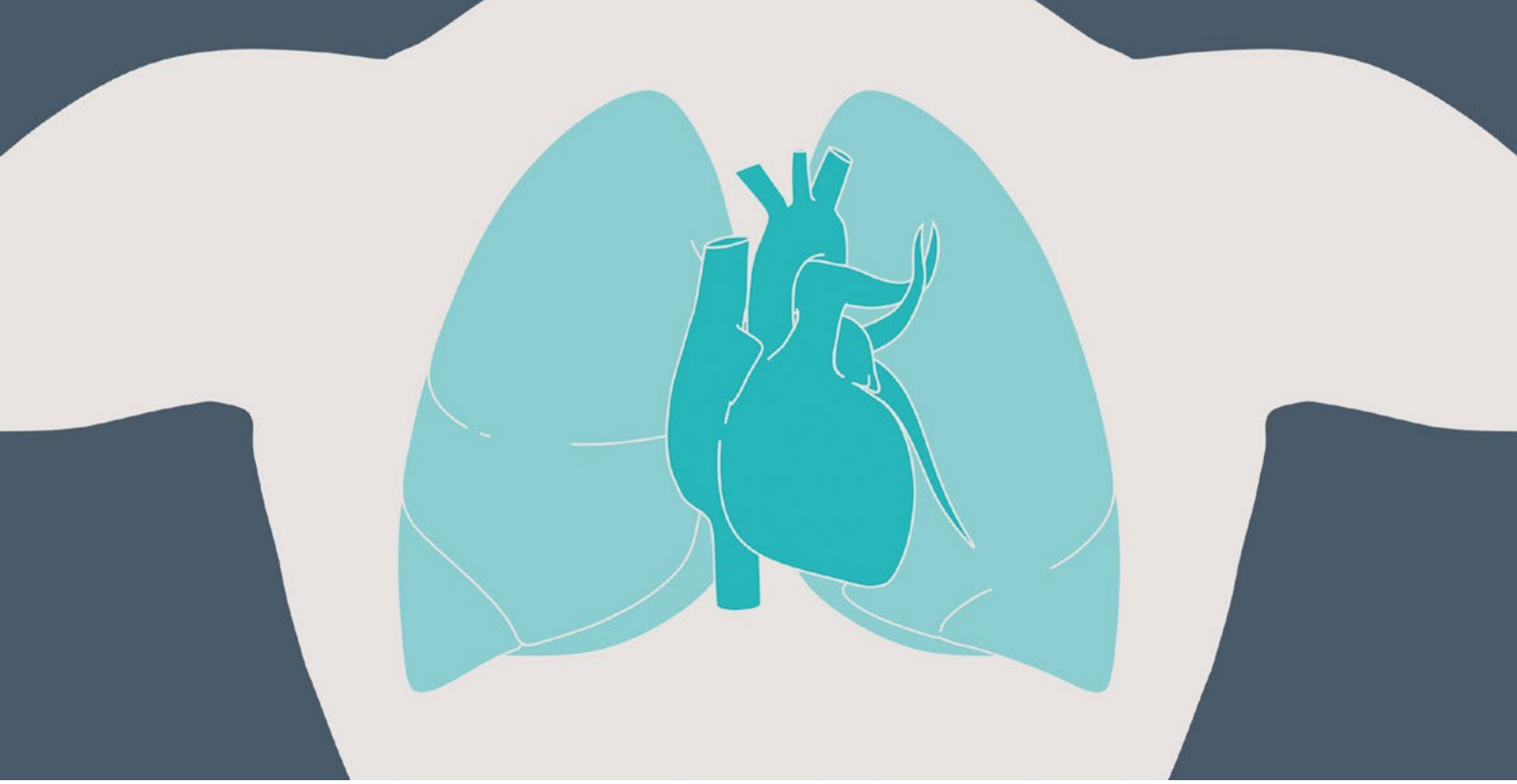
$$CFI = \frac{CI_{TD} \times 1{,}000}{GEDV}$$

Cardiac power index (CPI)

CPI represents the power of left ventricular cardiac output in watts. It is the product of pressure (MAP) and flow (CO). In clinical studies it has been found to be the strongest independent predictor of hospital mortality in cardiogenic shock patients.[28, 29]

$$CPI = CI_{PC} \times MAP \times 0.0022$$

GEF	CFI	CPI
25–35%	4.5–6.5 1/min	0.5–0.7 W/m²



Left ventricular contractility (dPmx)

From the arterial pressure curve, the pressure changes during the systolic phase can be analysed and a measure of the pressure increase over time (analysed in speed) is calculated. The steeper the upslope of the curve, the higher the contractility of the left ventricle.

