A heritage of developing leading solutions that advance the care and treatment of the acutely ill

Since the introduction of the Swan-Ganz catheter in the early 1970s, Edwards Lifesciences has partnered with clinicians to develop products and systems that advance the care and treatment of the acutely ill. What has resulted is an extensive line of hemodynamic monitoring tools including catheters, sensors and bedside patient monitors that continue to build on this gold standard in critical care medicine.

Critical care clinicians around the world have used Edwards products to clinically manage more than 30 million patients. Hemodynamic monitoring products such as the Swan-Ganz catheter, FloTrac system and PreSep oximetry catheter enable clinicians to make more informed and rapid decisions when treating patients in surgical and critical care settings.

For additional educational resources visit: edwards.com/education
Acknowledgements
A special thank you to Christine Endres for her support to Anjali Berry, Diane K. Brown, RN, Pom Chaiyakal, Amanda Gage, Erin Glines-Foorman, RN, Sheryl Stewart and Susan Willig, for their guidance and expertise.
QUICK GUIDE TO
Cardiopulmonary Care

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In 1998, the first Quick Guide to Cardiopulmonary Care was published with the 2nd Edition of the Quick Guide being released in 2009. The intent of the Quick Guide was to provide a ready reference for hemodynamic monitoring and oxygenation assessment of the critically ill. To date, over 250,000 versions of the Quick Guide have been distributed globally through print and digital platforms. In addition, the Quick Guide has been translated into French, German, Italian, Spanish, Portuguese, Japanese and Chinese.

The 3rd Edition of the Quick Guide reflects current practice and changes in technology. Critical care is no longer a location bound by four walls.

Critically ill patients are being cared for in many different parts of the hospital now — especially as the patient population ages and acuity increases. During the last 10 years, less and noninvasive monitoring techniques have become part of routine assessment and care. Decision trees and algorithms using physiologic monitoring parameters have been published and are used in daily practice.

In this edition, the order of content reflects current concepts in assessment strategies and technology enhancements in which to monitor the patient. Additionally, pertinent sections of the Quick Guide to Central Venous Access have been incorporated to make this edition a more comprehensive reference guide.
The Quick Guide is organized into sections that build upon physiologic rationale. The first section begins with a review of oxygen delivery and consumption, including the determinants, implications of an imbalance, and the monitoring tools available.

More recent noninvasive technology is reviewed for the continuous monitoring of blood pressure and cardiac output. Basic monitoring techniques, including minimally-invasive monitoring technologies and functional hemodynamic parameters are presented in the next section. Advancements in technology have allowed for less invasive or minimally-invasive techniques, in both cardiac output and venous oxygen saturation assessment. Published decision trees employing the use of parameters obtained with less invasive technologies are provided.

The subsequent sections then present more advanced monitoring techniques including the Swan-Ganz catheter, which has been the hallmark of changing critical care practice since the early 1970s. Catheters range from a two-lumen catheter to an all-in-one catheter that provide the clinician with continuous pressure, continuous cardiac output, continuous end-diastolic volumes, and continuous venous oximetry. Many critically ill patients require this type of advanced, continuous monitoring and with the proper application of decision trees, patient care can be enhanced.

Because the practice of critical care and its related technologies are always changing and improving, the Quick Guide is not meant to address all aspects and needs in this arena. Rather, it has been written to provide a quick reference in which to enable the clinician to provide the best care possible to critically ill patients.
# Quick Guide to Cardiopulmonary Care

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Anatomy and Physiology

ADVANCING CRITICAL CARE THROUGH SCIENCE-BASED EDUCATION

SINCE 1972
Anatomy and Physiology

Ensuring that the tissues receive adequate oxygen and also that the tissues are able to consume the amount they require, is an important part of assessing the critically ill patient. Therefore, the goal of cardiorespiratory monitoring is to evaluate the components of oxygen delivery and consumption and to assess the utilization of oxygen at the tissue level. Parameters obtained from the physiologic profile are used to assess and optimize oxygen transport to meet the tissue needs of the critically ill patient. Basic cardiac anatomy, applied physiology, and pulmonary function are all components of oxygen delivery. Threats to the process of tissue oxygen balance can lead to inadequate utilization at the cellular level. Intervention strategies are directed at identifying the relationship of oxygen delivery to oxygen consumption to potentially eliminate the development of tissue hypoxia.
**Oxygen Delivery**

**(DO₂ = CO₂ x CO x 10)**

DO₂ is the amount of oxygen delivered or transported to the tissues in one minute and is comprised of oxygen content and the cardiac output. The adequacy of oxygen delivery is dependent upon appropriate pulmonary gas exchange, hemoglobin levels, sufficient oxygen saturation and cardiac output.

**Oxygen Content (CO₂):** amount of oxygen carried in the blood, both arterial and venous:

\[
(1.38 \times \text{Hgb} \times \text{SaO₂}) + (0.0031 \times \text{PaO₂})
\]

1.38: amount of O₂ that can combine with 1 gram of hemoglobin
0.0031: solubility coefficient of O₂ in the plasma*

\[
\begin{align*}
\text{CaO₂} &= (1.38 \times \text{Hgb} \times \text{SaO₂}) + (0.0031 \times \text{PaO₂}) \\
&\quad \text{Normal 20.1 mL/dL}
\end{align*}
\]

\[
\begin{align*}
\text{CvO₂} &= (1.38 \times \text{Hgb} \times \text{SvO₂}) + (0.0031 \times \text{PvO₂}) \\
&\quad \text{Normal 15.5 mL/dL}
\end{align*}
\]

**Oxygen Delivery (DO₂):** amount of oxygen transported in blood to tissues. Both arterial and venous O₂ delivery can be measured:

Arterial oxygen delivery (DO₂): \( \text{CO} \times \text{CaO₂} \times 10 \)

5 L/min x 20.1 mL/dL x 10 = 1005 mL/min†

Venous oxygen return (DvO₂): \( \text{CO} \times \text{CvO₂} \times 10 \)

5 L/min x 15.5 mL/dL x 10 = 775 mL/min

*Oxygen carrying capacity has been referenced between 1.34-1.39.
† Assumes Hgb of 15gm/dL
Oxygen Consumption

Oxygen consumption refers to the amount of oxygen used by the tissues, i.e., systemic gas exchange. This value cannot be measured directly but can be assessed by measuring the amount of oxygen delivered on the arterial side compared to the amount on the venous side.

### Oxygen Consumption (VO₂)

**Arterial Oxygen Transport – Venous Oxygen Transport**

\[
VO₂ = (CO \times CaO₂) - (CO \times CvO₂)
\]

\[
= CO \times (CaO₂ - CvO₂)
\]

\[
= CO \times [(SaO₂ \times Hgb \times 13.8) - (SvO₂ \times Hgb \times 13.8)]
\]

Normals: 200 – 250 mL/min  
120 – 160 mL/min/m²

**Note:** 13.8 = 1.38 x 10

### Conditions and Activities Altering Demand and VO₂

<table>
<thead>
<tr>
<th>Condition/Activity</th>
<th>Percentage</th>
</tr>
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<tr>
<td>Fever (one degree C)</td>
<td>10%</td>
</tr>
<tr>
<td>Work of breathing</td>
<td>40%</td>
</tr>
<tr>
<td>Shivering</td>
<td>50-100%</td>
</tr>
<tr>
<td>Work of breathing</td>
<td>40%</td>
</tr>
<tr>
<td>ET suctioning</td>
<td>7-70%</td>
</tr>
<tr>
<td>Post op procedure</td>
<td>7%</td>
</tr>
<tr>
<td>Sepsis</td>
<td>50-100%</td>
</tr>
<tr>
<td>MSOF</td>
<td>20-80%</td>
</tr>
<tr>
<td>Visitor</td>
<td>22%</td>
</tr>
<tr>
<td>Dressing change</td>
<td>10%</td>
</tr>
<tr>
<td>Position change</td>
<td>31%</td>
</tr>
<tr>
<td>Bath</td>
<td>23%</td>
</tr>
<tr>
<td>Sling scale weighing</td>
<td>36%</td>
</tr>
<tr>
<td>Chest X-Ray</td>
<td>25%</td>
</tr>
</tbody>
</table>

Note: 13.8 = 1.38 x 10
Other Assessment Parameters for Oxygen Utilization

Arterial-Venous Oxygen Difference

C(a-v)O₂: normally 5 vol %
20 vol % – 15 vol % = 5 vol %

*Note: Vol% or mL/dL*

Oxygen Extraction Ratio

O₂ER: normally 22 – 30%

O₂ER: \( \frac{CaO₂ - CvO₂}{CaO₂} \times 100 \)

\( CaO₂ = 20.1 \quad CvO₂ = 15.6 \)

O₂ER = \( \frac{20.1 - 15.6}{20.1} \times 100 = 22.4\% \)

Oxygen Extraction Index

Dual oximetry estimates oxygen extraction ratio. Evaluates the efficiency of oxygen extraction. Reflects cardiac reserve to increases in \( O₂ \) demand. Normal range is 20%–30%.

O₂EI = \( \frac{SaO₂ - SvO₂}{SaO₂} \times 100 \)

\( SaO₂ = 99, \quad SvO₂ = 75 \)

O₂EI = \( \frac{99 - 75}{99} \times 100 = 24.2\% \)

CO vs SvO₂ Correlations

\( SvO₂ \) reflects balance between oxygen delivery and utilization relationship to Fick equation.

\( VO₂ = C(a - v)O₂ \times CO \times 10 \)

\( CO = \frac{VO₂}{C(a - v)O₂} \)

\( C(a - v)O₂ = \frac{VO₂}{(CO \times 10)} \)

\( S(a - v)O₂ = \frac{VO₂}{(CO \times 10)} \)

\( SvO₂ \) reflects changes in oxygen extraction and the balance between \( DO₂ \) and \( VO₂ \).
VO₂/DO₂ Relationships

The relationship between oxygen delivery and consumption can theoretically be plotted on a curve. Since normally the amount of oxygen delivered is approximately four times the amount consumed, the amount of oxygen required is independent of the amount delivered. This is the supply independent portion of the curve. If oxygen delivery decreases, the cells can extract more oxygen in order to maintain normal oxygen consumption levels. Once the compensatory mechanisms have been exhausted, the amount of oxygen consumed is now dependent on the amount delivered. This portion of the graph is called supply dependent.

Oxygen debt occurs when the delivery of oxygen is insufficient to meet the body requirements. The implication of this concept is that additional oxygen delivery must be supported to “repay” this debt once it has occurred.

Factors Influencing Accumulation of O₂ Debt

Oxygen Demand > Oxygen Consumed = Oxygen Debt
Decreased oxygen delivery
Decreased cellular oxygen extraction
Increased oxygen demands
Functional Anatomy

For hemodynamic monitoring purposes, the right and left heart are differentiated as to function, structure and pressure generation. The pulmonary capillary bed lies between the right and left heart. The capillary bed is a compliant system with a high capacity to sequester blood.

The circulatory system consists of two circuits in a series: pulmonic circulation, which is a low-pressure system with low resistance to blood flow; and the systemic circulation, which is a high-pressure system with high resistance to blood flow.

### RIGHT AND LEFT HEART DIFFERENCES

<table>
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<th>Left Heart</th>
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<tr>
<td>Receives deoxygenated blood</td>
<td>Receives oxygenated blood</td>
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<tr>
<td>Low pressure system</td>
<td>High pressure system</td>
</tr>
<tr>
<td>Volume pump</td>
<td>Pressure pump</td>
</tr>
<tr>
<td>RV thin and crescent shape</td>
<td>LV thick and conical shape</td>
</tr>
<tr>
<td>Coronary perfusion biphasic</td>
<td>Coronary perfusion during diastole</td>
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### ANATOMICAL STRUCTURES

![Diagram of the circulatory system showing bronchus, pulmonary artery, alveolus, pulmonary vein, aortic valve, tricuspid valve, right atrium, right ventricle, pulmonic valve, left ventricle, and mitral valve with systemic and pulmonic circulations.]
Coronary Arteries and Veins

The two major branches of the coronary arteries arise from each side of the aortic root. Each coronary artery lies in the atrioventricular sulcus and is protected by a layer of adipose tissue.

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<th>Areas Supplied</th>
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<td>Right Coronary Artery (RCA)</td>
<td>Sinus Node 55%, AV Node 90%, Bundle of His (90%)</td>
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<tr>
<td></td>
<td>RA, RV free wall</td>
</tr>
<tr>
<td></td>
<td>Portion of IVS</td>
</tr>
<tr>
<td>Posterior Descending Branch</td>
<td>Portion of IVS</td>
</tr>
<tr>
<td>(Provided by RCA ≥ 80%)</td>
<td>Diaphragmatic aspect of LV</td>
</tr>
<tr>
<td>Left Main Coronary Artery Bifurcates</td>
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</tr>
<tr>
<td>Left Anterior Descending (LAD)</td>
<td>Left anterior wall</td>
</tr>
<tr>
<td></td>
<td>Anterior portion of IVS</td>
</tr>
<tr>
<td></td>
<td>Portion of right ventricle</td>
</tr>
<tr>
<td>Left Circumflex</td>
<td>Sinus node 45%, LA, 10% AV node</td>
</tr>
<tr>
<td>(Provides posterior descending branch ≤ 20%)</td>
<td>Lateral and posterior wall of LV</td>
</tr>
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<th>Coronary Veins</th>
<th>Location Drains Into</th>
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<td>Thebesian Veins</td>
<td>Directly into R and L ventricles</td>
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<td>Great Cardiac Vein</td>
<td>Coronary sinus in the RA</td>
</tr>
<tr>
<td>Anterior Cardiac Veins</td>
<td>RA</td>
</tr>
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</table>
**CORONARY ARTERIES**

Blood is supplied to heart tissues by branches of the coronary arteries.

