PiCCO - Technology

Haemodynamic Monitoring at the Highest Level

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

2012
PulsioFlex + PiCCO Module

2007
PiCCO_2

2002
PiCCO plus

1997
PiCCO classic
CONTENT

Basics of haemodynamic monitoring
How the PiCCO technology works
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Basics of haemodynamic monitoring

Monitoring cardio-circulatory function is of major importance in all intensive care patients.

Monitoring with standard parameters: ECG non-invasive blood pressure and pulse oxymetry provides insufficient information for deciding on the adequacy of treatments. Only advanced haemodynamic monitoring with invasive measurement of cardiac output and its determinants (preload, afterload, contractility) as well as the quantification of pulmonary oedema allows for early goal directed therapy.
Haemodynamic parameters

Oxygen Supply
DO₂ I

Oxygen Consumption
VO₂ I

Central Venous Oxygen Saturation
ScvO₂

Cardiac Output
CI

Stroke Volume
SVI

Heart Rate
HR

Preload
GEDI, SVV, PPV

Afterload
SVRI

Contractility
GEF, CFI, dPmx

Pulmonary Oedema
ELWI, PVPI

Liver Function
PDR IC G

O₂ Uptake
O₂ Transport
O₂ Extraction
O₂ Utilisation

Volume
Vasopressors
Inotropes
Blood Transfusion

Arterial Oxygen Content
O₂ Saturation
SaO₂

Haemoglobin
Hb

Oxygen Supply
O₂ Transport
O₂ Extraction
O₂ Utilisation

Oxygen Consumption
O₂ Uptake
O₂ Transport
O₂ Extraction
O₂ Utilisation
The PiCCO technology requires a central venous catheter and the PiCCO catheter which has a temperature sensor at the tip.

**How PiCCO technology works**

**A. axillaris**
- PICCO catheter 4F 8 cm

**A. brachialis**
- PICCO catheter 4F 16 cm
- PICCO catheter 4F 22 cm

**A. femoralis**
- PICCO catheter 5F 20 cm
- PICCO catheter 3F 7 cm (paediatrics)

**A. radialis**
- PICCO catheter 4F 50 cm

PICCO technology is less invasive than the placement of a right heart catheter which is placed in the pulmonary artery. In addition to the PiCCO catheter, a central venous access is required. However, for most critical care patients this is standard care.

Fig. Recommended placement of PICCO catheter
Two components of the PiCCO technology

The PiCCO technology is based on two physical principles, namely transpulmonary thermodilution and pulse contour analysis. Both principles allow the calculation of haemodynamic parameters and have been clinically tested and established for more than 20 years\(^\text{(1,2)}\).

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 (for example 15mls 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 ensure an accurate average 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 per day.
Pulse contour analysis

The theoretical basis of pulse contour analysis was published for the first time in 1899. 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 point of view, the arterial pressure curve provides information about when the aortic valves open (moment of the increase of the systolic pressure) and also 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 mls which is ejected by the left ventricle with every single heart beat.

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

This is especially true in intensive care patients where a potentially rapid change in the vascular compliance occurs due to the disease process or due to medications. An individual calibration factor is determined with the initial calibration and needs to be updated regularly.

In the PiCCO technology, this calculation factor is derived from the transpulmonary thermodilution measurement.
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)\(^5\).

**Fig. Analysis of the arterial pressure curve for the area under the systole**

![Pressure Curve](image)

**Fig. Basic formula to calculate Pulse Contour Cardiac Output (PCCO)**

\[
P_{CCO} = \text{cal} \times \text{HR} \times \int_{\text{systole}} \left( \frac{P(t)}{SVR} + \frac{C(p) \times dP}{dt} \right) dt
\]

- Patient-specific calibration factor (determined with thermodilution)
- Heart rate
- Area under the pressure curve
- Compliance
- Shape of pressure curve

The PiCCO pulse contour algorithm is extensively validated and has proved to be very reliable in daily clinical routine:

