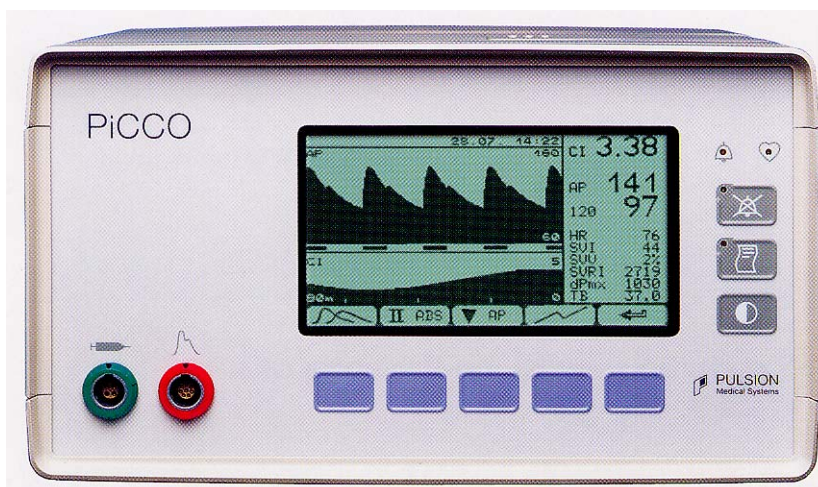




How does an **arterial line** measure

- ◆ Preload
- ◆ Afterload
- ◆ Cardiac Contractility
- ◆ Continuous Cardiac Output &
- ◆ Lung water?



Read on



**PULSION  
Pacific**

## **PiCCO Monitor**

### **Table of Contents**

<b>1</b>	<b>INTRODUCTION.....</b>	<b>3</b>
<b>2</b>	<b>INTERMITTENT VOLUMETRIC THERMODILUTION.....</b>	<b>4</b>
2.1	PRINCIPLES OF CARDIAC OUTPUT DETERMINATION.....	4
2.2	PRINCIPLES OF VOLUME CALCULATION.....	5
2.2.1	<i>Mean Transit Time (MTt) and Intrathoracic Thermal Volume (ITTV).....</i>	<i>5</i>
2.2.2	<i>Exponential Downslope Time (DSt) and Pulmonary Thermal Volume (PTV).....</i>	<i>6</i>
2.3	PARAMETERS OBTAINED BY ARTERIAL THERMODILUTION.....	7
2.3.1	<i>Cardiac Output Arterial (COa).....</i>	<i>7</i>
2.3.2	<i>Global End Diastolic Volume (GEDV).....</i>	<i>8</i>
2.3.3	<i>Intrathoracic Blood Volume.....</i>	<i>10</i>
2.3.4	<i>Extravascular Lung Water (EVLW).....</i>	<i>11</i>
2.3.5	<i>Cardiac Function Index (CFI).....</i>	<i>13</i>
<b>3</b>	<b>PULSE CONTOUR ANALYSIS.....</b>	<b>14</b>
3.1	PRINCIPLE OF MEASUREMENT.....	14
3.2	CALIBRATION OF PULSE CONTOUR ANALYSIS.....	15
3.3	PARAMETERS OBTAINED BY PULSE CONTOUR ANALYSIS.....	15
3.3.1	<i>Pulse Contour Cardiac Output (PCCO).....</i>	<i>16</i>
3.3.2	<i>Arterial Blood Pressure (AP).....</i>	<i>16</i>
3.3.3	<i>Stroke Volume Variation (SVV).....</i>	<i>17</i>
3.3.4	<i>Systemic Vascular Resistance (SVR).....</i>	<i>17</i>
3.3.5	<i>Index Of Left Ventricular Contractility (dP/dTMAX).....</i>	<i>17</i>

## 1 INTRODUCTION

Only measurement of clinically relevant hemodynamic parameters results in correct diagnosis and appropriate therapy in critically ill patients. Today, intravascular pressure and cardiac output (C.O.) monitoring are frequently performed in the operating room and intensive care unit. Currently, C.O. is mostly measured intermittently although continuous measurement would be preferable. Continuous measurement of cardiac output appears to be a significant improvement in hemodynamic monitoring of critically ill patients. The ideal method for continuous cardiac output should be as safe as possible, easy to use and be applicable to all critically ill patients both adults and children. A majority of current techniques for measuring continuous cardiac output are complex, cumbersome and expensive. The most common technique for measuring continuous C.O. is thermodilution utilizing a heating pulmonary artery catheter (PAC).

Compared to this method, estimation of C.O. from the arterial pulse contour is less invasive and produces a real "beat to beat" C.O. Additionally, pulse contour cardiac output (PCCO) is easily applicable in critically ill patients both adult and paediatric populations.

The arterial pulse contour method for measuring cardiac output was originally described by Otto Frank in 1899. Since then, a variety of pressure contour equations for estimating beat to beat stroke volume have been developed.

The PiCCO is a device for continuous cardiac output measurement combined with cardiac preload volume and lung water monitoring without the need for a pulmonary artery catheter. The PULSION PiCCO computes the C.O. utilizing an improved arterial pulse contour analysis algorithm. Pulse contour cardiac output (PCCO) is calibrated by means of an arterial thermodilution measurement. A bolus of cold normal saline or 5% dextrose in water is injected through any central venous catheter. A thermodilution curve is recorded by an arterial thermodilution catheter that also serves as an arterial line. In addition to calibration of the PCCO, arterial thermodilution also yields cardiac preload volume and an estimate of both, intrathoracic blood volume (ITBV) and extravascular lung water. The following parameters can be derived by arterial thermodilution:

		Absolute Parameters		Indexed Parameters	
Arterial Thermodilution	Parameter	Abbr.	Unit	Abbr.	Unit
	Cardiac output, arterial	C.O.a	l/min	Cl <sub>a</sub>	l/min/m <sup>2</sup>
	Cardiac function index	CFI	1/min		
	Global end diastolic volume	GEDV	ml		
	Intrathoracic blood volume	ITBV	ml	ITBVI	ml/m <sup>2</sup>
	Extravascular lung water	EVLW	ml	EVLWI	ml/kg