- Aorta
- Pulmonary Trunk
- Superior Vena Cava
- Left Atrium
- Left Coronary Artery
- Right Atrium
- Right Coronary Artery
- Circumflex Artery
- Left Anterior Descending
- Marginal Artery
- Left Ventricle
- Posterior Descending Artery
- Right Ventricle

**CORONARY VEINS**

Blood is drained by branches of the cardiac veins.

- Superior Vena Cava
- Pulmonary Trunk
- Aorta
- Right Atrium
- Left Atrium
- Great Cardiac Vein
- Inferior Vena Cava
- Left Ventricle
- Right Ventricle
Cardiac Cycle: Electrical Correlation to Mechanical

Electrical cardiac cycle occurs prior to mechanical cardiac cycle. Atrial depolarization begins from the SA node. This current is then transmitted throughout the ventricles. Following the wave of depolarization, muscle fibers contract which produces systole.

The next electrical activity is repolarization which results in the relaxation of the muscle fibers and produces diastole. The time difference between the electrical and mechanical activity is called electro-mechanical coupling, or the excitation-contraction phase. A simultaneous recording of the ECG and pressure tracing will show the electrical wave before the mechanical wave.

**ELECTRICAL – MECHANICAL CARDIAC CYCLE**

![Diagram of Electrical – Mechanical Cardiac Cycle]

- ECG
- RA
- RV
- Atrial Depolarization
- Vent Depolarization
- Ventricular Repolarization
- Atrial Systole
- Atrial Filling
- Atrial Kick
- Ventricular Systole
- Ventricular Diastole
Mechanical Cardiac Cycle Phases

**SYSTOLE**

1. **Isovolumetric Phase**
   - Follows QRS of ECG
   - All valves closed
   - Majority of oxygen consumed

2. **Rapid Ventricular Ejection**
   - Aortic valve opens
   - Occurs during ST segment
   - 2/3 or more of blood volume ejected

3. **Reduced Ventricular Ejection**
   - Occurs during “T” wave
   - Atria are in diastole
   - Produces “v” wave in atrial tracing

**DIASTOLE**

1. **Isovolumetric Relaxation**
   - Follows “T” wave
   - All valves closed
   - Ventricular pressure declines further
   - LV pressure dips below LA pressure

2. **Rapid Ventricular Filling**
   - AV valves open
   - Approximately 70% of blood volume goes into ventricle

3. **Slow Filling Phase: End-Diastole**
   - Atrial “kick”
   - Follows “P” wave during sinus rhythms
   - Atrial systole occurs
   - Produces “a” wave on atrial tracings
   - Remaining volume goes into ventricle
Coronary Artery Perfusion

Coronary artery perfusion for the left ventricle occurs primarily during diastole. The increase in ventricular wall stress during systole increases resistance to such an extent that there is very little blood flow into the endocardium. During diastole there is less wall tension so a pressure gradient occurs that promotes blood flow through the left coronary arteries. The right ventricle has less muscle mass, therefore less wall stress during systole, so that due to less resistance, more blood flows through the right coronary artery during systole. Optimal RV performance depends in part on this biphasic perfusion. There must be adequate diastolic pressure in the aortic root for both coronary arteries to be perfused.
Cardiac Output Definition

Cardiac output (liters/minute, L/min): amount of blood ejected from the ventricle in a minute.

Cardiac Output = Heart Rate x Stroke Volume
Heart Rate = beats/min
Stroke Volume = mL/beat; amount of blood ejected from ventricle in one beat

CO = HR x SV

Normal Cardiac Output: 4 – 8 L/min
Normal Cardiac Index : 2.5 – 4 L/min/m²
CI = CO/BSA
BSA = Body Surface Area
Normal Heart Rate Range: 60 – 100 BPM
Normal Stroke Volume: 60 – 100 mL/beat

Stroke volume: difference between end-diastolic volume (EDV), [the amount of blood in the ventricle at the end of diastole]; and end-systolic volume (ESV), [blood volume in the ventricle at the end of systole]. Normal SV is 60 to 100 mL/beat.

SV = EDV – ESV

SV also calculated by: SV = CO / HR x 1000

Note: 1000 used to convert L/min to mL/beat

When stroke volume is expressed as a percentage of end-diastolic volume, stroke volume is referred to as the ejection fraction (EF). Normal ejection fraction for the LV is 60 – 75%. The normal EF for the RV is 40 – 60%.

EF = (SV / EDV) x 100

DETERMINANTS OF CARDIAC OUTPUT

- Cardiac Output
- Heart Rate
- Stroke Volume
- Preload
- Afterload
- Contractility
Preload Definition and Measurements

Preload refers to the amount of myocardial fiber stretch at the end of diastole. Preload also refers to the amount of volume in the ventricle at the end of this phase. It has been clinically acceptable to measure the pressure required to fill the ventricles as an indirect assessment of ventricular preload. Left atrial filling pressure (LAFP) or pulmonary artery occlusion pressure (PAOP) and left atrial pressures (LAP) have been used to evaluate left ventricular preload. Right atrial pressure (RAP) has been used to assess right ventricular preload. Volumetric parameters (RVEDV) are the preferred preload measure as they eliminate the influence of ventricular compliance on pressure.

**Preload**

- RAP/CVP: 2 – 6 mmHg
- PAD: 8 – 15 mmHg
- PAOP/LAP: 6 – 12 mmHg
- RVEDV: 100 – 160 mL

**Frank–Starling Law**

Frank and Starling (1895, 1918) identified the relationship between myocardial fiber length and force of contraction. The more the diastolic volume or fiber stretch at the end of the diastole, the stronger the next contraction during systole to a physiologic limit.
Ventricular Compliance Curves

The relationship between end-diastolic volume and end-diastolic pressure is dependent upon the compliance of the muscle wall. The relationship between the two is curvilinear. With normal compliance, relatively large increases in volume create relatively small increases in pressure. This will occur in a ventricle that is not fully dilated. When the ventricle becomes more fully dilated, smaller increases in volume produce greater rises in pressure. In a non-compliant ventricle, a greater pressure is generated with very little increase in volume. Increased compliance of the ventricle allows for large changes in volume with little rise in pressure.

**Normal Compliance**

Pressure/volume relationship is curvilinear:
- a: Large increase in volume = small increase in pressure
- b: Small increase in volume = large increase in pressure

**Decreased Compliance**

*Stiffer, less elastic ventricle*
- Ischemia
- Increased afterload
- Hypertension
- Inotropes
- Restrictive cardiomyopathies
- Increased intrathoracic pressure
- Increased pericardial pressure
- Increased abdominal pressure

**Increased Compliance**

*Less stiff, more elastic ventricle*
- Dilated cardiomyopathies
- Decreased afterload
- Vasodilators
Afterload Definition and Measurements

Afterload refers to the tension developed by the myocardial muscle fibers during ventricular systolic ejection. More commonly, afterload is described as the resistance, impedance, or pressure that the ventricle must overcome to eject its blood volume. Afterload is determined by a number of factors, including: volume and mass of blood ejected, the size and wall thickness of the ventricle, and the impedance of the vasculature. In the clinical setting, the most sensitive measure of afterload is systemic vascular resistance (SVR) for the left ventricle and pulmonary vascular resistance (PVR) for the right ventricle. The formula for calculating afterload include the gradient difference between the beginning or inflow of the circuit and the end or outflow of the circuit.

Afterload

Pulmonary Vascular Resistance (PVR): <250 dynes - sec - cm⁻⁵
\[ PVR = \frac{MPAP-PAOP \times 80}{CO} \]

Systemic Vascular Resistance (SVR): 800-1200 dynes - sec - cm⁻⁵
\[ SVR = \frac{MAP-RAP \times 80}{CO} \]

Afterload has an inverse relationship to ventricular function. As resistance to ejection increases, the force of contraction decreases, resulting in a decreased stroke volume. As resistance to ejection increases, an increase in myocardial oxygen consumption also occurs.
Contractility Definition and Measurements

Inotropism or contractility refers to the inherent property of the myocardial muscle fibers to shorten independent of preload and/or afterload.

Contractility changes can be plotted on a curve. It is important to note that changes in contractility result in shifts of the curves, but not the underlying basic shape.

Measurements of contractility cannot be directly obtained. Clinical assessment parameters are surrogates and all include determinants of preload and afterload.

**Contractility**

Stroke Volume

\[ SV = (CO \times 1000)/HR \]

\[ SVI = SV/BSA \]

Left Ventricular Stroke Work Index

\[ LVSWI = SVI (MAP – PAOP) \times 0.0136 \]

Right Ventricular Stroke Work Index

\[ RVSWI = SVI (PA \text{ mean} – CVP) \times 0.0136 \]

**VENTRICULAR FUNCTION CURVES**

A: Normal Contractility
B: Increased Contractility
C: Decreased Contractility
Family of Ventricular Function Curves

Ventricular function can be represented by a family of curves. The performance characteristics of the heart can move from one curve to another, depending upon the state of preload, afterload, contractility or ventricular compliance.

**VENTRICULAR FUNCTION CURVES**

![Graph showing different states of preload and afterload with labels A, B, and C for normal, decreased, and increased states respectively.]
Pulmonary Function Tests

Definitions:

**Total Lung Capacity (TLC):** maximal amount of air within the lung at maximal inspiration. (~5.8L)

**Vital Capacity (VC):** maximal amount of air that can be exhaled after a maximal inspiration. (~4.6L)

**Inspiratory Capacity (IC):** maximal amount of air that can be inhaled from resting level after normal expiration. (~3.5L)

**Inspiratory Reserve Volume (IRV):** maximal amount of air that can be inhaled after a normal inspiration during quiet breathing. (~3.0L)

**Expiratory Reserve Volume (ERV):** maximal amount of air that can be exhaled from the resting level following a normal expiration. (~1.1L)

**Functional Residual Capacity (FRC):** amount of air remaining in the lungs at the end of normal expiration. (~2.3L)

**Residual Volume (RV):** volume of gas remaining in lungs after maximal expiration. (~1.2L)

All pulmonary volumes and capacities are about 20–25% less in women than men.
Acid Base Balance

Arterial Blood Gas Analysis

Simple acid base abnormalities can be divided into metabolic and respiratory disorders. Values obtained from blood gas analysis can assist in determining the disorder present.

Definitions

**Acid:** A substance which can donate hydrogen ions  
**Base:** A substance which can accept hydrogen ions  
**pH:** The negative logarithm of H⁺ ion concentration  
**Acidemia:** An acid condition of the blood with pH < 7.35  
**Alkalemia:** An alkaline (base) condition of the blood with pH > 7.45

**PCO₂:** Respiratory Component  
**PaCO₂:** Normal ventilation 35 – 45 mmHg  
Hypoventilation > 45 mmHg  
Hyperventilation < 35 mmHg  

**HCO₃:** Metabolic Component  
Balanced 22 – 26 mEq/L  
Base Balance -2 to +2  
Metabolic Alkalosis > 26 mEq/L  
Base excess > 2 mEq/L  
Metabolic Acidosis < 22 mEq/L  
Base deficit < 2 mEq/L

Normal Blood Gas Values

<table>
<thead>
<tr>
<th>Component</th>
<th>Arterial</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.40 (7.35 – 7.45)</td>
<td>7.36 (7.31 – 7.41)</td>
</tr>
<tr>
<td>PO₂ (mmHg)</td>
<td>80 – 100</td>
<td>35 – 45</td>
</tr>
<tr>
<td>SO₂ (%)</td>
<td>≥ 95</td>
<td>60 – 80</td>
</tr>
<tr>
<td>PCO₂ (mmHg)</td>
<td>35 – 45</td>
<td>42 – 55</td>
</tr>
<tr>
<td>HCO₃ (mEq/L)</td>
<td>22 – 26</td>
<td>24 – 28</td>
</tr>
<tr>
<td>Base excess/deficit</td>
<td>-2 – +2</td>
<td>-2 – +2</td>
</tr>
</tbody>
</table>
Oxyhemoglobin Dissociation Curve

The oxyhemoglobin dissociation curve (ODC) graphically illustrates the relationship that exists between the partial pressure (PO2) of oxygen and oxygen saturation (SO2). The sigmoid-shaped curve can be divided into two segments. The association segment or upper portion of the curve represents oxygen uptake in the lungs or the arterial side. The dissociation segment is the lower portion of the curve and represents the venous side, where oxygen is released from the hemoglobin.

**NORMAL OXYHEMOGLOBIN DISSOCIATION CURVE**

![Graph showing Oxyhemoglobin Dissociation Curve]

The affinity of hemoglobin for oxygen is independent of the PO2 – SO2 relationship. Under normal conditions, the point at which the hemoglobin is 50% saturated with oxygen is called the P50 at a PO2 of 27 mmHg. Alterations in the hemoglobin-oxygen affinity will produce shifts in the ODC.

**FACTORS SHIFTING OXYHEMOGLOBIN DISSOCIATION CURVE**

- **Leftward shift:**
  - Increased affinity
  - Higher SO2 for PO2
  - ↑ pH, Alkalosis
  - Hypothermia
  - ↓ 2-3 DPG

- **Rightward shift:**
  - Decreased affinity
  - Lower SO2 for PO2
  - ↓ pH, Acidosis
  - Hyperthermia
  - ↑ 2-3 DPG

The clinical significance of shifting the ODC is that SO2 and PO2 assessment parameters may not accurately reflect the patients’ clinical status. A shift of the ODC to the left can lead to tissue hypoxia in spite of normal or high saturation values.
**Pulmonary Gas Exchange Equations**

Assessing pulmonary function is an important step in determining the cardiorespiratory status of the critically ill patient. Certain equations can be employed to evaluate pulmonary gas exchange, to evaluate the diffusion of oxygen across the pulmonary capillary unit, and to determine the amount of intrapulmonary shunting. An alteration in any of these will impact oxygen delivery.