As the upslope also depends on the individual compliance of the aorta, the parameter should primarily be viewed and evaluated as part of the overall trend.

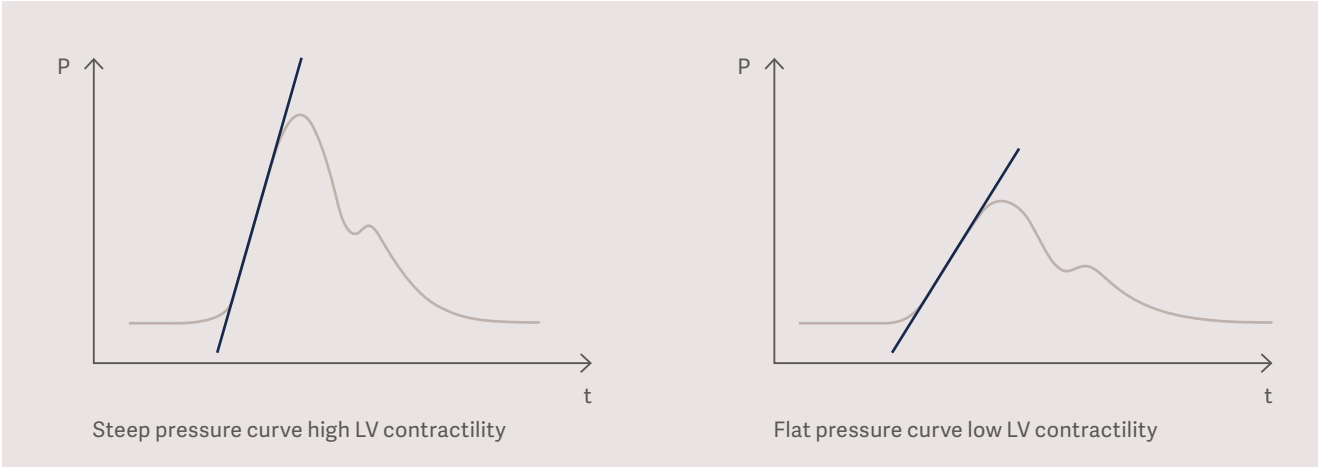


Diagram of steep / flat pressure increase with high / low contractility

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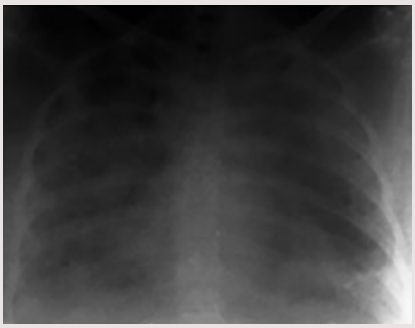
dPmx

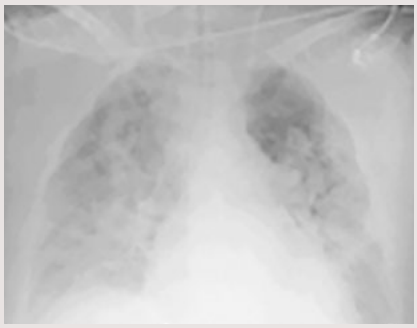
mmHg/s trend information

Assessment of pulmonary edema using PiCCO Technology

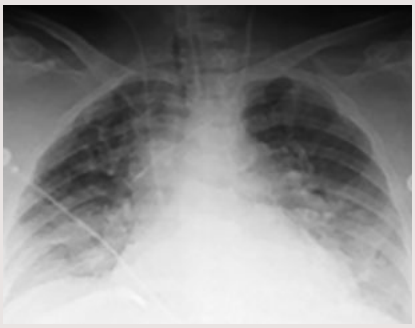
Examples of chest X-rays that do not reflect the level of pulmonary edema