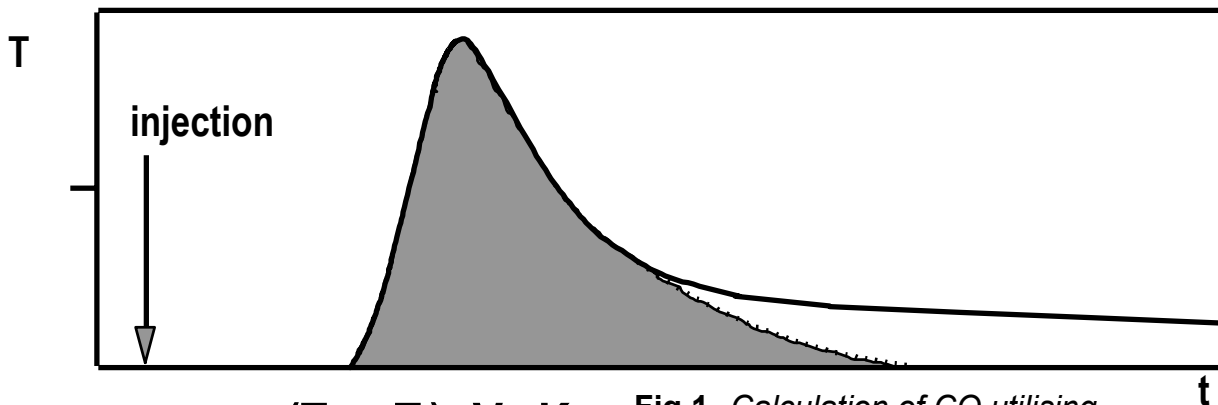
**After initial calibration the following parameters can continuously be derived by pulse contour analysis:**

		Absolute Parameters		Indexed Parameters	
Pulse Contour	Parameter	Abbr.	Unit	Abbr.	Unit
	Pulse contour cardiac output	PCCO	l/min	PCCI	l/min/m <sup>2</sup>
	Systolic arterial blood pressure	APsys	mmHg		
	Diastolic arterial blood pressure	APdia	mmHg		
	Mean arterial blood pressure	MAP	mmHg	ITBVI	ml/m <sup>2</sup>
	Heart rate	HR	1/min		
	Stroke volume	SV	ml	SVI	ml/m <sup>2</sup>
	Stroke volume variation	SVV	%		
	Systemic vascular resistance	SVR	dyn/sec/cm <sup>-5</sup>	SVRI	dyn/sec/cm <sup>-5</sup> /m <sup>2</sup>
Index of left ventricular contractility	DP/dtmax	mmHg/s			

## 2 INTERMITTENT VOLUMETRIC THERMODILUTION

### 2.1 Principles Of Cardiac Output Determination

Cardiac output (C.O.) is generally determined using the Stewart-Hamilton method. To accomplish thermodilution determination, a known volume of cold (at least 10<sup>0</sup>C lower than blood temperature) solution is injected intravenously as fast as possible. The recorded downstream temperature change is dependent on the flow and on the volume through which the cold indicator has passed. As a result, a thermodilution curve can be constructed. The PiCCO detects the cold indicator in the arterial system (preferably in the femoral artery). Cardiac output (C.O.) by thermodilution is calculated as follows:



$$C.O. = \frac{(T_b - T_i) \cdot V_i \cdot K}{\int \Delta T_b \cdot dt}$$

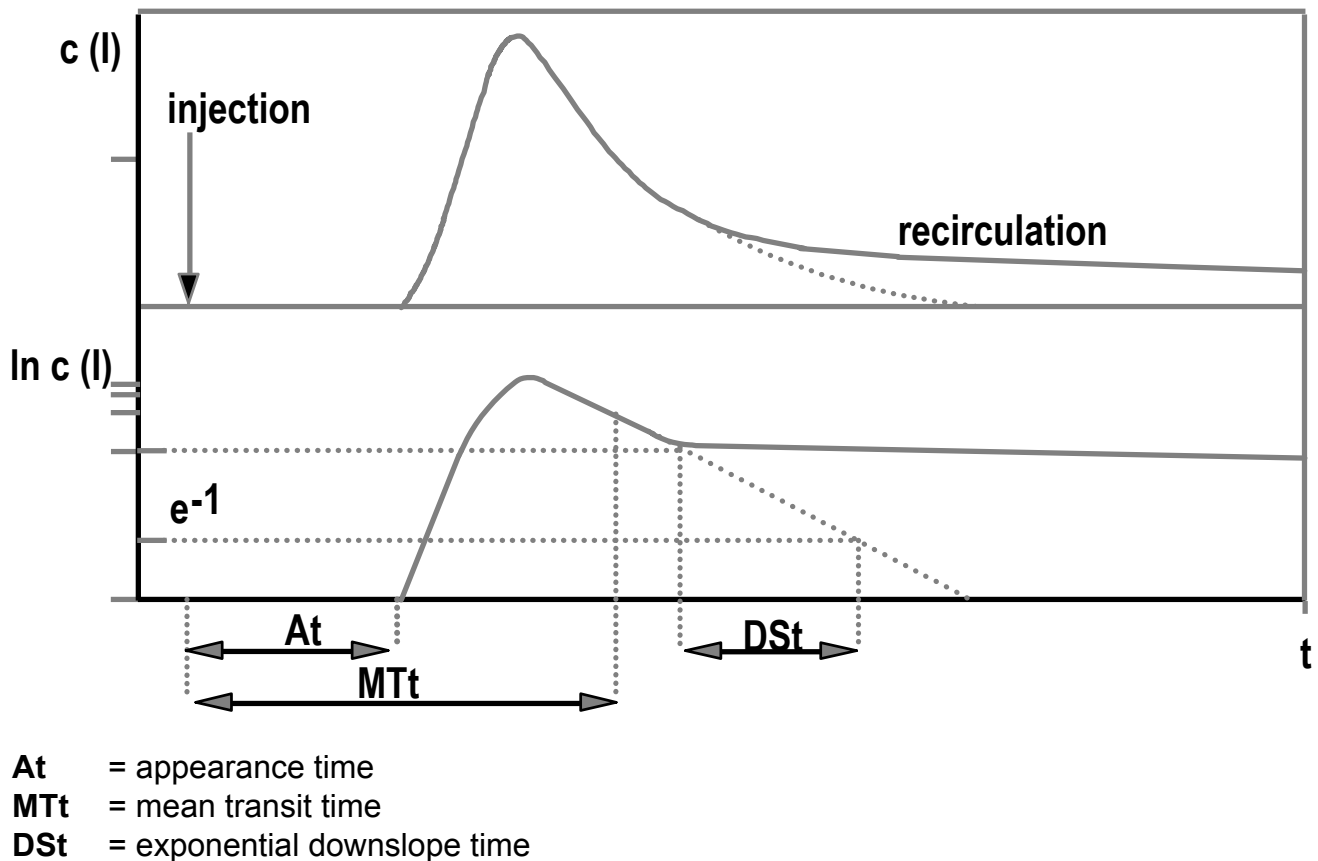
Fig 1 Calculation of CO utilising the Stewart-Hamilton Equation