Alveolar Gas Equation: PAO₂ is known as the ideal alveolar PO₂ and is calculated knowing the composition of inspired air.

\[ \text{PAO}_2 = [(\text{PB} - \text{PH}_2\text{O}) \times \text{FiO}_2] - \text{PaCO}_2 \div 0.8 \]

**Alveolar–arterial Oxygen Gradient (A–a Gradient or P(A–a)O₂)**

P(A-a)O₂: Assesses the amount of oxygen diffusion across the alveolar capillary unit. Compares the alveolar gas equation to the arterial partial pressure of oxygen.

\[ [(\text{PB} - \text{PH}_2\text{O}) \times \text{FiO}_2] - \text{PaCO}_2 \times [\text{FiO}_2 + (1 - \text{FiO}_2) / 0.8] - (\text{PaO}_2) \]

- Normal: < 15 mmHg on room air
- Normal: 60 – 70 mmHg on FiO₂ 1.0

**PB:** Atmospheric pressure at sea level: 760

**PH₂O:** Pressure of water: 47 mmHg

**FiO₂:** Fraction of inspired oxygen

**PaCO₂:** Partial pressure of CO₂

**0.8:** Respiratory quotient (VCO₂ / VO₂)

### A–a Gradient Calculation

\[
(\text{Barometric Pressure} - \text{Water Vapor Pressure}) \times \text{Patient’s FiO}_2 - \frac{\text{PaCO}_2}{0.8} \quad \text{Patient’s PaO}_2
\]

\[
(760 - 47) \times 0.21 - \frac{40}{0.8} \quad 90
\]

\[
713 \times 0.21 - 50 \quad 90
\]

\[
99.73 - 90 = 9.73
\]

A–a Gradient \( \approx 10 \)

Assumes breathing at sea level, on room air, with a PaCO₂ of 40 mmHg and PaO₂ of 90 mmHg.
**Intrapulmonary Shunt**

Intrapulmonary shunt (Qs/Qt) is defined as the amount of venous blood that bypasses an alveolar capillary unit and does not participate in oxygen exchange. Normally a small percentage of the blood flow drains directly into either the thebesian or pleural veins which exit directly into the left side of the heart. This is considered an anatomical or true shunt, and is approximately 1 – 2% in normal subjects and up to 5% in ill patients.

The physiologic shunt or capillary shunt occurs when there are either collapsed alveolar units or other conditions where the venous blood is not oxygenated.

Some controversies exist in regards to measuring Qs/Qt. A true shunt is said to be accurately measured only when the patient is on an FiO₂ of 1.0. Venous admixture which produces a physiologic shunt can be determined when the patient is on an FiO₂ of < 1.0. Both determinations require pulmonary artery saturation values to complete the calculation.

\[
\text{Qs/Qt} = \frac{\text{CcO}_2 - \text{CaO}_2}{\text{CcO}_2 - \text{CvO}_2}
\]

\[
\text{CcO}_2 = \text{Capillary oxygen content} = (1.38 \times \text{Hgb} \times 1) + (\text{PAO}_2 \times 0.0031)
\]

\[
\text{CaO}_2 = \text{Arterial oxygen content} = (1.38 \times \text{Hgb} \times \text{SaO}_2) + (\text{PaO}_2 \times 0.0031)
\]

\[
\text{CvO}_2 = \text{Venous oxygen content} = (1.38 \times \text{Hgb} \times \text{SvO}_2) + (\text{PvO}_2 \times 0.0031)
\]
Ventilation Perfusion Index (VQI) has been described as a dual oximetry estimate of intrapulmonary shunt (Qs/Qt).

Assumptions involved in the equation are:
1. Dissolved oxygen is discounted
2. 100% saturation of pulmonary end-capillary blood
3. Hgb changes are not abrupt

Limitations of VQI include:
1. VQI can only be calculated if SaO₂ < 100%
2. Poor agreement with Qs/Qt if PaO₂ > 99 mmHg
3. Good correlation when Qs/Qt > 15%

**Equation Derivations**

\[
\frac{Qs}{Qt} = 100 \times \frac{[1.38 \times \text{Hgb} + (0.0031 \times \text{PAO}_2) - \text{CaO}_2]}{[1.38 \times \text{Hgb} + (0.0031 \times \text{PAO}_2) - \text{CvO}_2]}
\]

\[
\text{VQI} = 100 \times \frac{[1.38 \times \text{Hgb} \times (1 - \frac{\text{SaO}_2}{100}) + (0.0031 \times \text{PAO}_2)]}{[1.38 \times \text{Hgb} \times (1 - \frac{\text{SvO}_2}{100}) + (0.0031 \times \text{PAO}_2)]}
\]

**Dual Oximetry, Simplifies the Shunt Equation**

\[
\text{VQI} = \frac{\text{SAO}_2 - \text{SaO}_2 = 1 - \text{SaO}_2}{\text{SAO}_2 - \text{SvO}_2 = 1 - \text{SvO}_2} \quad \text{or} \quad \frac{1 - \text{SpO}_2}{1 - \text{SvO}_2}
\]
Advanced Noninvasive Monitoring

Advancing Critical Care Through Science-Based Education

Since 1972
The ClearSight System

Continuous blood pressure monitoring as well as continuous arterial pressure based cardiac output (APCO) historically has depended upon an indwelling arterial catheter connected to a disposable pressure transducer or cardiac output sensor. The creation of pulse contour-based noninvasive systems provided the opportunity to measure blood pressure, cardiac output and other hemodynamic parameters without the need for an arterial line.

In 2012, Edwards Lifesciences acquired BMEYE B.V. who created the ccNexfin system – a noninvasive system that uses a finger cuff with an infrared light system and an inflatable bladder to accurately measure continuous beat-to-beat blood pressure and cardiac output. This technology has been validated against multiple technologies including an upper arm cuff, invasive radial line, transpulmonary thermodilution and esophageal echo-Doppler. The ccNexfin system was rebranded by Edwards Lifesciences as the ClearSight system in 2014.
The Edwards ClearSight technology with the EV1000 clinical platform noninvasively provides:

- Blood Pressure (BP)
- Cardiac Output (CO)
- Stroke Volume (SV)
- Pulse Rate (PR)
- Stroke Volume Variation (SVV)
- Systemic Vascular Resistance (SVR)

System Components

- ClearSight noninvasive finger cuff
- Pressure controller
- Heart reference sensor
How it Works

Finger Cuff

Each ClearSight finger cuff consists of:

- An inflatable blood pressure bladder
- An infrared light
- A receiving light sensor

The infrared light and receiving light sensor work together to continually measure the changing arterial volume, which pulsates at the same rhythm as the heart. The pressure controller continually adjusts the pressure in the finger cuff’s inflatable bladder with the result that the arteries and bladder are equal in pressure and the arteries no longer pulsate. The volume of the arteries at this point is referred to as the unloaded volume.

Physiocal

Using a process called Physiocal, the ClearSight system determines and periodically updates the target unloaded volume, known as the setpoint, in order to calibrate the blood pressure measurement.
When measurements are initiated, the ClearSight system runs Physiocal, which can be identified by its characteristic staircase-shaped waveform. This waveform indicates that the ClearSight system is stepping up and down in pressure in order to calculate the proper unloaded arterial volume.

![Waveform Image]

Typically, the first blood pressure waveform and its associated data will be displayed on the monitor in approximately 20 seconds.

Physiocal periodically recalibrates the system which is essential for tracking a changing setpoint. Changes may result from smooth muscle tone changes during events such as vasoconstriction, vasodilation, and temperature change. This calibration initially begins at 10 beat intervals, and increases to 70 beat intervals depending on the stability of the finger physiology.

![Waveform Image]
Volume Clamp Method

The volume clamp method is the process that:

- Controls the pressure in the ClearSight finger cuff to maintain the unloaded volume: the pressure that is required to continuously maintain the unloaded volume is equal to the blood pressure in the finger
- Directly measures the finger cuff pressure in order to display it as a waveform on the EV1000 monitor

The volume clamp control loop, located within the pressure controller, consists of the following steps (see figure below):
1. The arterial volume, which was measured by the infrared light and receiving light sensor, is compared to the Physiocal setpoint.

2. The pressure needed to counteract any arterial diameter change is determined by a controller.

3. a) The controller then sends a signal to the control valve which dynamically manages the amount of pressure applied to the finger cuff

b) At the same time, the transducer directly senses the cuff pressure and translates it into a point on the blood pressure waveform.

This volume clamp control loop, including adjustment of the cuff pressure, is performed 1000 times every second resulting in a real-time finger pressure waveform.
Brachial Reconstruction

Since the arteries narrow as the distance from the heart increases, increased resistance and backwards reflection of the pressure waves occurs. This results in varying pressure levels and waveform shapes.

The brachial site has long been the clinical standard for noninvasive blood pressure measurements. The finger site, however, has slightly lower mean pressure levels and usually an increasingly peaked waveform. Therefore, the finger pressure waveform must be transformed to be comparable to a brachial site waveform. The ClearSight system does so using a mathematical transfer function based on a vast clinical database.
The Heart Reference Sensor, or HRS, is put in place to compensate for hydrostatic pressure changes due to differences in height between the finger component and the heart component.

Without the HRS, changes in the patient’s finger position, relative to the heart, will affect the blood pressure measurements. With the HRS in use, and the heart component remaining at the heart level, any movements of the patient’s hand are automatically compensated for and will not affect the blood pressure measurements.
Pulse Contour Method – Calculating CO and SV

The ClearSight system pulse contour method, which is based on a physiological model of circulation, is used to noninvasively and continuously calculate beat-to-beat SV and CO.

When pumping blood through the body, the left side of the heart experiences an impedance referred to as afterload, shown here as Zin. This impedance is experienced due to the relationship between blood pressure and blood flow, which in this case is equivalent to SV.

\[ \frac{\Delta P}{\text{SV}} = Z_{in} \]

By re-arranging this relationship and individualizing it for each patient, we can use BP and afterload to calculate SV.

\[ \text{SV} = \frac{\Delta P}{Z_{in}} \]

We can calculate BP, the first component of SV, by determining the area under the systolic portion of the brachial arterial waveform.
We can estimate afterload, the second component of SV, using a physiological model of the afterload experienced by the heart. This model is individualized for each patient using the patient’s age, gender, height and weight.

\[ Z_{in} \]

\( Z_0 \) – characteristic impedance; \( C_w \) – arterial compliance; \( R_p \) - peripheral resistance (a corollary of SVR)

Once we have calculated BP and estimated afterload, we obtain a final estimate of SV for each heart beat.

CO is then calculated by multiplying pulse rate by SV.

\[ CO = SV \times PR \]

All other hemodynamic parameters are then calculated from the arterial waveform in combination with SV and CO, including pulse rate, SVV and SVR.
Sensor Setup

Applying the finger cuff

1. Place the index, middle, or ring finger in cuff #1 between both optical components and center between the two knuckles.

2. Gently lead the cuff cable between two fingers toward the back of the hand.

3. Wrap the finger cuff snugly around the contour of the finger ensuring that the end of the finger cuff lies inside the green zone. The image of the finger should lie on the top side of the finger.

4. Connect the finger cuff to the pressure controller.

5. If using 2 cuffs repeat steps 1-4 on an adjacent finger with cuff #2.
Applying the HRS

1. Secure the pressure controller to the Velcro strap with the cuff connections facing the fingers. Wrap the Velcro strap around the wrist and secure it.

2. Connect the HRS to its respective port on the pressure controller.

3. Secure the finger side of the HRS to one of the finger cuffs.

4. Secure the heart side of the HRS to the patient at heart level.

**NOTE:** Use surgical tape to properly secure both ends of the HRS if necessary.
Connecting the pump unit

1. Begin by connecting the pump unit to an electrical outlet.

2. Connect the pressure controller cord to the right side of the pump unit.

3. Connect the system power cable and the communication cable to the EV1000 monitor panel (1) and pump unit (2).

Connecting to a patient monitor (optional)

1. Connect patient monitor adapter cable to the pump unit and pressure cable (1).

2. Connect pressure cable to the patient monitor (2).
The EV1000 Monitor Setup

Entering patient data
1. Tap the **Settings** icon on the navigation bar.
2. Tap the **Patient Data** button.
3. Tap the **New Patient Data** button or update existing patient information by tapping the grey rectangles next to each field.

**NOTE:** Accurate measurements of CO can only be obtained if the following patient data are properly entered: gender, age, weight, and height.
4. Tap the **Home** button to confirm.

Zeroing the HRS
1. Place both ends of the HRS at the same vertical level.
2. Tap the **Clinical Actions** icon on the navigation bar.
3. Tap the **Zero & Waveform** button.
4. Tap the **Zero** button under **Align & Zero HRS**.

**NOTE:** The HRS can be zeroed before being placed on a patient.
Zeroing the patient monitor (if applicable)
1. Move pressure output selection to zero position.
2. Press the Zero button on connected patient monitor.
3. Move the pressure output selection to signal position.

Starting/Stopping a measurement
1. Zero the HRS sensor (see Zeroing the HRS).
2. Tap the Pump Unit Start/Stop icon on the navigation bar. An arterial waveform will appear at the top of the screen.
3. Press the Home button to confirm.
4. You may also view the arterial waveform on the trend screen by tapping the Screen Selection icon on the navigation bar, selecting the Trend Screen icon, and tapping the Arterial Pressure Waveform icon.
5. Use the same icon to remove the waveform.
6. To stop a measurement, tap the Pump Unit Start/Stop icon on the navigation bar.
Clinical Applications and Patients

The ClearSight system provides hemodynamic monitoring to those patients who could benefit from continuous monitoring and/or goal-directed therapy but would not receive an arterial line. These are usually patients undergoing moderate risk surgeries.