ELWI = 21 ml/kg
Severe pulmonary edema



ELWI = 14 ml/kg
Moderate pulmonary edema



ELWI = 8 ml/kg
No pulmonary edema

Extravascular lung water index (ELWI)

A pulmonary edema is characterized by an accumulation of fluid in the interstitium of the lung tissue and / or the alveoli. This leads to impaired gas exchange and may even cause pulmonary failure. The amount of the pulmonary edema can easily be quantified at the bedside by measuring the extravascular lung water index (ELWI). The usual clinical signs of pulmonary edema (white-out on the chest X-ray, low oxygenation index, decreased lung compliance) are non-specific and only indicative when the edema is at an advanced stage. In the critical care routine, the chest X-ray is often used to estimate pulmonary edema in patients at risk. This approach is imperfect as the chest X-ray provides only a black and white density image of all components in the chest, including gas volume, blood volume, pleural effusion, bones, muscles, lung tissue, fat, skin edema and also pulmonary edema.

A more advanced approach using extravascular lung water provides a systematic route to therapeutic options. Extravascular lung water is indexed to the body weight in kg, written as the extravascular lung water index (ELWI). By indexing to the patient's predicted body weight (PBW), underestimation of lung water, particularly in obese patients, is avoided.

ELWI
3–7 ml/kg

Pulmonary vascular permeability index (PVPI)

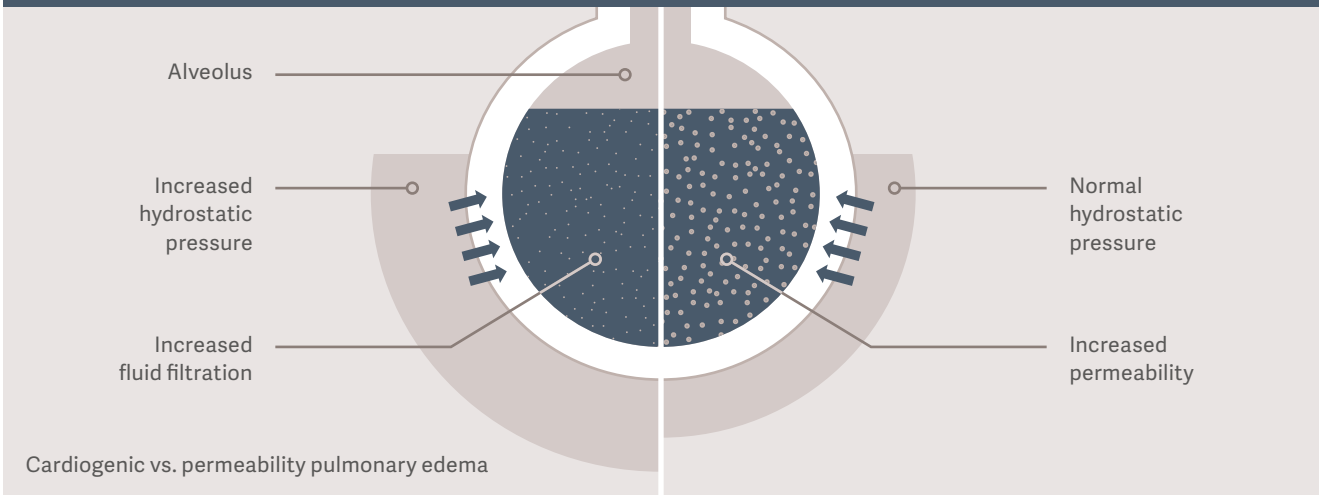
When pulmonary edema is present (measured using extravascular lung water), the next important question is: What is the reason for the pulmonary edema? In general there are two main sources of pulmonary edema:

Cardiogenic pulmonary edema

Caused by intravascular fluid overload, hydrostatic pressure increases. This causes fluids to leak into the extravascular space.

Permeability pulmonary edema

Vascular permeability is increased by an inflammatory reaction caused, for example, by sepsis. This leads to the increased transfer of fluids, electrolytes and proteins from the intravascular to the extravascular space, even with a normal to low intravascular fluid status and hydrostatic pressure.