**Overview of comparative studies on cardiac output measurement using PiCCO pulse contour and pulmonary arterial thermodilution\(^5\)-13**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Accuracy (l/min)</th>
<th>Standard deviation (l/min)</th>
<th>Regression coefficient</th>
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<tr>
<td>Felbinger TW et al., J Clin Anesth 2005</td>
<td>0.22</td>
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<td>Della Rocca G et al., Can J Anesth 2003</td>
<td>0.080</td>
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<td>Mielck F et al., JCVA 2003</td>
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<td>Felbinger TW et al., J Clin Anesth 2002</td>
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<td>Della Rocca G et al., BJA 2002</td>
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<td>Rauch H et al., Acta Anaesth Scand 2002</td>
<td>0.14</td>
<td>1.16</td>
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<td>Godje O et al., Med Sci Monit 2001</td>
<td>-0.020</td>
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<td>Zoliner C et al., JCVA 2000</td>
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<td>Buhre W et al., JCVA 1999</td>
<td>0.003</td>
<td>0.63</td>
<td>0.93</td>
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</table>
The cardiac output (CO) is determined from the transpulmonary thermodilution. The thermodilution curves are analysed and the CO is determined by using a modified Stewart-Hamilton algorithm \(^{(14,15)}\). This way of calculating the cardiac output is also used in a similar way by the right heart (pulmonary artery) catheter.

The CO is calculated from the area under the thermodilution curve

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

- \(T_b\) = Blood temperature
- \(T_i\) = Injectate temperature
- \(V_i\) = Injectate volume
- \(\int \Delta T_b \times dt\) = Area under the thermodilution curve
- \(K\) = Correction constant; comprises specific weight, blood and injectate temperature
Clinical studies confirm the accuracy of the cardiac output values measured with transpulmonary thermodilution. 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.

<table>
<thead>
<tr>
<th>Author</th>
<th>Patient group</th>
<th>Age</th>
<th>N</th>
<th>n</th>
<th>r</th>
<th>Accuracy</th>
<th>Variation</th>
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<td>Liver transplant</td>
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<td>von Spiegel et al., 1996</td>
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<td>Zöllner et al., 1999</td>
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<td>18</td>
<td>160</td>
<td>0.91</td>
<td>-0.33%</td>
<td>12.0%</td>
</tr>
</tbody>
</table>

*PE= Percentage error according to Critchley

\[ N = \text{Number of patients}; n = \text{Number of measurements}; r = \text{Regression coefficient}; ni = \text{not indicated} \]
Physiological principles

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. and has also been described by other authors.

Large volume

Small volume

Fig. Large volume of intravascular and extravascular volumes

Fig. Small volume of intravascular and extravascular volumes
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 Down-Slope time (DSt)

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

Both mean transit time and exponential down-slope 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).

\[ \text{ITTV} = \text{CO} \times \text{MTt} \]

Pulmonary thermal volume

The exponential downslope time always characterises the volume of the largest mixing chamber in a row of mixing chambers. In the cardio-pulmonary 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).

\[ \text{PTV} = \text{CO} \times \text{DSt} \]

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.

\[ \text{ITTV} - \text{PTV} = \text{GEDV} \]

As both cardiac output and the transit times are derived from the same thermodilution signal, this raises the question of mathematical coupling. This topic has been investigated several times \(^{23}\), clearly showing that CO increases without any corresponding increase in GEDV.

\[ \text{GEDV} \]

*Fig. Scheme and calculation of the intrathoracic thermal volume*

*Fig. Scheme and calculation of the pulmonary thermal volume*

*Fig. Calculation of global end-diastolic volume (GEDV)*

*Fig. Percentage changes in CO and GEDV index induced by volume loading and dobutamine infusion \(^{23}\)*
Quantification of a pulmonary oedema

Using further calculations, the PICCO technology also provides quantification of the amount of pulmonary oedema, expressed as Extravascular Lung Water (EVLW). The only additional information required for this calculation is the amount of intravascular volume (ITBV). In a clinical study using double-indicator dilution technology to measure ITBV and EVLW\(^{(26)}\), it was 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).

\[
\text{ITTV} = \text{CO} \times \text{MTt}
\]

\[
\text{ITBV} = \text{GEDV} \times 1.25
\]

\[
\text{EVLW} = \text{ITTV} - \text{ITBV}
\]

Fig. Calculation of Extravascular Lung Water (EVLW)