Examples of surgeries that could utilize the ClearSight system:

- Abdominal surgery – including colorectal
- Major general surgery – including oncological procedures
- Thoracic surgery – one-lung ventilation, induced pneumothorax
- Orthopedic surgery – fractured hip, joint replacement, spine
- Bariatric surgery
- Obstetric and extensive gynecological surgery
- Urology – radical and/or high risk of bleeding procedures

ClearSight System Limitations

The ClearSight technology performance may be affected in critically ill patients where flow to the finger could be compromised. This may occur in patients who:

- Are peripherally constricted secondary to a compensated shock state or hypothermia
- Are peripherally constricted secondary to high-dose vasopressors
- Have a mechanical obstruction to the hand or arm
Basic Monitoring

ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972
Physiologic Pressure Monitoring

Pressure monitoring is a basic tool in the armament of the clinician monitoring the critically ill patient. Disposable pressure transducers (DPT) convert a mechanical physiologic signal (i.e. arterial, central venous pressure, pulmonary artery pressure, intra-cranial pressure) to an electrical signal which is amplified and filtered and displayed on a bedside physiologic monitor in both a waveform and numeric value in mmHg.

Components of a Physiologic Pressure Measurement System

- Invasive catheter
- Edwards TruWave kit
- Normal saline flush solution (500 or 1000mL) (Heparin per institutional policy)
- Pressure infusion bag ( Appropriately sized for flush solution bag)
- Reusable pressure cable specific to TruWave transducer and bedside physiologic monitor
- Bedside physiologic monitor
Observation of best practices in set-up, calibration, and maintenance of a physiologic pressure transducer system is crucial in obtaining the most accurate pressure readings from which diagnosis and interventions are made.

**Best Practice in Setting Up a Physiologic Pressure Measurement System for Intravascular Monitoring**

1. Wash hands

2. Open TruWave disposable pressure transducer packaging and inspect contents. Replace all caps with non-vented caps and ensure that all connections are tight

3. Remove the TruWave transducer from its packaging and insert into an Edwards Lifesciences mounting back-plate that is secured on an IV pole

4. To de-air and prime IV flush bag and TruWave transducer: Invert normal saline bag (anticoagulation per institution policy). Spike IV bag with fluid administration set, keeping drip chamber upright. While keeping IV bag inverted, gently squeeze air out of bag with one hand while pulling flush (Snap-tab) with the other hand until air is emptied from IV bag and drip chamber is filled to desired level (½ or full)

5. Insert flush bag into pressure infuser bag (DO NOT INFLATE) and hang on IV pole at least 2 feet (60cm) above the transducer
6. With gravity only (no pressure in Pressure Bag), flush TruWave transducer holding pressure tubing in upright position as the column of fluid raises through the tubing, pushing air out of the pressure tubing until the fluid reaches the end of the tubing (flushing under pressure creates turbulence and increased occurrence of bubbles)

7. Pressurize the pressure bag until it reaches 300 mmHg

8. Fast-flush transducer tubing while tapping on tubing and stopcocks to remove any residual bubbles

9. Connect non-disposable pressure cable that is compatible with bedside monitor to disposable pressure transducer and bedside monitor

10. Connect tubing to arterial catheter, and then aspirate and flush system to assure catheter is intra-vascular and remove residual bubbles

11. Level the stopcock just above the TruWave transducer to the phlebostatic axis

12. Open the stopcock to atmospheric air. Zero pressure, per bedside monitor’s instructions for use

13. Inspect pressure trace on bedside monitoring screen to confirm appropriate pressure scale, alarm settings, pressure label, color coding, and physiologic waveform is present
Best Practice in Leveling and Zeroing a Physiologic Pressure Transducer System

1. Level the transducer’s closest stopcock (Vent port) to the physiologic pressure source. Intra-vascular monitoring should be level to the heart or the phlebostatic axis (fourth intercostal space at the chest’s anterior-posterior midpoint). This removes the effects of hydrostatic pressure on the pressure transducer.

2. Leveling should be performed with a carpenter’s level or a laser leveler (PhysioTrac laser leveler). Leveling by visual estimation is not recommended as it is proven to be unreliable with significant inter-user variability.

3. Zero referencing eliminates the effects of atmospheric and hydrostatic pressure.

4. Open the reference stopcock to air by removing the non-vented cap, keeping sterility intact.

5. After removing non-vented cap, turn stopcock off to the patient.

6. Initiate “Zero” function on bedside monitor and confirm pressure waveform and numeric value display 0 mmHg.

7. Once the “zero” is observed, turn the stopcock back to the vent port and replace the non-vented cap.
Best Practice in Maintaining Physiologic Pressure Transducer System

- **Keep transducers level:**
  Re-level transducer whenever the patient’s height or position changes in relation with transducer

- **Re-zero transducer:**
  Periodic zeroing of physiologic pressure transducer every 8 – 12 hours

- **Check pressure infuser bag:**
  Maintain a pressure of 300 mmHg to assure constant flow of flush solution and system fidelity

- **Check flush bag volume:**
  Change < ¼ full to assure constant flow of flush solution and system fidelity

- **Check system integrity:**
  Assure system is free of bubbles that may develop over time, stopcocks are properly aligned, connections are tight, and catheter is free from kinking

- **Check frequency response:**
  Perform square wave test every 8 – 12 hours to assess for over or under damping of system
Impact of Improper Leveling on Pressure Readings

Intravascular pressure readings may have error introduced if alignment with the phlebostatic axis is not maintained. The amount of error introduced is dependent upon the degree of offset.

For every inch (2.5 cm) the heart is offset from the reference point of the transducer, a 2 mmHg of error will be introduced.

Heart aligned with transducer = 0 mmHg error

Heart 10” (25cm) LOWER than transducer = Pressure 20 mmHg erroneously LOW

Heart 10” (25cm) HIGHER than transducer = Pressure 20 mmHg erroneously HIGH
Waveform Fidelity and Optimal Frequency Response

All physiologic pressure transducers are damped. Optimal damping results in a waveform and displayed value that is physiologically correct.

An overdamped physiologic pressure system will result in an underestimated systolic pressure and an overestimated diastolic pressure.

An underdamped physiologic pressure system will result in an overestimation of systolic pressure and an under estimation of diastolic pressure.

A square wave test can be used as a simple method of evaluating the frequency response at the bedside.

*Note: See page 54 for further information and examples of square wave tests.*
Pressure Monitoring Systems

This schematic identifies the components of a standard pressure monitoring system. The Edwards Swan-Ganz catheter and arterial catheter can be attached to a pressure monitoring line. The tubing must be non-compliant to accurately transmit the patient’s pressure waves to the transducer. The disposable pressure transducer is kept patent by a pressurized solution (300 mmHg). An integral flush device with a restrictor limits the flow rate to approximately 3 mL/hour for adults. Typically, heparinized normal saline is used as the flush solution with a range of heparin from 0.25u/1mL to 2u/1mL ratio. Non-heparinized solution has been used with patients with a sensitivity to heparin.

1. TruWave Transducers
2. Normal Saline flush bag in pressure bag
3. Radial Arterial line
4. Swan-Ganz catheter PA and RA ports
5. TruWave pressure cable / trifurcated
6. Bedside monitor
7. Trifurcated fluid administration line
Determining Dynamic Response

Optimal pressure monitoring requires a pressure system that accurately reproduces the physiologic signals applied to it. Dynamic response characteristics of the system include the natural frequency and damping coefficient. Activate the flush device to perform a square wave test in order to measure the natural frequency and calculate the amplitude ratio.

**Perform a Square Wave Test**

Activate the flush device by pulling the snap tab or pull tab. Observe the bedside monitor. The waveform will sharply rise and “square off” at the top. Observe the tracing as it returns to baseline.

**Calculate the Natural Response (fn)**

Estimated by measuring the time of one full oscillation (mm).

\[ fn = \frac{\text{paper speed (mm/sec)}}{\text{oscillation width/mm}} \]
**Determine the Amplitude Ratio**

Estimate by measuring the amplitudes of two consecutive oscillations to determine an amplitude ratio, $A_2 / A_1$.

**Plot to Determine Damping Coefficient**

Plot the natural frequency ($f_n$) against the amplitude ratio to determine the damping coefficient. The amplitude ratio is on the right and the damping coefficient is on the left.

---

**Simple Evaluation of Dynamic Response**

Determining the dynamic response characteristics of a pressure monitoring system by calculating the amplitude ratio and damping coefficient may not be feasible at the bedside when a rapid assessment of the waveform is required. A simple evaluation of dynamic response can be obtained by performing a square wave test and by observing the resultant oscillations. In order to perform this assessment accurately, a flush device that can be activated rapidly and then released is required. A flush device that does not close rapidly after activation (squeeze or press type) may not close the restrictor quickly and may produce erroneous results.
Square Wave Testing

1. Activate snap or pull tab on flush device
2. Observe square wave generated on bedside monitor
3. Count oscillations after square wave
4. Observe distance between the oscillations

Optimally Damped:
1.5 – 2 oscillations before returning to tracing. Values obtained are accurate.

Underdamped:
> 2 oscillations. Overestimated systolic pressure, diastolic pressures may be underestimated.

Overdamped:
< 1.5 oscillations. Underestimation of systolic pressures, diastolic may not be affected.
Measuring Technique

*Hydrostatic Zero Reference*

To obtain accurate pressure measurements, the level of the air-fluid interface must be aligned with the chamber or vessel being measured.

The phlebostatic axis has been well defined as the appropriate landmark for intracardiac pressures. The phlebostatic axis has most recently been defined as the bisection of the 4th intercostal space at the mid-point between the anterior and posterior chest wall.

Physiologic pressures are measured relative to the atmospheric pressure. Therefore, the transducer must be zeroed to the atmospheric pressure to eliminate its impact on the readings. Hydrostatic pressure occurs when the level of the zeroing stopcock is not in alignment with the phlebostatic axis.

The phlebostatic axis is used for both intracardiac and intra-arterial pressure monitoring. Accurate values can be obtained with the patient supine and with the head of bed up to 45 to 60 degrees as long as the zeroing stopcock has been aligned with the phlebostatic axis.
Intra-arterial Monitoring

*Components of the Arterial Pulse*

**Peak systolic pressure:** begins with opening of aortic valve. This reflects maximum left ventricular systolic pressure and may be termed the ascending limb

**Dicrotic notch:** reflects closure of the aortic valve, marking the end of systole and the onset of diastole

**Diastolic pressure:** relates to the level of vessel recoil or amount of vasoconstriction in the arterial system. May be termed the descending limb

**Anacrotic notch:** A presystolic rise may be seen during the first phase of ventricular systole (isovolumetric contraction). The anacrotic notch will occur before the opening of the aortic valve

**Pulse pressure:** difference between systolic and diastolic pressure

**Mean arterial pressure:** average pressure in the arterial system during a complete cardiac cycle. Systole requires one-third of the cardiac cycle, diastole normally during two-thirds. This timing relationship is reflected in the equation for calculating MAP. \( MAP = SP + (2DP)/3 \)

*Bedside physiologic monitors use various algorithms to incorporate the area under the curve for determining the mean pressure.*
### ABNORMAL ARTERIAL PRESSURE WAVEFORMS

<table>
<thead>
<tr>
<th>Waveform Description</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated systolic pressure</td>
<td>Systemic hypertension, Arteriosclerosis, Aortic insufficiency</td>
</tr>
<tr>
<td>Decreased systolic pressure</td>
<td>Aortic stenosis, Heart failure, Hypovolemia</td>
</tr>
<tr>
<td>Widened pulse pressure</td>
<td>Systemic hypertension, Aortic insufficiency</td>
</tr>
<tr>
<td>Narrowed pulse pressure</td>
<td>Cardiac tamponade, Congestive heart failure, Cardiogenic shock, Aortic stenosis</td>
</tr>
<tr>
<td>Pulsus bisferiens</td>
<td>Aortic insufficiency, Obstructive hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Pulsus paradoxus</td>
<td>Cardiac tamponade, Chronic obstructive airway disease, Pulmonary embolism</td>
</tr>
<tr>
<td>Pulsus alternans</td>
<td>Congestive heart failure, Cardiomyopathy</td>
</tr>
</tbody>
</table>
Central Venous Access

Types of Central Venous Access Devices

A central venous catheter (CVC) is, by definition, a catheter whose tip resides in the central circulation. There are many types: tunneled, non-tunneled/percutaneously inserted, peripherally inserted, and implanted. The following will focus on the non-tunneled/percutaneously inserted central venous catheters. CVCs come in multiple configurations to facilitate volume resuscitation, simultaneous administration of multiple medications, as well as monitoring of central venous pressure. In addition, CVCs are manufactured with different materials and coatings to mitigate thrombogenicity, as well as catheter-related blood stream infections.

Multi-lumen catheters allow for multiple therapies and monitoring to be performed through a single venous access insertion site, and are often seen in the critical care environment. They are often inserted for intermittent or continuous infusion of multiple medications or fluid as well as intermittent or continuous central venous pressure measurements. These multi-lumen catheters are used for the administration of blood products, crystalloids, colloids, medications and nutritional therapies. Increasing the number of lumens with the same size outer diameter catheter (French size) may decrease the individual lumen size, or increases the reported gauge available, therefore, decreasing potential flow through the lumen.

Introducers are used to direct and place intravascular catheters, especially pulmonary artery catheters (PAC), within a designated blood vessel. They may be left in place to serve as a central venous access after removal of the PAC. Introducers may be used by themselves as a large bore central venous catheter for rapid volume resuscitation.