Where

- T<sub>b</sub> = Blood temperature before the injection of cold bolus
- T<sub>i</sub> = Temperature of the injection solution (injectate)
- V<sub>i</sub> = Injectate volume
- ∫ ΔT<sub>b</sub> • dt = Area under the thermodilution curve
- K = Correction constants, made up of specific weights and specific heat of blood and injectate

## 2.2 Principles Of Volume Calculation

Specific volumes can be calculated by multiplying cardiac output with characteristic time variables of the thermodilution curve. The PULSION PiCCO determines the **mean transit time (MTt)** of the thermodilution curve as well as the **exponential downslope time (DSt)**.



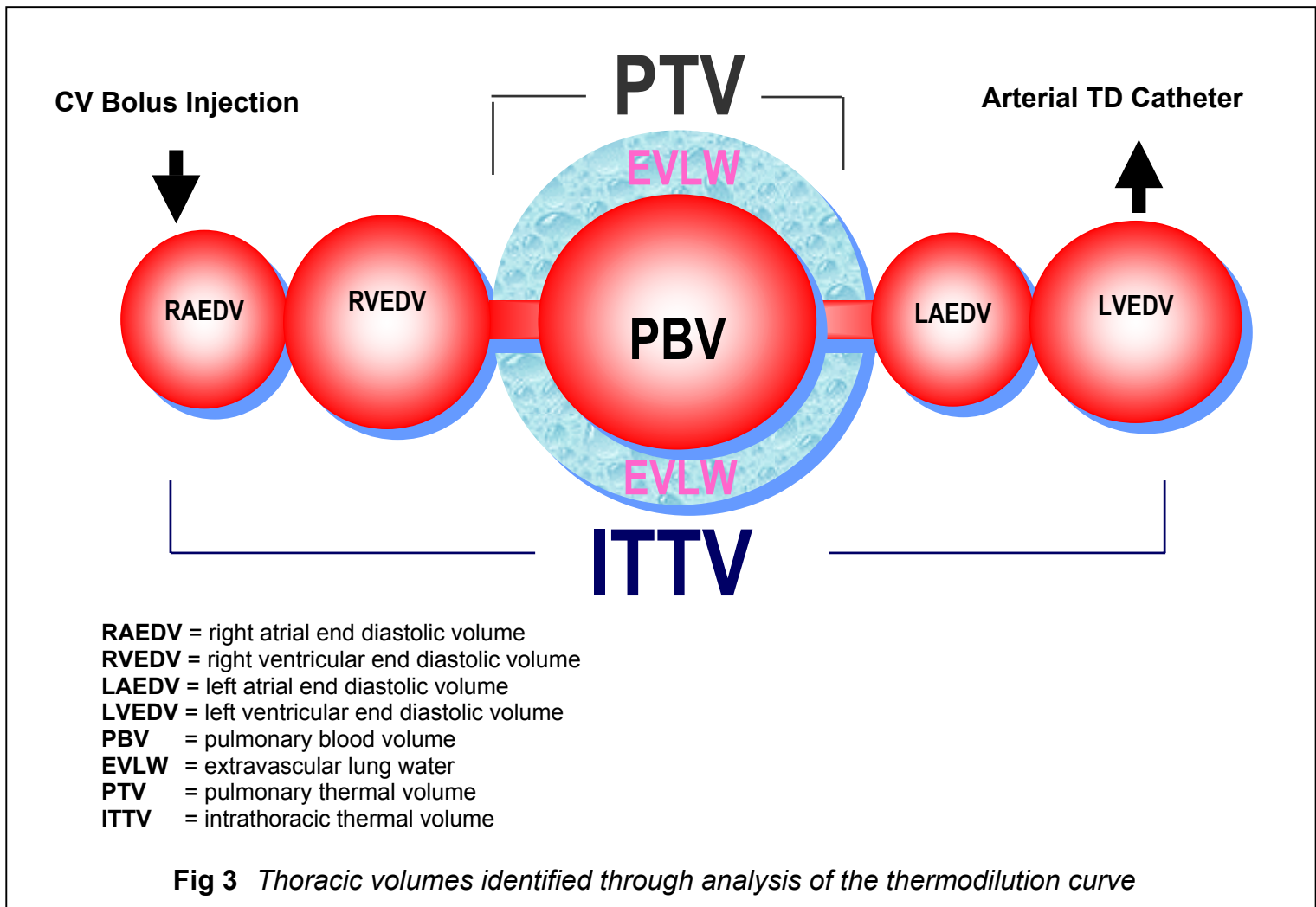
**Figure 2** Analysis of the thermodilution curve to determine thoracic volumes

### 2.2.1 Mean Transit Time (MTt) and Intrathoracic Thermal Volume (ITTV)

MTt is the mean transit time of the cold indicator from the site of injection to the site of detection. The product of mean transit time and cardiac output represent the volume transversed by the relevant indicator, ie total volume between the sites of injection and detection. When applied to femoral arterial thermodilution this volume is known as the intrathoracic thermal volume (ITTV). The ITTV represents the pulmonary blood volume (PBV), extravascular lung water (EVLW) and end diastolic volumes (EDV) of the atria and ventricles (refer to fig 3).