Cardiogenic vs. permeability pulmonary edema

A differential diagnosis of the pulmonary edema is important because the therapeutic approach is quite different. In cardiogenic pulmonary edema, a negative fluid balance is sought, while in cases of permeability pulmonary edema treating the cause of inflammation has priority. The pulmonary vascular permeability index (PVPI) enables this differential diagnosis. This parameter is calculated from the relation between extravascular lung water (EVLW) and pulmonary blood volume (PBV). A PVPI value in the range of 1 to 3 points to a cardiogenic pulmonary edema, while a PVPI value greater than 3 suggests a permeability pulmonary edema.

OK

PVPI

< 3 cardiogenic edema / ≥ 3 permeability edema

Indications and clinical benefits

PiCCO indications

PiCCO Technology is indicated in patients who present with unstable hemodynamics and unclear volume status and in situations of therapeutic conflict. These situations are usually present in:

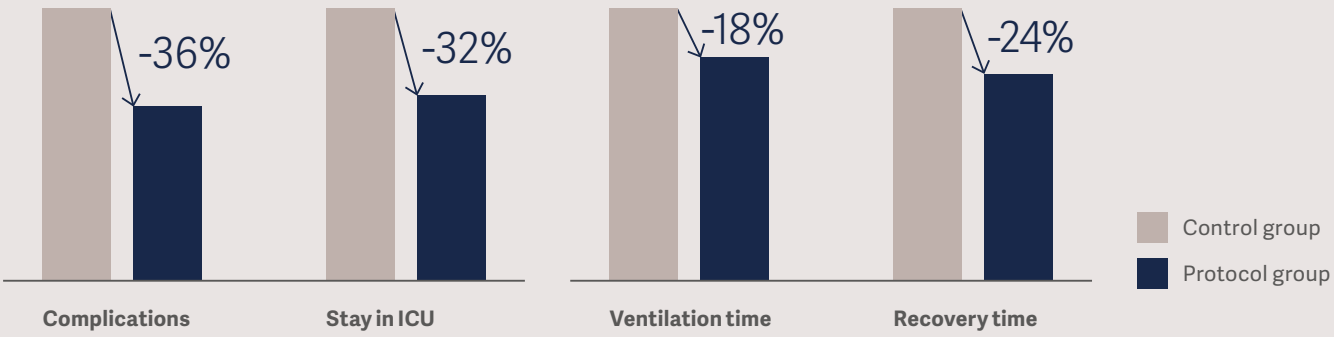
- Septic shock
- Cardiogenic shock
- Traumatic shock
- ARDS
- Severe burn injuries
- Pancreatitis
- High risk surgical procedures

Medical benefits

Monitoring does not lower patient mortality or morbidity. Per se, however, it provides valuable information which should be used to set up a treatment plan and apply goal-directed therapy to the patient as early as possible. The success of early goal-directed therapy (EGDT) is documented and clearly shows the following advantages:

- Reduction in ventilation time
- Reduction of ICU stay
- Reduction in complications
- Reduction of recovery time

Goal-directed therapy based on validated information improves outcomes.



Preload – GEDV (Göpfert et al. 2013 [30])

Preload – GEDV (Göpfert et al. 2007 [31])

Overview of technologies and further parameters

Along with PiCCO Technology, Pulsion has other innovative technologies that may be used with the PulsioFlex Monitoring Platform.