Advanced Venous Access (AVA) devices combine the ability of a sheath introducer to insert a pulmonary artery catheter and to infuse multiple fluids in one multipurpose device.
Applications of Central Venous Access Devices

- Rapid fluid administration – for example, in cases of, or at high risk of, high blood loss
  - Multiple trauma
  - Complex orthopedic surgery
  - Large vascular surgery
  - Extensive abdominal surgery
  - Tumor de-bulking
  - Sepsis
  - Burns

- Administration of IV fluids requiring dilution within the central circulation to avoid vascular damage (i.e., chemotherapy, total parenteral nutrition)

- Administration of vasoactive and/or incompatible drugs

- Frequent blood sampling (in patients without an arterial line) and/or blood administration therapies

- Chronically ill patients in whom peripheral IV access is limited or unavailable

- Central venous pressure (CVP) monitoring for assessment of intravascular fluid status

- Measurement of oxygen saturation levels in blood returning to the heart (ScvO₂)

- Monitoring and access for either pre- or post-pulmonary artery catheter insertion (same insertion site)
Relative Contraindications may Include Patients with

- Recurrent sepsis
- Hypercoagulable state where catheter could serve as a focus for septic or bland thrombus formation
- Heparin coated catheters where patients have a known sensitivity to heparin

Complications

- Carotid artery puncture or cannulation secondary to the proximity of the internal jugular
- Pneumothorax (air in plural space collapsing lung), internal jugular (IJ) approach has a lower incidence of a pneumothorax than a sub-clavian or low anterior (IJ) approach. Patients with overinflated lungs (i.e., COPD or PEEP) may have an elevated risk of pneumothorax especially with a sub-clavian approach
- Hemothorax (blood in plural space collapsing lung), secondary artery puncture or laceration
- Hemorrhage within chest (hemothorax, tamponade) or from insertion site
- Thoracic duct puncture or laceration
- Air embolism, increased risk in patients who are spontaneously breathing (negative pressure) as opposed to mechanical ventilation (positive pressure)
- In-situ complications; vessel damage, hematoma, thrombosis, dysrhythmia, cardiac perforation, catheter migration SVC to RA, or extravascular
Mitigating Complications

Mitigating catheter-related bloodstream infections:

- Hand hygiene
- Chlorhexidine skin antisepsis
- Sterile gown and gloves with hat and mask
- Maximal barrier precautions upon insertion
- Optimal catheter site selection, with subclavian veins as the preferred site

Mitigating inadvertent carotid puncture/cannulation, multiple sticks

- Ultrasound guided central line placement

Note: The tip of a CVC should never be placed within the right atrium due to the risk of cardiac perforation resulting in a tamponade.
Central Venous Catheter Specifics

*Polyurethane (Commonly Used for Catheter Body):*

- Tensile strength, which allows for thinner wall construction and smaller external diameter
- High degree of biocompatibility, kink and thrombus resistance
- Ability to soften within the body

*Lumens and Functionality:*

- More than one lumen increases the functionality of the CVC insertion single site
- Multi-lumen catheters may be more prone to infection because of increased trauma at the insertion site or because multiple ports increase the frequency of manipulation
- Quad or triple lumen 8.5 French (Fr) catheters have more functional ports but are usually of a smaller lumen (i.e., 8.5 Fr 18/18/18/16 gauge vs. 8.5 Fr 15/14 gauges)
- Double lumen 8.5 French (Fr) catheters have larger lumens which are useful for rapid volume resuscitation but have limited number of functional ports (i.e., 8.5 Fr 18/18/18/15 gauges vs. 8.5 Fr 15/14 gauges)
**Flow Characteristics**

- Primarily determined by a catheter’s internal diameter and length, but also affected by driving pressure (IV height or pressure infuser bag) as well as fluid viscosity (i.e., crystalloid vs. blood)
- Larger lumens are often used for higher viscosity fluids to increase flow (i.e., TPN and blood)

Flow rates are usually calculated with normal saline at a head height of 40” (101.6 cm).

**Length**

Central venous catheters come in varying lengths, the most common of which are between 15 – 20 cm. Required length is dependent upon patient size and site of insertion to reach the desired catheter tip location approximately 2 cm proximal to the right atrium.

**Solution for Excess Catheter, Box Clamp**

When catheter placement is achieved with excess catheter between the backform and site of insertion a box-clamp can be employed to anchor and secure the catheter at the site of insertion. This prevents catheter pistoning in-and-out of the skin and decreases chance of infection.
Lumen Designations and Infusion Rates

**CVC PORT DESIGNATION**

<table>
<thead>
<tr>
<th>Distal (or largest gauge)</th>
<th>Medial</th>
<th>Proximal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood administration</td>
<td>TPN or medications</td>
<td>Medication administration</td>
</tr>
<tr>
<td>High volume fluids</td>
<td></td>
<td>Blood sampling</td>
</tr>
<tr>
<td>Colloid fluid administration</td>
<td></td>
<td>Drug therapy</td>
</tr>
<tr>
<td>Drug therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVP monitoring</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*These are suggestions only.

**CVC PORT COLOR DESIGNATION**

<table>
<thead>
<tr>
<th>Port</th>
<th>Double</th>
<th>Triple</th>
<th>Quad</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal</td>
<td>White</td>
<td>White</td>
<td>White</td>
</tr>
<tr>
<td>Medial (1)</td>
<td>Blue</td>
<td>Blue</td>
<td>Blue</td>
</tr>
<tr>
<td>Medial (2)</td>
<td></td>
<td></td>
<td>Gray</td>
</tr>
<tr>
<td>Distal</td>
<td>Brown</td>
<td>Brown</td>
<td>Brown</td>
</tr>
</tbody>
</table>

**CVC INFUSION RATES**

7 Fr Double Lumen and Triple Lumen Polyurethane Multi-Med Catheters

**AVERAGE PERFORMANCE FLOW RATE**

<table>
<thead>
<tr>
<th>Catheter</th>
<th>16 cm Length (mL/hr)</th>
<th>20 cm Length (mL/hr)</th>
<th>Cross-Section Gauge Equivalence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Triple Lumen</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal</td>
<td>1670</td>
<td>1420</td>
<td>18</td>
</tr>
<tr>
<td>Medial</td>
<td>1500</td>
<td>1300</td>
<td>18</td>
</tr>
<tr>
<td>Distal</td>
<td>3510</td>
<td>3160</td>
<td>16</td>
</tr>
<tr>
<td><strong>Double Lumen</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal</td>
<td>3620</td>
<td>3200</td>
<td>16</td>
</tr>
<tr>
<td>Distal</td>
<td>3608</td>
<td>3292</td>
<td>16</td>
</tr>
</tbody>
</table>

*Average flow rates shown are normal saline infusion, room temperature and 101.6 cm head height.
Infection Mitigation

Coatings

Catheter coatings may include the bonding of the catheter surface with antimicrobial and/or antiseptic agents to decrease catheter-related infection and thrombotic complications. Heparin-bonding process is one example; other agents reported in the literature include antibiotics such as minocycline and rifampin, or antiseptic agents like chlorhexidine and silver sulfadiazine.

“Oligon” Antimicrobial Catheter Material

Materials, in particular metals, that are antimicrobial in minute amounts are called oligodynamic. One of the most potent of these is silver, with the antimicrobial form being silver ions. The bactericidal action of silver ions is effective against a broad spectrum of bacteria, including the common strains which cause infection and the more virulent antibiotic-resistant strains. Silver has been in medical use for decades and was used in systemic drugs before the advent of antibiotics. Today, silver is used routinely in antibacterial salves (silver sulfadiazine), to prevent infection and blindness in newborns (silver nitrate), and in medical devices and catheters.

Antibiotic- and antiseptic-coated catheters have demonstrated reduced rates of catheter colonization and associated bloodstream infection in some clinical trials, but it is important to remember that heparin-induced thrombocytopenia and/or allergy to the antibiotic used on a catheter could result in patient morbidity.

Catheter and Accessory Features

- Soft tip to avoid injury or perforation
- Radiopaque for radiographic visualization in determining catheter placement
- Depth markings on all catheters and guidewires
Introducers as a Central Line

Sometimes an introducer is used for central venous access when rapid volume resuscitation is needed or is left in place following the removal of a pulmonary artery catheter. Components of the introducer system usually include:

- Flexible polyurethane sheath
- Guidewire and dilator
- Side port
- Hemostasis valve

After insertion, the guidewire and dilator are removed, leaving the sheath in place. Fluids may be administered through the side port, while the hemostasis valve prevents bleedback and/or air embolization.

A single-lumen infusion catheter can be used with the introducer, placed through the hemostasis valve (after swabbing the valve with betadine), to convert to a double-lumen access. An obturator should be used to safely occlude the lumen as well as to prevent air entry when the catheter is not in place.
**Infusion Catheter**

The infusion catheter is a two-piece assembly consisting of an infusion catheter and a stylet. With the stylet removed, the infusion catheter permits access to the central venous circulation via a percutaneous sheath introducer. The infusion catheter is indicated for use in patients requiring administration of solutions, blood sampling and central venous pressure monitoring. With the stylet in place, the product serves as an obturator, ensuring patency of the introducer valve and sheath.
Insertion Sites

Typically, central venous catheters are inserted via the subclavian or internal jugular (IJ) veins. The subclavian vein begins at the lateral border of the first rib and arches through the space between the first rib and clavicle. It joins the internal jugular to become the innominate (or brachiocephalic) vein, which then flows into the superior vena cava to the heart. The subclavian vein can be approached either infraclavicularly (below the clavicle) or supraclavicularly (above the clavicle). Alternative sites include the external jugular and femoral veins.

Note the natural “windows” for supraclavicular venipuncture: 1) supraclavicular triangle formed by the clavicle, trapezius, and sternocleidomastoid muscles; 2) clavicular sternocleidomastoid triangle formed by the two bellies of the sternocleidomastoid muscle and the clavicle.
Note the close proximity of arterial and venous structure. Venipunctures in the lateral region of the clavicle are more prone to arterial puncture, brachial plexus injury, and pneumothorax. Note the prominent thoracic duct and higher apex of the lung on the left and the perpendicular entry of the left IJ into the left subclavian vein.
Catheter Tip Placement

Central venous catheters should be inserted so that the tip is approximately 2 cm proximal to the right atrium (for right-sided approaches) and similarly placed or well within the innominate vein (for left-sided approaches), with the tip parallel with the vessel wall. A chest x-ray must be done post insertion, as it provides the only definitive evidence for catheter tip location.

Probably the most important factor in the prevention of complications is the location of the catheter’s tip. The pericardium extends for some distance cephalad along the ascending aorta and superior vena cava. In order to guarantee an extrapericardial location, the catheter’s tip should not be advanced beyond the innominate vein or the initial segment of the superior vena cava. (It is important to note that a portion of the superior vena cava lies within the pericardium.)

Some practitioners may prefer a deep SVC placement (within the lower third of the SVC), but nearly half the length of the SVC is covered by pericardial reflection that slopes downward toward its lateral edge. To avoid the risk of arrhythmias and tamponade, the tip of a CVC should lie above this reflection and not in the right atrium.

Tips to assure catheter tip not extravascular or against a wall might include:

- Syringe aspiration yields blood freely
- Venous pressure fluctuates with respiration
- Advancement of the catheter is unhindered
Monitoring Central Venous Pressure

Central venous pressure (CVP) measurements are widely used in both medical and surgical patients as a simple and easily available guide to fluid therapy after hemorrhage, accidental and surgical trauma, sepsis and emergency conditions associated with blood volume deficits.

Central venous catheters are used to measure the pressure under which the blood is returned to the right atrium and to give an assessment of the intraventricular volume and right heart function. The CVP is a useful monitor if the factors affecting it are recognized and its limitations are understood. Serial measurements are more useful than individual values, and the response of the CVP to a volume infusion is a useful test of right ventricular function. The CVP does not give any direct indication of left heart filling but may be used as a crude estimate of left-sided pressures in patients with good left ventricular function. Preload, or the volume status of the heart, has been measured as CVP or PAOP, for the right and left ventricles, respectively.

However, there are many factors that influence CVP values, for example, cardiac performance, blood volume, vascular tone, intrinsic venous tone, increased inta-abdominal or intrathoracic pressures and vasopressor therapy. Therefore using CVP to assess either preload or volume status of the patient may be unreliable.
Normal CVP Waveform

Waveforms seen on the monitor reflect the intracardiac events. The normal CVP waveform consists of three peaks (a, c and v waves) and two descents (x and y). The a wave represents atrial contraction and follows the P wave on the ECG trace. This is the atrial kick that loads the right ventricle just prior to contraction. As atrial pressure decreases, a c wave, resulting from closure of the tricuspid valve, may be seen. The x descent represents the continually decreasing atrial pressure. The v wave represents the atrial events during ventricular contraction — passive atrial filling — and follows the T wave on the ECG. When the atrial pressure is sufficient, the tricuspid valve opens, and the y descent occurs. Then the cycle repeats.
Accurate recognition of these waves requires that they be aligned with an ECG trace. As mechanical events follow electrical events, the waveforms can be identified by lining them up with the ECG events.

**CVP WAVEFORM**

*Reading CVP waveforms with spontaneous inspiratory artifact*

![CVP Waveform Diagram]

- **A wave**
- **V wave**
- **Location of A and V waves at end expiration**
- **Inspiratory artifact**
Advanced
Minimally Invasive Monitoring

ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972
The FloTrac System Algorithm

Arterial Pressure-Based Cardiac Output

The Edwards FloTrac system algorithm is based on the principle that aortic pulse pressure is proportional to stroke volume (SV) and inversely related to aortic compliance.

Standard Deviation of Arterial Pressure

Initially, the FloTrac system algorithm assesses pulse pressure by using the standard deviation of the arterial pressure ($\sigma_{AP}$) around the MAP value, measured in mmHg, making it independent of the effects of vascular tone. This standard deviation of the pulse pressure is proportional to the volume displaced or the stroke volume. This is calculated by analyzing the arterial pressure waveform over 20 seconds at 100 times per second, creating 2,000 data points from which $\sigma_{AP}$ is calculated.

Traditional: CO = HR * SV
FloTrac system:

$$APCO = PR \times (\sigma_{AP} \times \chi)$$

Where $\chi = M(HR, \sigma_{AP}, C(P), BSA, MAP, \mu_{3ap}, \mu_{4ap} \ldots)$

$\sigma_{AP}$ = standard deviation of arterial pulse pressure in mmHg is proportional to pulse pressure.

$Khi(\chi)$ = scaling multivariate parameter proportional to the effects of vascular tone on pulse pressure.

$M =$ multivariate polynomial equation.

BSA = body surface area calculated by Dubois’ equation for body surface area.