### 2.2.2 Exponential Downslope Time (DSt) and Pulmonary Thermal Volume (PTV)

DSt is the exponential downslope time of the arterial thermodilution curve. The product of DSt and CO represent the largest individual mixing volume in a series of indicator dilution mixing chambers. When applied to femoral arterial thermodilution this volume is known as pulmonary thermal volume (PTV) and represents the pulmonary blood volume and extravascular lung water (refer to fig 3)



Once the PTV and ITTV are known the following preload volumes {global end diastolic volume (GEDV) and intrathoracic blood volume (ITBV)} and extravascular lung water (EVLW) can be calculated with the following formulas.

$$\begin{aligned}
 \text{GEDV} &= \text{ITTV} - \text{PTV} \\
 \text{ITBV} &= \text{GEDV} + \text{PBV} = 1.25 \times \text{GEDV} \\
 \text{EVLW} &= \text{ITTV} - \text{ITBV}
 \end{aligned}$$

### 2.3 Parameters obtained by arterial thermodilution

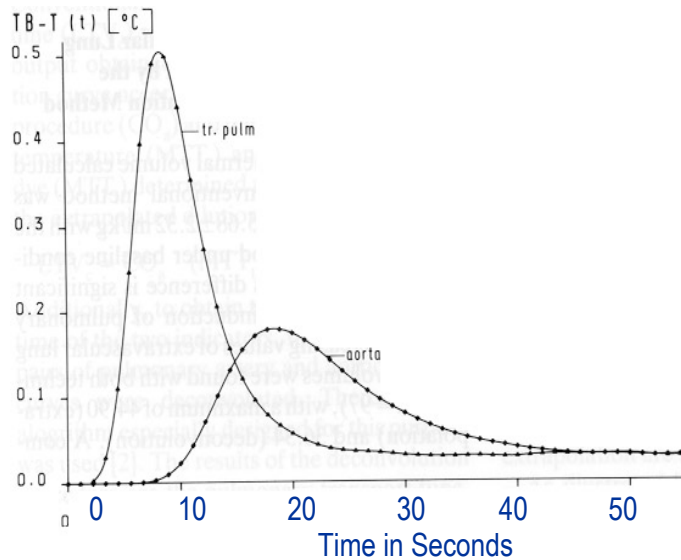
The following parameters are derived by the PiCCO from a central venous injection and arterial detection with a thermodilution catheter (1,2)

**The application of a pulmonary artery catheter is not necessary.**

Parameter	Absolute Parameter		Indexed Parameters	
	Abbr.	Unit	Abbr.	Unit
Cardiac output arterial	COa	l/min	Cl <sub>a</sub>	l/min/m <sup>2</sup>
Cardiac function index	CFI	l/min		
Global End Diastolic Volume	GEDV	ml	GEDVI	ml/kg
Intrathoracic blood volume	ITBV	ml	ITBVI	ml/m <sup>2</sup>
Extravascular Lung Water	EVLW	ml	EVLWI	ml/kg

#### 2.3.1 Cardiac Output Arterial (COa)

Arterial thermodilution cardiac output (COa) serves as the basic parameter for calculation of various blood volumes. The arterial thermodilution curves are four to five times longer than those of the pulmonary artery (refer to following diagram). Compared to pulmonary artery thermodilution, the arterial thermodilution measurement has minimal ventilatory variations. As a result, COa provides a representative mean value over the ventilatory cycle (6,8,9,11,14,15).



**Fig. 4.** Simultaneous recording of the pulmonary artery and aortic temperature dilution curves.

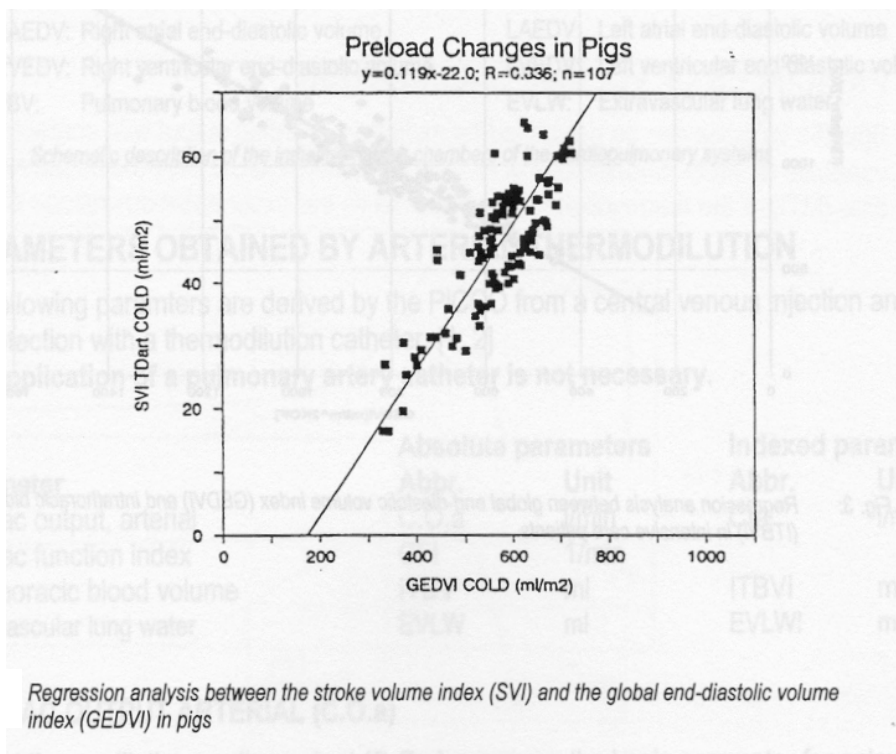
### 2.3.2 Global End Diastolic Volume (GEDV)