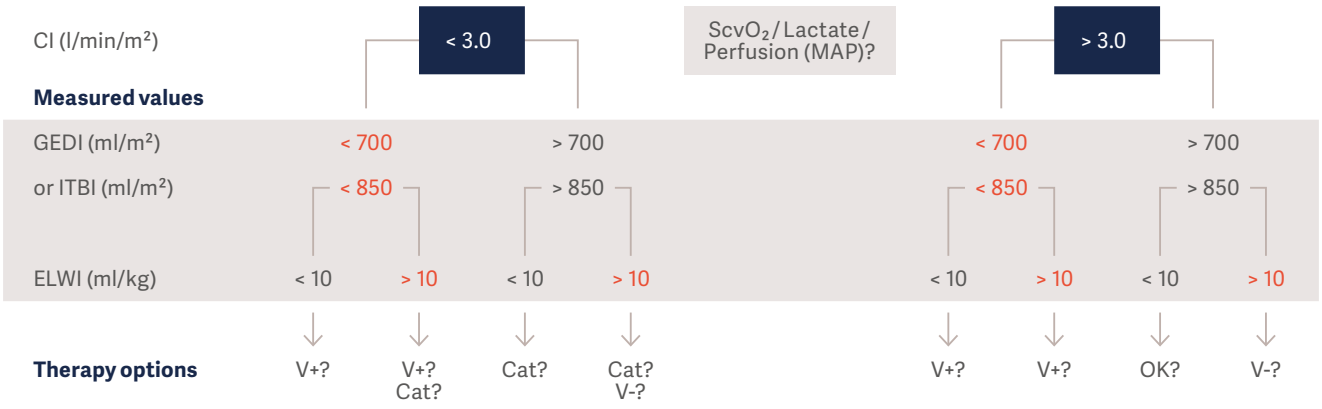
The PulsioFlex Monitor is equipped with the ProAQT Technology. You can easily extend the hemodynamic spectrum with modules featuring PiCCO, CeVOX, and LiMON technologies. In the future, additional innovations will be integrated in the technology portfolio of the PulsioFlex Platform. The following table lists the parameters available with the current modules:

Method		PiCCO	ProAQT	CeVOX	LiMON
Pulse contour analysis (continuous)	**Flow**	CI_{PC*}, SVI	$CI_{Trend/Cal**}$, SVI		
	Contractility	dPmx, CPI	dPmx, CPI		
	Afterload	SVRI	SVRI		
	Volume responsiveness	SVV, PPV	SVV, PPV		
Thermodilution (discontinuous)	**Flow**	CI_{TD***}			
	Preload	GEDI			
	Contractility	CFI, GEF			
	Pulmonary edema	ELWI, PVPI			
Oxymetry	**Oxygen saturation**			$ScvO_2$	
ICG elimination	**Liver function**				PDR, R15

* Cardiac index derived from pulse contour ** Calibrated from internal or external reference value
*** Cardiac index derived from thermodilution

Hemodynamic decision model

This decision model is not obligatory. It cannot replace the individual therapeutic decisions of the treating physician.



Targeted values

GEDI (ml/m²) (if ELWI > 10 → 700–800)	> 700
GEF (%)	> 25
CFI (1/min)	> 5
ELWI (ml/kg) (slow response)	≤ 10

Volume responsiveness?
(Passive leg raising / endexpiratory occlusion test / volume challenge / SVV / PPV?)

Contractility problem?
(GEF / CFI / echo?)

V+ = volume loading, V- = volume withdrawal , Cat = catecholamine / cardiovascular agents
Please reevaluate your clinical decisions and the set target parameters.

OEM partner modules

Patient centered flexibility

The versatile PiCCO Technology has been developed to match and adapt to different clinical settings.

To allow easy integration into your existing product portfolio, Getinge is partnering with various monitoring companies like GE, Philips, Dräger, Mindray and Nihon Kohden.

Getinge modules can be implemented seamlessly with negligible footprint and maintaining the already familiar user interface. All while relying on the clinically well proven and documented advantages of the PiCCO Technology.

All OEM partner modules are fully compatible with the original PiCCO disposable products.

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Hand in hand for better patient care

Getinge is built on a genuine compassion for people's health, safety, and wellbeing. Founded in 1904 with roots dating back to 1838, Getinge has grown organically and fostered partnerships to become a global market leader.

Getinge's portfolio offers solutions and support throughout the clinical pathway, and features recognizable and reliable brands, such as Maquet and Pulsion.

Ours is a legacy of trust, and an ongoing commitment to advancing medical technology. We maintain close global partnerships with clinical leaders to address real-world clinical needs. We help you protect patients, proactively avoid complications, and prevent common causes of escalating health-care costs.

As a global leader in medical technology, Getinge has the firsthand experience to improve everyday life for people – today and tomorrow.

GETINGE

Pulsion Medical Systems SE · Hans-Riedl-Str. 17 · 85622 Feldkirchen · Germany · +49 89 459914-0 · zentrale.pulsion@getinge.com

www.getinge.com

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