MAP = mean arterial pressure calculated by taking sum of sampled pressure point values over 20 seconds and dividing it by the number of pressure points.
μ = statistical moments determined by skewness (symmetry) and kurtosis (distinctness of a peak) calculated along several mathematical derivatives.

$$\text{APCO} = \text{PR} \times \frac{\text{sd(AP)}}{\chi}$$

- Measures pulse rate
- Beats identified by upslope of waveforms
- Pulse rate computed from time period of beats
- Based upon the basic physiological principle of pulse pressure’s (PP) proportionality to SV
- sd(AP)* utilized to compute a robust assessment of key PP characteristics
- Computed on a beat by beat basis
- Compensates for differences in vascular tone (compliance and resistance)
- Patient-to-patient differences estimated from biometric data
- Dynamic changes estimated by data and waveform analysis

**Khi (χ) and the Conversion of mmHg to mL/beat**

The conversion of standard deviation of arterial pressures (mmHg) into mL/beat is performed by multiplying it by a conversion factor known as *Khi (χ)*. *Khi* is a multivariate polynomial equation which assesses the impact of the patient’s ever-changing vascular tone on pulse pressure. *Khi* is calculated by analyzing the patient’s pulse rate, mean arterial pressure, standard deviation of mean arterial pressure, large-vessel compliance as estimated by patient demographics, and skewness and kurtosis of the arterial waveform. *Khi* is updated and applied to the FloTrac system algorithm on a rolling 60-second average.
• **Pulse rate**: The patient’s pulse rate is calculated by counting the number of pulsations in a 20-second period and extrapolated to a per minute value.

• **Mean arterial pressure (MAP)**: An increase in average pressure often indicates an increase in resistance, and vice versa.

• **Standard deviation of arterial pressure ($\sigma_{AP}$)**: Pulse pressure is proportional to $\sigma_{AP}$ and to stroke volume. Increases and decreases in the standard deviation also provide information on pressure amplitude. When this pressure amplitude is correlated with kurtosis, it compensates for differential compliance and wave reflectance that vary from one arterial location to another. This then allows the monitoring of cardiac output from different arterial locations.

• **Large vessel compliance**: Work reported by Langewouters found a direct correlation among age, gender, and MAP with respect to aortic compliance. An equation was derived from these studies by which a patient’s compliance could be estimated with the inputs of age and gender. According to Langewouters et al, the arterial compliance ($C$), as a function of pressure, could be estimated using the following equation:

$$C(P) = L \cdot \frac{A_{\max}}{\pi \cdot P_1} \cdot \frac{1}{1 + \left(\frac{P - P_0}{P_1}\right)^2}$$

$L = \text{estimated aortic length}$

$A_{\max} = \text{aortic root cross sectional area maximum}$

$P = \text{arterial pressure}$

$P_0 = \text{pressure at which compliance reaches its maximum}$

$P_1 = \text{the width of compliance curve at half of maximum compliance}$

Additional measures of weight and height (BSA) were also found to correlate with vascular tone and were added to enhance the calculation of aortic compliance.
• **Skewness (a measure for lack of symmetry, \( \mu_3 \)):**

Symmetry characteristics on arterial pressure can indicate a change in vascular tone and/or resistance. Two different functions may have the same mean and standard deviation but will rarely have the same skewness. For example, an arterial pressure waveform in which the data points increase quickly in systole and fall slowly can result as an increase in vasoconstriction and would have increased skewness.

![Decreased Skewness](image1)

Decreased Skewness
Low Resistance

![Increased Skewness](image2)

Increased Skewness
Constant MAP
High Resistance
Kurtosis (a measure of how peaked or flat the pressure data points are distributed from normal distribution, $\mu_{4\text{ap}}$): Pressure data with high kurtosis has the pressure rise and fall very quickly relative to the normal pulse pressure and can be directly associated with large vessel compliance.

1) A high kurtosis value will indicate a distinct peak near the mean, with a drop thereafter, followed by a heavy “tail.”
2) A low kurtosis value will tend to indicate that the function is relatively flat in the region of its peak and suggests decreased central tone, as is often seen, for example, in the neonatal vasculature.

$Khi$ ($\chi$) mmHg to mL/beat

Taking all of these variables into consideration, the FloTrac system algorithm continuously assesses the impact of vascular tone on pressure every 60 seconds. The result of the analysis is a conversion factor known as $Khi$ ($\chi$). $Khi$ is then multiplied by the standard deviation of the arterial pressure to calculate stroke volume in milliliters per beat. This stroke volume is multiplied by the pulse rate to obtain cardiac output in liters per minute.

Stroke Volume (mL/beat) = $\sigma_{AP}$ (mmHg) * $\chi$ (mL/mmHg)
No Manual Calibration Needed

Other arterial pressure cardiac output devices (pulse contour or pulse power) require calibration as they cannot auto correct for the patient’s changing vascular tone. Since the FloTrac system algorithm continuously adjusts for the patient’s ever-changing vascular tone, it does not require manual calibration. As a component of the calibration, \( Khi \) auto corrects for changes in vascular tone through a complex waveform analysis. This feature also eliminates the need for a central or peripheral venous line, required for indicator dilution methods used in manual calibration.

Technical Considerations

The FloTrac system algorithm is dependent upon a high fidelity pressure tracing. Attention to best practice in pressure monitoring is important by: priming with gravity, pressure bag kept to 300 mmHg, adequate I.V. bag flush volume, sensor stopcock is kept level to phlebostatic axis, and periodic testing of optimal dampening with a square wave test. FloTrac sensor kits are especially configured to optimize frequency response therefore adding additional pressure tubing or stopcocks is highly discouraged.
The FloTrac System 4.0

The FloTrac system algorithm has evolved based on a broad and expanding patient database that allows ongoing system performance improvements. In this latest evolution (v.4.0), Edwards continues to expand the database to include a more diverse surgical patient population in order to continuously inform and evolve the algorithm. Specifically, more of the following high-risk surgical patients were added to the database including, but not limited to gastrointestinal, esophageal, pancreaticoduodenectomy (whipple), kidney transplant, nephrectomy, hip replacement and esophagectomy. The expanded patient database has informed the algorithm to recognize and adjust for more patient conditions.

These updates are in addition to changes made in FloTrac systems 3rd generation software which continuously assess the arterial waveform for characteristic changes associated with hyperdynamic and vasodilated conditions. As part of this effort, additional physiologically-based variables (see image below) were added to the algorithm’s vascular tone Khi factor in order to adjust automatically for hyperdynamic and vasodilated patients. Once identified it accesses a specially designed algorithm to account for such conditions.
In addition to a broader database the FloTrac System 4.0 algorithm adjusts for rapid changes in pressure that occur during vasopressor administration through Khi-fast. Khi-fast is assessed every 20 seconds and is inversely affected by pressure. Khi continues to assess vascular tone every 60 seconds and Khi-fast every 20 seconds resulting in a more physiologic response to changes in resistance.

FloTrac System Algorithm Evolution

1st Generation Algorithm
- Introduced Automatic Vascular Tone Adjustment (10 min avg)
- Data Base Patients: primarily cardiac patients

2nd Generation Algorithm
- Improved Automatic Vascular Tone Adjustment (1 min avg)
- Added fluid optimization screen enhancements
- Data Base Patients: includes high risk surgical patients

3rd Generation Algorithm
- Adjusted for hyperdynamic patients
- Includes certain sepsis patients and liver resection

Limited Release Algorithm (Enhanced SVV)
- Adjusted for certain types of arrhythmias

FloTrac System 4.0 Algorithm
- CO/SV better matches physiology after vasopressors

FloTrac Sensor Setup

1. Open FloTrac sensor packaging and inspect contents. Replace all caps with non-vented caps and ensure that all connections are tight.

2. Remove the FloTrac sensor from packaging and insert into an Edwards Lifesciences mounting back-plate that is secured on an I.V. pole.

3. **To de-air and prime I.V. bag and FloTrac sensor**: Invert normal saline I.V. bag (anticoagulation per institution policy). Spike I.V. bag with fluid administration set, keeping drip chamber upright. While keeping I.V. bag inverted, gently squeeze air out of bag with one hand while pulling flush tab with the other hand until air is emptied from I.V. bag and drip chamber is filled half-way.

4. Insert I.V. bag into the **Pressure Bag** and hang on I.V. pole **(do not inflate)**.

5. With gravity only (**no pressure in Pressure Bag**), flush FloTrac sensor holding pressure tubing in upright position as the column of fluid raises through the tubing, pushing air out of the pressure tubing until the fluid reaches the end of the tubing.
6. Pressurize the **Pressure Bag** until it reaches 300 mmHg.

7. Fast-flush the FloTrac sensor and tap on tubing and stopcocks to remove any residual bubbles.

8. Connect the **green** FloTrac connecting cable to the **green** capped connector on the FloTrac sensor. Then connect the opposite end of the cable to the FloTrac connection on the back of the Edwards monitor.

9. Connect the bedside monitor’s arterial pressure cable to the **red** cable connector on the FloTrac sensor.

10. Connect tubing to arterial catheter, then aspirate and flush system to assure no residual bubbles remain.

11. Level the FloTrac sensor to the phlebostatic axis. **Note:** *It is important to keep the FloTrac sensor level to the phlebostatic axis at all times to ensure accuracy of cardiac output.*

   - Open the stopcock to atmospheric air.
   - Select **Zero Arterial Pressure**, then select and press **Zero**.
   - **Zero** the arterial channel on the bedside monitor.

12. **Cardiac output will display within 40 seconds and will update every 20 seconds thereafter.**

13. Inspect arterial pressure trace on bedside monitoring screen or the waveform confirmation screen on the Edwards monitor.
1. Connect the power adapter and ethernet cable for both EV1000 panel and databox. Press the \( \uparrow \) button on the panel.

2. When the boot up is complete, enter new patient data (patient ID, gender, age, height, and weight) or continue same patient.

3. If entering new patient data, use the touch screen to select and enter values. Press Home to continue.

4. Connect the FloTrac trifurcated databox cable to the back of the EV1000 databox. Then connect the green FloTrac connecting cable to the green capped connector on the FloTrac sensor.

5. Connect the bedside monitor’s arterial pressure cable to the red cable connector on the FloTrac sensor.
6. Touch **Clinical Actions** and then touch **Zero & Waveform**.

7. Open the FloTrac sensor to atmospheric air. Touch -0- for arterial channel. Then touch **Home**. Close the FloTrac sensor to atmospheric air.

8. Cardiac output will be displayed within 40 seconds and will update every 20 seconds thereafter.

9. Monitor patient in real-time with one of the available screens.

10. Choose parameters to view on screen by touching outside of the parameter globe. Displayed parameters are outlined, whereas the selected parameters are circled with blue fill.

11. Visual targets and alarms can be set by touching inside parameters globe.
Vigileo Monitor Setup and Zeroing

1. Press the button on the front panel to turn the Vigileo monitor ON. The screen will display an opening message, indicating that a Power-On Self-Test (POST) is being performed.

2. When the POST is complete, patient information (gender, age, height, and weight) must be entered before cardiac output monitoring can occur.

3. Use the navigation knob to select and enter values. Press Continue to continue selection and open the Home screen.

4. Connect the FloTrac connecting cable to the FloTrac cable connector at the back of the Vigileo monitor. Align the arrows at the top of the cable connector on the monitor to the arrow on the FloTrac connecting cable.

5. Connect the other end of the FloTrac cable to the green capped FloTrac sensor.

6. Rotate navigation knob until the CO frame is outlined in yellow and then press the knob to open CO menu.
7. From the **CO Menu**, rotate the navigation knob until **Zero Arterial Pressure** is highlighted and then press the knob. The Zero Arterial Pressure screen will appear.

8. Open the FloTrac sensor to atmospheric air. Rotate the navigation knob on the Vigileo monitor to **Zero** and press the knob. Select **Return** to exit screen. Close the FloTrac sensor to atmospheric air.

9. Cardiac output will be displayed within 40 seconds after arterial pressure is registered by the FloTrac sensor.
Stroke Volume Variation

Trending Dynamic Parameters

Hemodynamic monitoring can be obtained continuously or intermittently and using either static or dynamic parameters. Static parameters are single snapshots taken at specific points in the cardiac or respiratory cycle. Dynamic parameters should be trended to assess rapid changes in the cardiovascular status over short periods of time. The table below shows examples of both static and dynamic parameters used to assess volume status and fluid responsiveness. Stroke volume variation (SVV) is a dynamic parameter and a sensitive indicator for preload responsiveness in controlled-ventilated patients.

**HEMODYNAMIC PARAMETERS FOR ASSESSING VOLUME STATUS AND FLUID RESPONSIVENESS**

<table>
<thead>
<tr>
<th>Static Parameters</th>
<th>Dynamic Parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial pulse pressure (NIBP)</td>
<td>Systolic pressure variation (SPV)</td>
</tr>
<tr>
<td>Mean arterial pressure (MAP)</td>
<td>Arterial pulse pressure variation (PPV)</td>
</tr>
<tr>
<td>Central venous pressure (CVP)</td>
<td>Stroke volume variation (SVV)</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure (PAOP)</td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td></td>
</tr>
<tr>
<td>Urine output</td>
<td></td>
</tr>
</tbody>
</table>
Advantages of Trending SVV with Cardiac Output

Clinicians understand the vital role of fluid balance in critically ill patients. Static pressure indicators such as those shown prior may not be sensitive enough to predict hypovolemia or a patient’s response to fluid administration. Instead, trending the flow-based parameters SVV and cardiac output together provides both an indication of fluid responsiveness and a means of verifying that fluid is beneficial to the patient’s status. The latest FloTrac system software gives the option of trending any two flow parameters, including SVV.

SVV uses calculations of left ventricular stroke volume from the pressure waveform to perform beat-to-beat analysis over the course of a breath. A number of studies have demonstrated the potential of SVV for predicting responsiveness to fluid challenge.

SVV is increasingly used to determine fluid responsiveness and to monitor the effects of volume therapy. Successful optimization is linked to improved patient outcomes including shorter hospital stays and lower morbidity rates. As a result, tools such as the FloTrac system are being adopted to provide insight into fluid optimization, blood flow and oxygen delivery.
The FloTrac system provides dynamic insight using an existing arterial catheter. The system includes advanced SVV trending screens that provide vital information enabling early action while complementing the clinical workflow.