Global end diastolic volume is the sum of all end diastolic volumes of the atria and the ventricles. Thus, GEDV is equivalent to preload volume of the total heart. GEDV can be determined through thermodilution at the bedside

$$\text{GEDV} = \text{COa} \times \text{MttTDa} - \text{DstTDa}$$

#### (Patho) physiological significance of GEDV

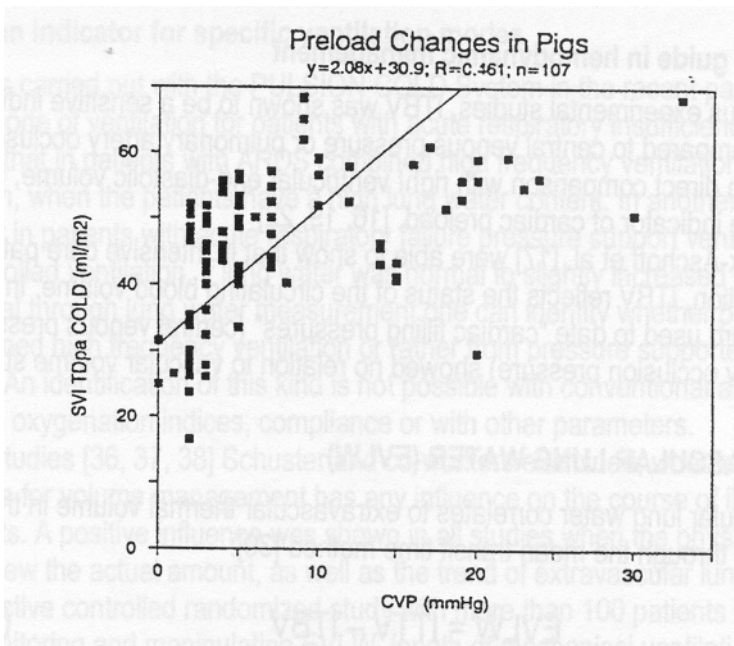
The following diagram demonstrates the Frank-Starling relationship between GEDVI and stroke volume index (SVI). The circulating blood volume of ten pigs was either suddenly decreased or increased. The relationship SVI/GEDVI for the volumes tested is linear in contrast to the well known curvilinear SVI/end-diastolic pressure relationship. The regression line for the SVI/GEDVI relationship does not intercept the Y-axis at zero. The y axis intercept at SVI = 0 is equivalent to the baseline preload volume of the heart that does not take part in the Frank-Starling mechanism (this dead space volume is often referred to as unstressed volume).



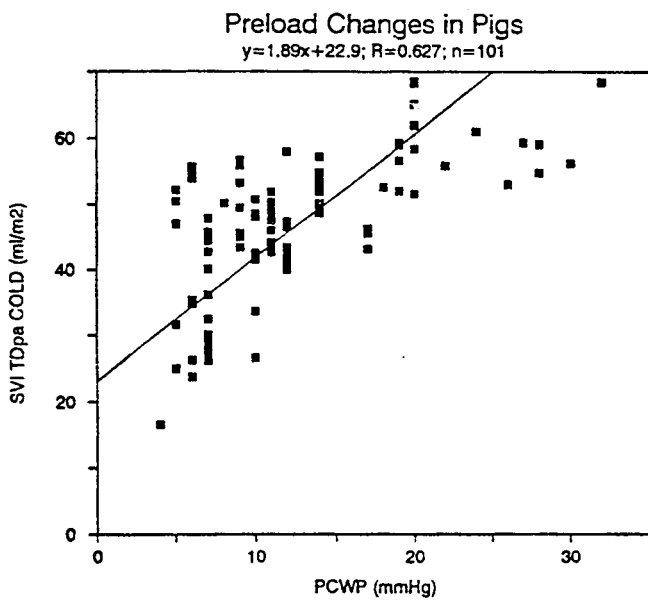
**Fig 5** Regression analysis between the stroke volume index (SVI) and the global end-diastolic volume index (GEDVI) in pigs

Traditionally central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) are used as indicators of cardiac preload. However, both CVP and PCWP are dependent on intravascular filling, intrathoracic pressure, vascular compliance and cardiac contractility. In contrast to pressures, GEDV represents cardiac preload volume. Following diagrams show the behaviour of central venous pressure (CVP fig 6) and pulmonary capillary wedge pressure (PCWP, fig 7) in the experiments mentioned above. The results

lead to the conclusion that CVP and PCWP are poor indicators for cardiac preload compared with GEDV.



**Fig 6**  
*Regression analysis between stroke volume index (SVI) and central venous pressure (CVP) in pigs*



**Figure 7**  
*Regression analysis between stroke volume index (SVI) and Pulmonary Capillary Wedge Pressure (PCWP) in pigs*

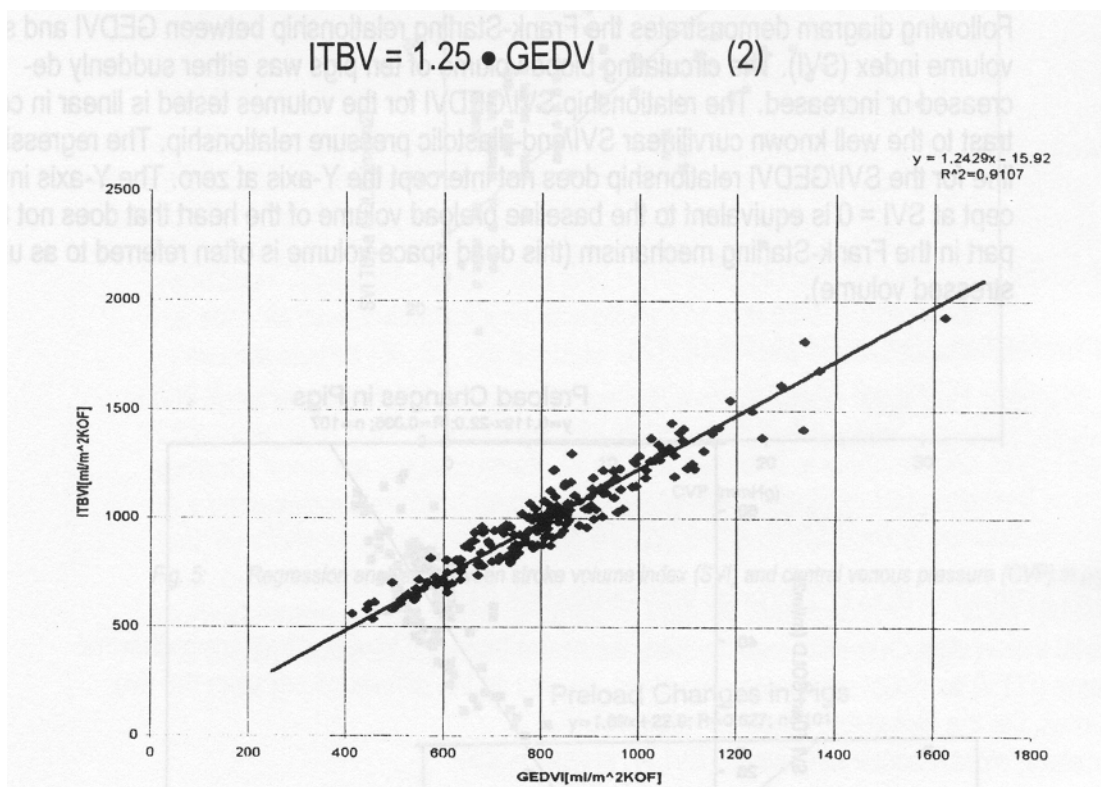
### 2.3.3 Intrathoracic Blood Volume

Cardiopulmonary or, more precisely, intrathoracic blood volume measurement has been performed with indicator dilution techniques for over 30 years.