As 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 are very accurate \(^{(25-27)}\).
Cardiac Index (CI), Stroke Volume Index (SVI)

Cardiac index is the amount of blood pumped by the heart per minute indexed to the body surface area (BSA); the cardiac index represents the global blood flow. The PiCCO technology provides discontinuously (transpulmonary thermodilution) and continuously (pulse contour analysis). A decrease in cardiac index is a clear alarm signal and requires appropriate measures to improve 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.

\[
\text{Cardiac Index (CI)} = \frac{\text{Stroke Volume Index (SVI)} \times \text{Heart rate}}{} 
\]

Cardiac Index parameters:
- **CI_{PC}**
  - 3 - 5 l/min/m²
- **SVI**
  - 40 - 60 ml/m²

Fig. Cardiac index and its determinants
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 is 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 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 directly be transferred into volume. So any volumetric parameter assessing the filling of the ventricle at the end of diastole reflects more precisely the actual preload.

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. 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. 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.

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

![Frank-Starling curve](image)

*Fig. Schematic Frank-Starling curve for verification of the preload status
A = Optimal preload, B = Volume responsive, C = Volume overload*
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:

\[
SVV = \frac{(SV_{\text{max}} - SV_{\text{min}})}{SV_{\text{mean}}}
\]

\[
PPV = \frac{(PP_{\text{max}} - PP_{\text{min}})}{PP_{\text{mean}}}
\]

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*
- Sinus rhythm
- Pressure curves free of artifacts

*PBW - Predicted Body Weight
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.

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

- 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.
Contractility

Contractility is another factor that influences cardiac output. Contractility of the myocardium represents the ability of the heart to contract independent of the influence from 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 a different degree of 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.

**Globale 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.

\[
\text{GEF} = \frac{4 \times \text{SV}}{\text{GEDV}}
\]

**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.

\[
\text{CFI} = \frac{\text{ClTD} \times 1000}{\text{GEDV}}
\]

**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.\(^{28,29}\)

\[
\text{CPI} = \text{Cl}_{pc} \times \text{MAP} \times 0.0022
\]

**Values**

- **GEF**: 25 - 35 %
- **CFI**: 4.5 - 6.5 l/min
- **CPI**: 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](image)

**Fig. Diagram of steep/flat pressure increase with high/low contractility**

- **dPmx**
  - 900 - 1200 mmHg/s (healthy heart)
Assessment of pulmonary oedema using PiCCO technology

Extravascular Lung Water Index (EVLW)

A pulmonary oedema is characterised 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 oedema can easily be quantified at the bedside by measuring the extravascular lung water index (ELWI). The usual clinical signs of pulmonary oedema (white-out on the chest x-ray, low oxygenation index, decreased lung compliance) are non-specific and only reliable later when the pulmonary oedema may already be advanced.

In the clinical routine, the interpretation of the chest x-ray is most often used to estimate the amount of pulmonary oedema in patients at risk. This approach is very complex as the chest x-ray only gives a black & white density image of all components in the chest, including gas volume, blood volume, pleural effusion, bones, muscles, lung tissue, fat, skin oedema and also pulmonary oedema.

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.

Severe pulmonary oedema
Moderate pulmonary oedema
No pulmonary oedema

Fig. Examples of chest x rays that do not reflect the level of pulmonary oedema
Pulmonary Vascular Permeability Index (PVPI)

When pulmonary oedema is present (measured using Extravascular Lung Water), the next important question is: What is the reason for the pulmonary oedema?

In general there are two main sources of pulmonary oedema:

1. **Cardiogenic pulmonary oedema**

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

2. **Permeability pulmonary oedema**

   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.