Using Fluid to Improve Hemodynamics

“The ability of the SVV variable to predict the responsiveness to such a small volume load and the continuous measurement of SVV and SV are of utmost clinical importance . . . The receiver-operating curve (ROC) also demonstrated the superiority of SVV over SBP as a predictor of fluid responsiveness.” Berkenstadt
Calculating Stroke Volume Variation

Stroke volume variation is a naturally occurring phenomenon in which the arterial pulse pressure falls during inspiration and rises during expiration due to changes in intra-thoracic pressure secondary to negative pressure ventilation (spontaneously breathing). Variations over 10 mmHg have been referred to as pulsus paradoxus. The normal range of variation in spontaneously breathing patients has been reported between 5-10 mmHg.

Reverse pulsus paradoxus is the same phenomenon with controlled mechanical ventilation, however, in reverse. Arterial pressure rises during inspiration and falls during expiration due to changes in intra-thoracic pressure secondary to positive pressure ventilation. In addition to reverse pulsus paradoxus, it has also been referred to as paradoxical pulsus, respiratory paradox, systolic pressure variation and pulse pressure variation. Traditionally SVV is calculated by taking the SVmax – SVmin / SV mean over a respiratory cycle or other period of time.
SVV and Assessing Fluid Response

SVV and its comparable measurement, pulse pressure variation (PPV), are not indicators of actual preload but of relative preload responsiveness. SVV has been shown to have a very high sensitivity and specificity when compared to traditional indicators of volume status (HR, MAP, CVP, PAD, PAOP), and their ability to determine fluid responsiveness. The following table of studies demonstrates SVV sensitivity and specificity in predicting fluid responsiveness against a specified infused volume and defined criteria for a fluid responder.

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients</th>
<th>Volume</th>
<th>Tidal Volume mL/Kg</th>
<th>Parameters Tested (Artery)</th>
<th>R²</th>
<th>Def. of Responder</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Michard</td>
<td>Sepsis</td>
<td>500 mL</td>
<td>8 to 12</td>
<td>Δ PP (R or F)</td>
<td>0.85</td>
<td>Δ CO ≥ 15%</td>
<td>94</td>
<td>96</td>
</tr>
<tr>
<td>Berkenstadt, et al</td>
<td>Neuro Surgery</td>
<td>100 mL</td>
<td>10</td>
<td>Δ SVV</td>
<td>0.53</td>
<td>Δ SV ≥ 5%</td>
<td>79</td>
<td>93</td>
</tr>
<tr>
<td>Reuter, et al</td>
<td>Cardiac</td>
<td>10 x BMI</td>
<td>10</td>
<td>Δ SVV</td>
<td>0.64</td>
<td>Δ SV ≥ 5%</td>
<td>79</td>
<td>85</td>
</tr>
</tbody>
</table>

Application of SVV

Normal SVV values are less than 10-15% on controlled mechanical ventilation. The following figures demonstrate using SVV as a guide for volume resuscitation with a goal SVV of < 13%. SVV increased to 19% with a stroke volume (SV) of 43 mL/beat, blood and normal saline were given to obtain a SVV of 6% and a SV of 58 mL/beat.
Stroke Volume Variation Limitations

Although a powerful tool in managing your patients’ volume resuscitation, traditionally SVV has the following limitations:

- **Mechanical ventilation:** Currently, literature supports the use of SVV on patients who are 100% mechanically (control mode) ventilated with tidal volumes of more than 8 mL/kg and fixed respiratory rates.

- **Spontaneous ventilation:** Currently, literature does not support the use of SVV with patients who are spontaneously breathing.

- **Arrhythmias:** Historically arrhythmias have dramatically affected SVV and its ability to be used to guide fluid resuscitation. SVVxtra limits this limitation with the exception of severe arrhythmias such as atrial fibrillation.

- **Other considerations while using SVV to guide fluid resuscitation:**
  - Heart rate (HR) <150 beats per minute
  - Heart rate to respiratory rate ratio below 3:1
  - Respiratory rate (RR) of <35
  - Chest must be closed
  - No right ventricular failure
  - Good arterial waveform required
  - Raised intra-abdominal pressure may exaggerate the cardio-pulmonary interaction
  - Raised intra-thoracic pressure may exaggerate the cardio-pulmonary interaction
### SVVxtra

#### Limiting Limitations with the FloTrac Algorithm

Historically arrhythmias have been considered a contraindication in applying SVV to guide fluid resuscitation. SVVxtra within the FloTrac algorithm allows the clinician to continue to use SVV despite the presence of premature atrial or ventricular contractions. SVVxtra restores the respiratory component of the arterial pressure curve so that SVV continues to reflect the physiological effects of mechanical ventilation on the heart.

The SVVxtra algorithm is based on five consecutive steps:

1. **Detection**
2. **Rejection**
3. **Interpolation**
4. **Restoration**
5. **Calculation**

If the frequency of arrhythmias has exceeded the algorithm’s ability to filter these arrhythmias, then a “Yellow Heart” icon will appear.
Interventional Effects on SVV

• PEEP
Increasing levels of positive end expiratory pressure (PEEP) may cause an increase in SVV, the effects of which may be corrected by additional volume resuscitation if warranted.

• Vascular Tone
The effects of vasodilatation therapy may increase SVV and should be considered before treatment with additional volume.

Summary

When used within its limitations SVV is a sensitive tool that can be used to guide the appropriate management of the patient’s preload to achieve optimal DO₂ to assist with fluid optimization. SVV is an available parameter with the FloTrac sensor and Vigileo monitor.

NOTE: Limitations associated with SVV are not limitations of the FloTrac system in calculating cardiac output. The FloTrac sensor can be used to monitor cardiac output, stroke volume and systemic vascular resistance in the spontaneously breathing patient or the mechanical ventilated patient.
FloTrac/Vigileo System SVV Algorithm

Does my patient need an increase in SV or CO?
(clinical examination, SV, CO, or ScvO₂ measurements, lactate level, renal failure...)

Yes

Is the arterial pressure tracing accurate?
(fast flush test)

Yes

Does my patient make significant respiratory efforts?
(clinical examination, airway pressure curve)

No

Is the tidal volume ≥8 mL/kg

No

Passive Leg Raising or Fluid Challenge Maneuver

Yes

Cardiac Rhythm, is the yellow heart icon on?

No

No fluid
(inotropes, vasodilators...)

<10%

How is SVV?

Yes

>15%

Fluid
(or less aggressive ventilation)

Patients who are preload responsive will usually see a maximal effect within 30-90 seconds and will reach a 10-15% increase in SV. PLR that induced an increase in stroke volume by more than 10% also predicted a volume induced increase in stroke volume by more than 15% with very good sensitivity and specificity.

1. Patient in a semirecumbent position (45° head up) or supine position
2. Note FloTrac system SV – T1 time on % change calculator
3. Simultaneously recline head and/or elevate feet (45° feet up)
4. Wait 1 minute
5. Note FloTrac system SV – T2 time on % change calculator
6. SV % increase > 10-15% = preload responsive
7. SV % increase < 10-15% ≠ preload responsive
8. Repeat as needed
Concerns or Limitations

Concern about the actual effects of performing a PLR on other pathologies such as neurologic injuries should be taken into consideration before a PLR maneuver is performed. Patients whose volume challenges represent a greater risk (ALI, ARDS, ARF), may be managed with a PLR percent increase that clearly exceeds 15%. In cases where a patient’s actual “recruitable” preload is affected by vasoconstriction associated with hypovolemia or cardiogenic shock, traditional indicators of preload (CVP, EDV) can be evaluated, or performing a fluid challenge can be considered.

FloTrac/Vigileo System Fluid Challenge Maneuver

Perform a fluid challenge with a known volume (i.e. 250-500 mL) and note percent change:

1. Note FloTrac system SV – T1 time on % change calculator
2. Infuse bolus of 250-500 mL
3. Note FloTrac system SV – T2 time on % change calculator
4. If SV % increase > 10-15% = preload responsive
5. Consider additional fluids
6. Repeat FloTrac/Vigileo system fluid challenge maneuver
7. If SV % < 10-15% ≠ preload responsive = stop fluids
Venous Oximetry Physiology and Clinical Applications

Physiology and Venous Oximetry

Maintaining the balance between oxygen delivery (DO₂) and consumption (VO₂) to the tissues is essential for cellular homeostasis and preventing tissue hypoxia and subsequent organ failure. Traditional monitoring parameters (HR, blood pressure, CVP, and SpO₂) have been proven to be poor indicators of oxygen delivery and secondary to compensatory mechanisms. Moreover, patients have demonstrated continued signs of tissue hypoxia (increased lactate, low ScvO₂) even after they have been resuscitated to normalized vital signs.

**ScvO₂ = EARLY WARNING AND PREVENTION**

Continuous fiberoptic venous oximetry is a valuable tool for monitoring the balance between oxygen delivery and consumption at the bedside. Continuous venous oximetry is a sensitive real-time indicator of this balance, which can be applied as a global or regional indicator – with mixed venous oxygen saturation (SvO₂) and central venous oxygen saturation (ScvO₂) being the most commonly monitored. SvO₂ is a true reflection of the global balance between oxygen delivery and consumption since it is measured in the pulmonary artery, where venous blood returning to the right heart from the superior venae cavae contributes.
vena cava (SVC), inferior vena cava (IVC) and the coronary sinus (CS) have mixed. SvO₂ has been extensively studied and used clinically to monitor the global balance between DO₂ and VO₂. SvO₂ monitoring has been available through either co-oximetry laboratory analysis or through continuous fiberoptic monitoring with advanced technology pulmonary artery catheters since the 1970s and mid-1980s, respectively.

Continuous fiberoptic ScvO₂ monitoring became available in 2003 on an 8.5 Fr central venous catheter platform (Edwards PreSep catheter). With the tip of the PreSep central venous catheter placed in the SVC, ScvO₂ can be measured and displayed on either a Vigileo or Edwards Vigilance II monitor. This capability is also available via 4.5 Fr and 5.5 Fr central venous oximetry catheters (Edwards PediaSat catheter) for pediatric use.

* PreSep Oligon oximetry catheters contain an integrated Oligon antimicrobial material. The activity of the antimicrobial material is localized at the catheter surfaces and is not intended for treatment of systemic infections. In vitro testing demonstrated that the Oligon material provided broad-spectrum effectiveness (≥ 3 log reduction from initial concentration within 48 hours) against the organisms tested: Staphylococcus aureus, Staphylococcus epidermidis, Klebsiella pneumoniae, Enterococcus faecalis, Candida albicans, Escherichia coli, Serratia marcescens, Acinetobacter calcoaceticus, Corynebacterium diphtheriae, Enterobacter aerogenes, GMRSa, Pseudomonas aeruginosa, Candida glabrata and VRE (Enterococcus faecium).
**Difference Between SvO\textsubscript{2} and ScvO\textsubscript{2}**

Since SvO\textsubscript{2} and ScvO\textsubscript{2} are affected by the same four factors (cardiac output, hemoglobin, oxygenation, and oxygen consumption), and trend together clinically, they are considered clinically interchangeable. The exception is when calculating global physiologic profiles that use SvO\textsubscript{2}, such as VO\textsubscript{2}.

SvO\textsubscript{2} is a global indicator of the balance between DO\textsubscript{2} and VO\textsubscript{2} as it is a reflection of all venous blood; IVC, SVC, and CS. ScvO\textsubscript{2} is a regional reflection (head and upper body) of that balance. Under normal conditions ScvO\textsubscript{2} is slightly lower than SvO\textsubscript{2} due in part to the mixing and amount of venous blood returning. In hemodynamically unstable patients, this relationship changes with ScvO\textsubscript{2} being higher than SvO\textsubscript{2} by approximately 7%. This difference can widen in shock states, up to 18%, but the values trend together more than 90% of the time.

**Global Venous Oximetry**
- SvO\textsubscript{2} – mixed venous oximetry

**Regional Venous Oximetry**
- ScvO\textsubscript{2} – head and upper extremities
- SpvO\textsubscript{2} – peripheral venous oximetry

**Organ Specific Venous Oximetry**
- SjvO\textsubscript{2} – cranial jugular bulb oximetry
- ShvO\textsubscript{2} – hepatic venous oximetry
- ScsO\textsubscript{2} – coronary sinus oximetry

**Continuous ScvO\textsubscript{2} Monitoring Technology**

All venous oximetry is measured through reflection spectrophotometry. Light is emitted from an LED through one of the two fiberoptic channels into the venous blood; some of this light is reflected back and received by another fiberoptic
channel, which is read by a photodetector. The amount of light that is absorbed by the venous blood (or reflected back) is determined by the amount of oxygen that is saturated or bound to hemoglobin. This information is processed by the oximetry monitor, and updated and displayed every two seconds as a percent value on the monitor.
Accuracy of Edwards Fiberoptic Continuous ScvO₂ Compared to Co-oximetry

In a laboratory bench environment continuous fiberoptic venous oximetry monitoring accuracy is approximately ± 2% at oximetry range of 30-99% as compared to a co-oximeter. With oxygen saturations from 9% to 100%, the results of the fiberoptic oximetry systems correlated significantly (P < 0.0001) with the standard blood gas co-oximetry system (r = 0.99). Clinical comparison measurements also showed a significant correlation (Pr = 0.94, P < 0.001) and close linear relationship as determined by regression analysis (r² = 0.88, P < 0.001). Difference of means (bias) was - 0.03% with a ± 4.41% precision per Liakopoulos et al.