#### ITBV estimation using single thermodilution method.

The PiCCO offers the possibility to assess ITBV derived from global end-diastolic volume (GEDV) determined by thermodilution measurement. GEDV correlates well with ITBV in both experimental and in clinical studies, as shown in figure 8.

Using regression analysis of GEDV (determined by thermodilution) and ITBV (determined by thermal-dye-dilution) a regression equation can be derived. Using this equation, ITBV (obtained without dye dilution) can be estimated (3,4,5)



**Fig 8** Regression analysis between global end diastolic volume index (GEDVI) and intrathoracic blood volume index (ITBVI) in intensive care patients

### **(Patho-) physiological significance of ITBV**

Intrathoracic blood volume (ITBV) consists of the global end-diastolic volume (GEDV, is approximately 4/5 of ITBV) and the pulmonary blood volume (PBV). Three volumes are found in the thorax, the intrathoracic blood volume, the intrathoracic gas volume and the extravascular lung water. Due to the limited expandability of the thorax, the volumes interact and change proportionally to each other. A potential fourth compartment is space occupying tumours or pleural effusions that change the overall volume of the chest cavity.

### **ITBV as a guide in hemodynamic management**

In numerous experimental studies, ITBV was shown to be a sensitive indicator of cardiac preload compared to central venous pressure or pulmonary capillary wedge pressure. In addition, in direct comparison with right ventricular end-diastolic volume, ITBV proves to be a sensitive indicator of cardiac preload. [16, 19, 22].

Lichtwarck-Aschoff et al. [17] were able to show that in intensive care patients on mechanical ventilation, ITBV reflects the status of the circulating blood volume. In contrast, the clinical standard used to date "cardiac filling pressures" (central venous pressure and pulmonary artery occlusion pressure) showed no relation to vascular, volume status. [20, 21]

### **2.3.4 Extravascular Lung Water (EVLW)**

Extravascular lung water correlates to extravascular thermal volume in the lungs and is evaluated through the mean transit time method [30]:

#### **2.3.4.1 EVLW estimation (EVLW\*) through estimated ITBV (ITBV)**

Arterial thermodilution results in the direct measurement of pulmonary thermal volume (PTV) and intrathoracic thermal volume (ITTV). From these values the extravascular lung water (EVLW) is assessable by the following:

$$\text{EVLW} = \text{ITTV} - \text{ITBV}$$

### **(Patho-) Physiological significance of EVLW**

The water content in the lungs increases through left heart failure, pneumonia, sepsis, intoxication, burns etc. Increased EVLW can occur through increased fluid transport to the interstitium, resulting from either high intravascular filtration pressure (left heart failure, volume overload) or an increased pulmonary vascular permeability for plasma proteins, which drag water along with them, corresponding to their colloid osmotic pressure. Changes in permeability of the lung are commonly seen in patients with endotoxic shock, pneumonia, sepsis, intoxication, and burns.

EVLW is the only determinable bedside parameter with which the lungs increased vascular permeability can be quantified.

Other methods of determining EVLW such as blood gases and the lung function indices are not organ specific, as they are not only dependent on the lung status, but also on the lung perfusion and ventilation. Correlation coefficients between EVLW and oxygenation indices are in the area of  $r = 0.5$  [25, 39, 40]

$$\text{EVLW}^* = \text{ITTV} - \text{ITBV}^* = \text{ITTV} - 1.25 \times \text{GEDV}$$

The x-ray of the lungs shows a density measurement of the total thorax, that's why it is dependent on the air and blood content as well as extravascular lung water. Furthermore, muscle and fat layers influence a quantitative density evaluation in an x-ray of the lungs [26, 27, 28, 29, 31, 32, 39]. The lung compliance is a parameter of the active surface film in the lungs and it does not correlate with water content [40].

### **EVLW as an indicator for specific ventilation modes**

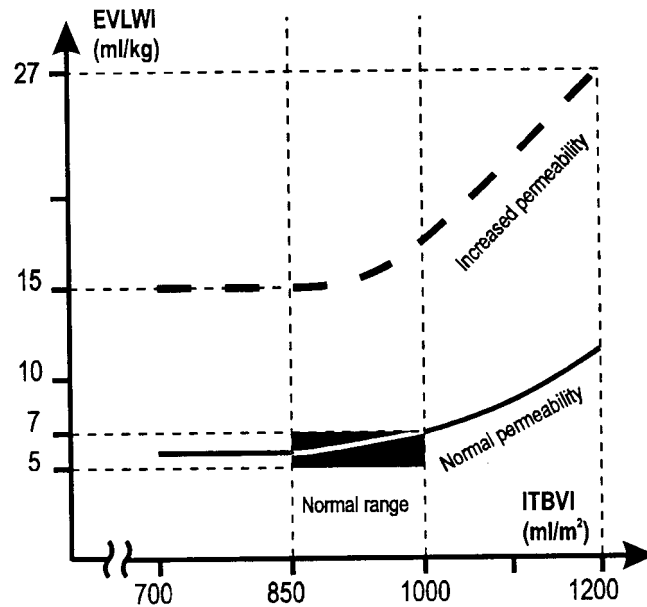
Two studies carried out with the PULSION COLD System in the recent past, address the choice of mode of ventilation for patients with acute respiratory insufficiency. Zeravik et al [42] found, that in patients with ARDS combined high frequency ventilation only improves oxygenation, when the patients have high lung water content. In another study it was shown, that in patients with acute respiratory failure pressure support ventilation was superior to controlled ventilation, if lung water was normal to slightly increased [43]. The results suggest, that through lung water measurement one can identify whether patients benefit from certain modes of mechanical ventilation.