A differential diagnosis of the pulmonary oedema is important because the therapeutic approach is quite different. In cardiogenic pulmonary oedema, a negative fluid balance is sought, while in cases of permeability pulmonary oedema 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 oedema, while a PVPI value greater than 3 suggests a permeability pulmonary oedema.
Along with the PiCCO technology, PULSION has other innovative technologies that may be used with the PulsioFlex monitoring platform. As standard the monitor is equipped with the ProAQT technology. You can easily extend this haemodynamic scope 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:

<table>
<thead>
<tr>
<th>Method</th>
<th>ProAQT</th>
<th>PiCCO</th>
<th>CeVOX</th>
<th>LiMON</th>
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<td>• Flow</td>
<td>Cl_{\text{inst}} SVI</td>
<td>Cl_{\text{PC}} SVI</td>
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<td>• Contractility</td>
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<td>• Afterload</td>
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<td>• Volume responsiveness</td>
<td>SVV, PPV</td>
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<td></td>
</tr>
<tr>
<td>• Oxygen saturation</td>
<td>ScvO_{2}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ICG elimination</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Liver function</td>
<td></td>
<td>PDR, R15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Haemodynamic Decision Model

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

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<table>
<thead>
<tr>
<th>CI (l/min/m²)</th>
<th>Measured Values</th>
<th>Therapy Options</th>
<th>Targeted Values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;3.0</td>
<td>V+? V+? Cat? V+?</td>
<td>1. GEDI (ml/m²) or ITBI (ml/m²)</td>
</tr>
<tr>
<td></td>
<td>&gt; 3.0</td>
<td>V+? V+? V-?</td>
<td>2. Optimise SVV (%)*</td>
</tr>
<tr>
<td>GEDI (ml/m²)</td>
<td>&lt; 700</td>
<td>Cat? V+? Cat?</td>
<td>GEF (%) or CFI (1/min)</td>
</tr>
<tr>
<td>or ITBI (ml/m²)</td>
<td>&lt; 850</td>
<td>Cat? V+? Cat?</td>
<td>ELWI (ml/kg) (slow response)</td>
</tr>
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<td>ELWI (ml/kg)</td>
<td>&lt; 10</td>
<td>Cat? V+? Cat?</td>
<td>≤ 10</td>
</tr>
<tr>
<td></td>
<td>&gt; 10</td>
<td>Cat? V+? Cat?</td>
<td>≤ 10</td>
</tr>
<tr>
<td></td>
<td>&gt; 700</td>
<td>Cat? V+? Cat?</td>
<td>≤ 10</td>
</tr>
<tr>
<td></td>
<td>&gt; 850</td>
<td>Cat? V+? Cat?</td>
<td>≤ 10</td>
</tr>
<tr>
<td></td>
<td>&gt; 700</td>
<td>Cat? V+? Cat?</td>
<td>≤ 10</td>
</tr>
<tr>
<td></td>
<td>&gt; 850</td>
<td>Cat? V+? Cat?</td>
<td>≤ 10</td>
</tr>
</tbody>
</table>

V+ = volume loading  V- = volume reduction  Cat = catecholamine / cardiovascular agents

*SVV is only applicable in fully ventilated patients without cardiac arrhythmia
Medical and economic benefits

Goal directed therapy based on validated information improves the outcome.

Monitoring per se does not lower patient mortality or morbidity. However, it provides valuable information which should be used to set up a treatment plan and thus apply goal-directed therapy to the patient as early as possible. The success of Early Goal Directed Therapy (EGDT) is documented in studies that clearly show the following advantages:

- Reduction in ventilation time
- Reduction of ICU stay
- Reduction in complications
- Reduction in medication requirement

![Graphs showing reduction in complications, stay in ICU, ventilation time, and recovery time.]

Economic aspects

The medical benefit which comes with reduced stay in hospital and less complications leads to an increase in the occupancy rate of the ICU beds. This in turn increases the patient turnover, resulting in strong economic benefits for the hospital.

Unpublished data from Klinikum Bogenhausen, Munich/Germany
**Literature**

14. Stewart GN. Researches on the circulation time and on the influences which affect it. J Physiol 1897; 22 (5): 159-83
15. Hamilton WF et al. Further analysis of the injection method, and of changes in haemodynamics under physiological and pathological conditions. Studies on the Circulation 1931; 534-551
The PiCCO technology was introduced into the market in 1997 by the Munich based company PULSION Medical Systems and has been continuously enhanced since then. PULSION has more than 20 years of experience in haemodynamic monitoring.

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

Today the PiCCO technology is the established standard for advanced haemodynamic monitoring, confirmed by the modular integration into the monitors of the world market leaders for patient monitoring including Philips/Dixtal, Dräger, GE & Mindray. The PiCCO technology is applied more than 140,000 times per year in more than 60 countries.