In several studies [36, 37, 38] Schuster and co-workers examined, whether consideration of an EVLW value for volume management had any influence on the course of illness of intensive care patients. A positive influence was shown in all studies when the physicians treating the patients, knew the actual amount, as well as the trend of extravascular lung water. In a prospective controlled randomised study with more than 100 patients it was shown, that, by monitoring and manipulating EVLW, the length of mechanical ventilation and stay in the ICU could be reduced [37]. **Consideration of EVLW for circulating volume management reduces the lung oedema, the days of mechanical ventilation as well as days in the intensive care.**

### **Relationship of ITBV to EVLW**

In the past few years many studies have shown that intravascular volume management of critically ill patients by volume measurement has many advantages in comparison to that of pressure management [16, 17, 18, 19, 20, 21, 22]. The level of EVLW is related to patient outcome [40], and any measure taken to reduce EVLW is most likely to shorten ventilation days and stay in the ICU [37] and reduce possible complications such as nosocomial pneumonia and pneumothoracies.

The hydrostatic component of increased EVLW can be reduced by volume restriction. Figure 9 shows that below the "normal range" of ITBV one cannot reduce EVLW anymore. Therefore ITBV, representing cardiac preload, should not be driven below this "normal range" in order to avoid a further reduction of cardiac output and hence, oxygenation.



**Figure 9** Patient management by combined use of EVLW and ITBV

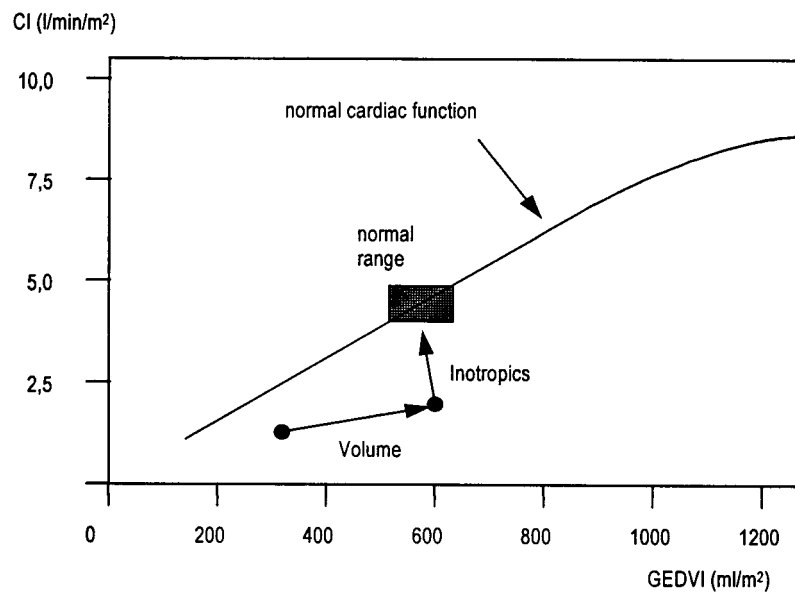
### 2.3.5 Cardiac Function Index (CFI)

Cardiac function index (CFI) is derived as the ratio of cardiac output divided by global end-diastolic volume:

$$CFI = CO / GEDVI$$

#### (Patho-) Physiological Significance Of CFI

The CFI is a preload independent variable reflecting the inotropic state of the heart [33, 34]. Figure 10 illustrates how positive inotropic stimulation makes the CFI curve steeper, and reduced contractility flattens the slope of the CFI curve.



**Figure 10** Schematic relationship between global end diastolic volume index (GEDVI) and cardiac index (CI)

### 3 Pulse Contour Analysis

#### 3.1 Principle of Measurement

During cardiac systole blood is ejected into the aorta. Simultaneously, blood flows out of the aorta into the peripheral vascular system. However, during the time of ejection, the sum of all blood flowing into the aorta is larger than the blood volume entering the peripheral vascular system. Thus, the volume of the aorta increases. In subsequent diastole, most of the remaining blood will empty into the peripheral vasculature. This behaviour is dependent on the ability of the aorta to expand and contract in response to the ejected volumes. The volume change and subsequent pressure change is described as the compliance function of the aorta.

The relationship between blood flow out of the aorta and pressure measured at the end of the aorta (femoral artery or other large artery) is determined by the compliance function. The compliance function can therefore be characterised by measuring blood pressure and blood flow (cardiac output) simultaneously. Transpulmonary thermodilution cardiac output (COa) determined simultaneously with continuous arterial pressure (AP) measurement is utilised to calibrate the pulse contour analysis to each individual patient's aortic compliance function.

For the continuous calculation of PCCO the PiCCO uses a calibration factor (cal) determined by the thermodilution cardiac output measurement and the heart rate (HR), as

well as the integrated values for the area under the systolic part of the pressure curve (P(t)/SVR), the aortic compliance (C(p) and the shape of the pressure curve, represented by change of pressure over change of time (dP/dT)

### 3.2 Calibration of Pulse Contour Analysis

To derive the calibration factor "cal" and the individual compliance function C(p) a reference transpulmonary thermodilution cardiac output is necessary. The PiCCO provides arterial thermodilution cardiac output for calibration. Transpulmonary cardiac output can be determined, by central venous injection of an indicator at least 10<sup>0</sup>C colder than the blood temperature, without the use of a pulmonary artery catheter. The thermodilution curve will be recorded using the arterial thermodilution catheter also used for arterial pressure monitoring.

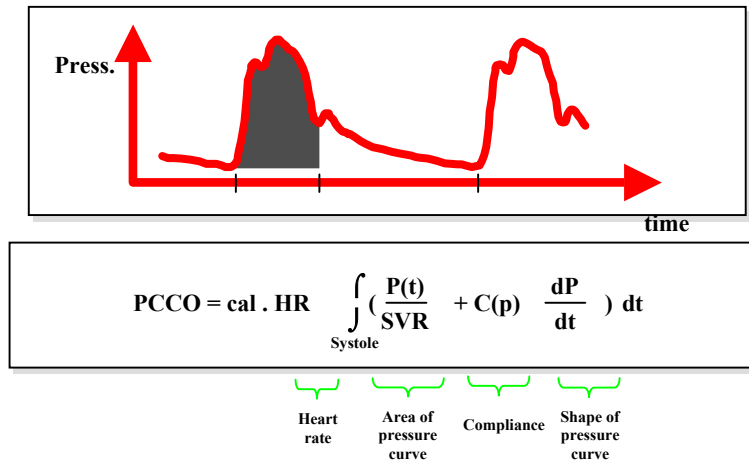
### 3.3 Parameters Obtained By Pulse Contour Analysis

Following parameters are derived by the PULSION PCCO, analysing the arterial pressure curve. SVV is displayed as the mean value over the last 30 seconds; all other derived parameters obtained by pulse contour analysis are displayed as mean values over the last 12 seconds.

		Absolute Parameters		Indexed Parameters	
Pulse Contour	Parameter	Abbr.	Unit	Abbr.	Unit
	Pulse contour cardiac output	PCCO	l/min	PCCI	l/min/m <sup>2</sup>
	Systolic arterial blood pressure	APsys	mmHg		
	Diastolic arterial blood pressure	APdia	mmHg		
	Mean arterial blood pressure	MAP	mmHg	ITBVI	ml/m <sup>2</sup>
	Heart rate	HR	1/min		
	Stroke volume	SV	ml	SVI	ml/m <sup>2</sup>
	Stroke volume variation	SVV	%		
	Systemic vascular resistance	SVR	dyn/sec/cm <sup>-5</sup>	SVRI	dyn/sec/cm <sup>-5</sup> /m <sup>2</sup>
Index of left ventricular contractility	dP/dtmax	mmHg/s			

### 3.3.1 Pulse Contour Cardiac Output (PCCO)

When the calibration factor "cal" and the individual compliance function C(p) is determined, continuous cardiac output can be measured using the heart rate and the area under the aortic flow curve as illustrated in figure 11. Displayed PCCO is the mean value of the last 12 seconds.



**Figure 11** Calculation of pulse contour cardiac output

In recent years, studies listed in the table below, have shown that continuous pulse contour cardiac output measurement is a reliable and a reproducible alternative to continuous cardiac output measurement utilizing a heating pulmonary artery catheter (PAC). [44, 45, 46, 47, 48, 49, 50, 51, 52]

Author	pat / obs	PCCO – COpa bias	r
Faltlhauser et al, 1996	9 pigs	-0.066 ± 1.10 l/min	0.89
Rauch et al, 1997	18/180	-0.139 ± 1.15 l/min	
Buhre et al, 1998	12/108	0.003 ± 0.63 l/min	0.94
Goedje et al, 1999	24/216	0.07 ± 0.7 l/min	0.92
Goedje et al, 1998	30/270	0.11 ± 0.6 l/min	0.91
Rödig et al, 1999	26/308	EF > 45 %: 0.18 l/min EF < 45 %: 0.12 l/min	

Reliability and reproducibility of PCCO

### 3.3.2 Arterial Blood Pressure (AP)

Arterial blood pressure is one of the most important diagnostic parameters for patient management. The PULSION PiCCO measures the arterial pressure continuously using a

special arterial thermodilution catheter with an additional pressure lumen. The arterial thermodilution and arterial pressure measurement can be done with the same catheter.

The arterial pressure is registered by a pressure transducer and displayed on the PULSION PiCCO display. Displayed AP is the mean value over the last 12 seconds. The pressure signal can be transferred to a conventional bedside monitor.

### 3.3.3 Stroke Volume Variation (SVV)

Stroke volume variation is presented as the change in stroke volume (in percent) calculated by the mean difference between highest and lowest stroke volume divided by a calculated mean stroke volume over the last 30 seconds. SVV is calculated according to:

$$\text{SVV} = (\text{SV}_{\text{rmax}} - \text{SV}_{\text{min}}) / \text{SV}_{\text{mean}}$$

SV max = mean value of maximum stroke volumes of the last 30sec

SV min = mean value of minimum stroke volumes of the last 30sec

SV mean = mean value of stroke volumes over the last 30sec

In mechanically ventilated patients, the SVV mainly depends on the intravascular volume of the patient. Big variations in stroke volume, induced by mechanically ventilation, is an indication of insufficient intravascular volume relative to the applied intrathoracic pressures. Thus, SVV allows a rough estimation of the vascular volume status. When high SVV is detected, it is recommended to perform a thermodilution measurement to quantify the volume status by measuring the ITBV [22].

### 3.3.4 Systemic Vascular Resistance (SVR)

The systemic vascular resistance is the quotient of driving pressure and cardiac output over the last 12 seconds. Here, the driving pressure represents the difference between mean arterial pressure (MAP) and central venous pressure (CVP).

$$\text{SVR} = \frac{(\text{MAP} - \text{CVP})}{\text{C.O.a}} \times 80$$

### 3.3.5 Index Of Left Ventricular Contractility (dP/dTMAX)

In basic physiology, contractility of the left ventricle is estimated by the maximum velocity of the left ventricular (LV) pressure curve. The majority of maximum pressure velocity increase fall into the ejection phase of the LV, which is represented by the up slope of the arterial pressure trace. Thus, the maximum velocity of the arterial pressure curve corresponds to the maximum power or contractility of the left heart.

Strictly speaking, the LV dP/dtmax can only be measured and quantitatively be assessed

as a parameter of contractility in the isoelectric phase of ventricular contraction. As a direct and continuous LV access in patients is contraindicated, it is recommended to measure the velocity of the pressure increase in a big vessel, to obtain a good indicator for the LV dP/dt<sub>max</sub>.

*(Patho-) Physiological significance of the contractility of the heart*

The cardiac output is essentially dependent on four parameters:

1. Preload
2. Left ventricular contractility
3. Afterload
4. Heart rate

Since afterload and heart rate influence output of the heart to a minor degree, two major possibilities to increase cardiac output remains. Primarily, there is a possibility to take advantage of the Frank-Starling relationship by increasing the preload within reasonable limits. But if the contractility is reduced, the application of volume (which increases the preload)} might be contraindicated. In this case, the contractility, which is a direct parameter for the force of the myocardium, can only be increased by inotropic stimulation.

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