Interference with ScvO₂ Readings

Technical issues and therapeutic interventions may affect fiberoptics. Both the large distal lumen and the sending/receiving optics reside at the tip of the catheter. Therefore, tip position may influence signal quality (SQI) and readings if the tip is positioned against a vessel wall. Fluids infused through the distal lumen may also influence SQI and readings (e.g., lipids such as TPN or propofol, green or blue dyes, and crystalloid infusions at high flow rates). Catheter kinking may also result in a high SQI.
Interpreting Venous Oximetry (SvO₂ and ScvO₂) Values

Normal range values for SvO₂ are 60-80% and 70% for ScvO₂. ScvO₂ usually runs 7% higher than SvO₂ in critically ill patients. Low oximetry readings usually indicate either low oxygen delivery (DO₂) or an increase in consumption (VO₂). Significantly elevated levels (> 80%) may indicate:

- Low metabolic demand
- Inability to use oxygen delivered to the tissues (sepsis)
- Significantly high cardiac output
- Shunting of oxygenated blood past tissue
- Technical errors

When Change is Significant

ScvO₂ and SvO₂ values are not static and fluctuate approximately ± 5%. These values may show significant changes with activities or interventions such as suctioning; however, the values should recover within seconds. Slow recovery is an ominous sign of the cardiopulmonary system’s struggle to respond to a sudden increase in oxygen demand. When monitoring ScvO₂, clinicians should look for changes of ± 5 -10% that are sustained for more than 5 minutes and then investigate each of the four factors that influence ScvO₂:

- Cardiac output
- Hemoglobin
- Arterial oxygen saturation (SaO₂) and
- Oxygen consumption

The first three (above) are indicators of DO₂, while the fourth is an indicator of VO₂.
Clinical Applications of ScvO₂

ScvO₂ and SvO₂ are affected by the same four factors and trend together more than 90% of the time. Thus most of the research and clinical applications documented for SvO₂ should apply to ScvO₂.

The figure below provides examples of clinical situations where ScvO₂ monitoring may be helpful in identifying imbalances between DO₂ and VO₂.
Continuous venous oximetry (ScvO₂) monitoring is an early, sensitive, and real-time indicator of the balance between DO₂ and VO₂ that can alert clinicians to an imbalance when traditional vital signs may not. ScvO₂ monitoring with the PreSep or PediaSat catheter is a practical tool which is no more invasive than a traditional central venous catheter. Venous oximetry is best used in conjunction with cardiac output monitoring. Moreover, keeping ScvO₂ values above 70% has been proven to lead to better patient outcomes.

**Summary**

Continuous venous oximetry (ScvO₂) monitoring is an early, sensitive, and real-time indicator of the balance between DO₂ and VO₂ that can alert clinicians to an imbalance when traditional vital signs may not. ScvO₂ monitoring with the PreSep or PediaSat catheter is a practical tool which is no more invasive than a traditional central venous catheter. Venous oximetry is best used in conjunction with cardiac output monitoring. Moreover, keeping ScvO₂ values above 70% has been proven to lead to better patient outcomes.

* SVV is an indicator of preload responsiveness.
VolumeView System

The VolumeView system expands the application of thermodilution technology through transpulmonary thermodilution. It uses these familiar concepts to measure and derive key elements of oxygen delivery such as cardiac output and volumetric variables to assess components of cardiac output such as preload and contractility. In addition, lung water measurements are available that can assist the clinician in treating patients with lung injury and cardiac failure.

Transpulmonary thermodilution cardiac output uses the same principles as right heart thermodilution except the thermal bolus is injected into the central venous system and moves across the right heart, lungs, left heart and out into the arterial tree where the thermal change is measured over time by an embedded thermistor on a catheter inserted into the femoral artery.

Transpulmonary thermodilution with the VolumeView system allows for the measurement and derived calculations of the elements that affect oxygen delivery through:

- Intermittent transpulmonary thermodilution cardiac output
- Calibrated continuous cardiac output
- Intermittent or continuous assessment of systemic vascular resistance
- Global End Diastolic Volume
- Global Ejection Fraction
- Cardiac Function Index

In addition:
- Extra-Vascular Lung Water
- Pulmonary Vascular Permeability Index
VolumeView System Setup

System Setup

1. EV1000 monitor
2. VolumeView sensor
3. VolumeView femoral arterial catheter
4. VolumeView thermistor manifold
5. TruWave pressure transducer
Intermittent Cardiac Output Calculation with the VolumeView System

Transpulmonary thermodilution uses the same modified Stewart-Hamilton equation to measure cardiac output that right heart thermodilution uses where the patient’s blood temperature, as well as the injectate temperature, is continuously monitored by a computer with each bolus. A computation constant is derived by the computer from an injectate solution of a known temperature, volume, and specific weight. The clinician enters the injectate volume into the computer.

After injection, the computer analyzes the area under the transpulmonary thermodilution curve to calculate cardiac output. The area under the curve is inversely proportional to the cardiac output. A series of boluses are performed and edited to obtain an average value. Once edited the measured and derived calculations are displayed and time stamped for retrospective review.
• Blood Temperature is monitored and collected through an embedded thermistor on the VolumeView femoral arterial catheter.

• The injectate temperature is collected and monitored through an in-line thermistor on the VolumeView thermistor manifold.

• The volume of the injectate is entered into the computer by the clinician.

• The area under the curve is calculated and analyzed by the computer by measuring the change in temperature over time in the femoral artery.

Once the values are accepted the continuous monitoring of cardiac output, SVV and other derived values are initiated by the VolumeView sensor and displayed on the far right hand side of the monitoring screen. The averaged TPTD values are displayed; intermittent cardiac output (iCO), intermittent Stroke Volume (iSV), Global End Diastolic Volume Index (GEDI), Extra Vascular Lung Water Index (EVLWI), Global Ejection Fraction (GEF), Intra Thoracic Blood Volume (ITBV), Pulmonary Vascular Permeability Index (PVPI), intermittent Systemic Vascular Resistance (iSVR) along with the globes which indicate where the values are within the target ranges.
Continuous Cardiac Output with VolumeView

VolumeView technology uses a calibrated arterial pressure based cardiac output (APCO) for its continuous cardiac output calculation. This pulse contour analysis is calibrated against the measured TPTD cardiac output and uses similar wave shaped variables to maintain the accuracy between calibrations as the FloTrac algorithm. The VolumeView algorithm adjusts the calculated continuous cardiac output display by a percent change based on its proprietary algorithm against the measured cardiac output.

Calculating Global End Diastolic Volume

The transpulmonary thermodilution measurement used to calculate cardiac output can also be used to calculate other physiologic parameters such as Global End Diastolic Volume, Global Ejection Fraction, and Extra Vascular Lung Water. These parameters are useful in evaluating and guiding volume resuscitation, ventricular performance, and changes in lung water that develops from disease or interventions.

Global End Diastolic Volume is closely related to the volume within all four chambers at the end of diastole. It can be used to assess preload and manage a patient’s volume resuscitation.
In order to calculate GEDV, Intra Thoracic Thermal Volume (ITTV) is calculated by identifying the beginning of the injection cycle from a pressure spike measured in the central venous pressure from the VolumeView CVC manifold. The VolumeView system’s TPTD algorithm then identifies the peak indicator concentration followed by its immediate downslope which is an indication of the mean transit time. Once cardiac output is known and the mean transit time is known, Intra Thoracic Thermal Volume can be calculated by multiplying cardiac output times the mean transit time. The downslope time is representative of the flow time through the lungs.

Intra Thoracic Thermal Volume is the first calculation of the cardiopulmonary volumes calculated from the TPTD procedure. It represents the total dilution volume within the thorax, which consists of the heart, lungs, and vasculature, that is calculated by the VolumeView TPTD algorithm.
The GEDV is a reflection of the volume within all four chambers at the end of diastole. The rate of change of the upslope and downslope of the thermodilution waveform is used to calculate the slope function which appropriately scales down ITTV to account for Pulmonary Thermal Volume in order to calculate GEDV. GEDV is computed by calculating ITTV and multiplying it against a scale that accounts for PTV.

GEDI is indexed against body surface area to give the Global End Diastolic Volume Index, or GEDI.
Global Ejection Fraction

Global Ejection Fraction, or GEF, can be used to assess global cardiac function. Stroke Volume is multiplied by 4 to account for the four chambers of the heart then divided by GEDV

Cardiac Output / Pulse Rate = Stroke Volume

Stroke Volume * 4 / GEDV = Global Ejection Fraction

Global Ejection Fraction normal range is between 25-35%

Extra Vascular Lung Water

VolumeView system can calculate the amount of Extra Vascular Lung Water or EVLW, which is an assessment of pulmonary edema.

Extra Vascular Lung Water is calculated by subtracting the Pulmonary Blood Volume from the Pulmonary Thermal Volume, leaving the thermal volume within the lungs. EVLW can be used to assess the level of pulmonary edema which may be the result of heart failure, volume overload, or lung injury and can interfere with the ability of the lungs to oxygenate the blood. This is indexed against the patient’s predicted body weight to obtain Extra Vascular Lung Water Index or EVLWI. EVLW can be used to assess the level of pulmonary edema.
The “normal” value for EVLWI is reported to be 3–7 mL/kg. Values above 10 mL/kg indicate pulmonary edema and values as high as 15-20 ml/kg indicate severe pulmonary edema. EVLW is a useful indicator of pulmonary edema and challenges with oxygenation.
Pulmonary Vascular Permeability Index

Pulmonary vascular permeability index, or PVPI, is also another tool that the clinician may use in assessing lung function. PVPI is calculated by dividing extra vascular lung water by pulmonary blood volume.

\[
\text{EVLW} / \text{PBV} = \text{PVPI}
\]

PVPI helps the clinician to differentiate which mechanisms are responsible for increased EVLW: PVPI is increased (> 3) in patients with increased pulmonary permeability due to lung injury and normal in patients with hydrostatic and cardiogenic pulmonary edema.
Swan-Ganz Catheters
Advanced and Standard Technology

ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972
The Swan-Ganz Pulmonary Artery Catheter

Standard Swan-Ganz Catheter

The standard thermodilution Swan-Ganz pulmonary artery catheter was introduced in 1972 by Dr. Jeremy Swan and Dr. William Ganz. This catheter gives clinicians the ability to measure right heart pressures, pulmonary artery occlusion pressure (“wedge”), sample mixed venous blood from the pulmonary artery, as well as measure cardiac output through thermodilution when used with a bedside physiologic monitor and pressure transducers. Although this catheter has undergone multiple advances over the years, the standard Swan-Ganz catheter is still available and in use around the world today.

The standard Swan-Ganz catheter measures:

- Right heart pressures:
  - Right atrial pressure (RAP)
  - Pulmonary artery pressures
    - Pulmonary artery systolic (PAS)
    - Pulmonary artery diastolic (PAD)
    - Pulmonary artery mean (PAM)
    - Pulmonary artery occlusion pressure (PAOP)

- Thermodilution cardiac output:
  - Edwards CO-Set iced, closed bolus injectate system
  - CO-Set room temperature, closed bolus injectate system

- Pulmonary artery blood sampling for laboratory analysis:
  - Mixed venous blood oxygen saturation (SvO₂)
  - Serial measurements of right heart chamber oxygen saturations
• Additional available features:
  ▪ Venous infusion port (VIP)
  ▪ Paceport catheter – temporary right atrial and/or ventricular trans-venous pacing
  ▪ Angiographic catheters – designed for high pressure dye injections used in radiographic examinations

Applications of standard Swan-Ganz catheters

• Right heart catheterization for right heart pressure measurements (PAS, PAD, PAOP) for diagnostic purposes
• Single point-in-time calculations of cardiac output using bolus thermodilution for diagnosing cardiac function
• Single mixed venous laboratory blood draws via the catheter to assess $SvO_2$ and the balance between oxygen delivery and consumption
• Serial right heart chamber venous blood draws to measure oxygen saturations indicating left to right intra-cardiac shunts
• Pulmonary artery angiography
• Temporary transvenous V or AV pacing
Advanced Technology Swan-Ganz Catheter

In addition to providing most of the same functionality as the standard Swan-Ganz catheter, the advanced technology Swan-Ganz catheter provides the ability to continuously monitor the patient’s balance between oxygen delivery and consumption as well as the ability to help investigate the root cause of an imbalance through analysis of the components of stroke volume (preload, afterload, and contractility). Through early identification of imbalances and root cause analysis, patients can be treated most appropriately and interventions assessed, thus potentially avoiding tissue hypoxia, organ dysfunction and crisis interventions.

The advanced technology Swan-Ganz catheter measures:

- Right heart pressures:
  - Right atrial pressure (RAP)
  - Pulmonary artery pressures
    - Pulmonary artery systolic (PAS)
    - Pulmonary artery diastolic (PAD)
    - Pulmonary artery mean (PAM)
    - Pulmonary artery occlusion pressure (PAOP)

- Thermodilution cardiac output:
  - CO-Set iced, closed bolus injectate system
  - CO-Set room temperature, closed bolus injectate system

- Pulmonary artery blood sampling for laboratory analysis:
  - Mixed venous blood oxygen saturation (SvO₂)

- SvO₂ – mixed venous oxygen saturation is continuously measured through fiberoptic reflectance technology and is a global indicator of the balance between oxygen delivery and consumption
- CCO – continuous cardiac output, measured through advanced thermodilution technology, is a key component of oxygen delivery

- RVEF – right ventricular ejection fraction is also continuously measured through advanced thermodilution technology and algorithm analysis indicates right ventricular function and filling which can be used to help assess right heart contractility

- RVEDV – right ventricular end diastolic volume is continuously calculated by dividing stroke volume (mL/beat) by RVEF (%) giving a key indicator of preload

- SVR and SVRI – continuous systemic vascular resistance can be calculated when the Vigilance II monitor obtains continuous MAP and CVP from the bedside physiologic monitor

Applications of advanced technology Swan-Ganz catheters

- Continuous assessment of right heart pressures (RAP, PAD, PAS, and PAOP)

- Continuous assessment of oxygen delivery and consumption (SvO₂)

- Continuous assessment of cardiac output (CCO) a primary component of DO₂

- Continuous assessment of preload through RVEDV, PAD, PAOP

- Continuous assessment of afterload through SVR, SVRI

- Continuous assessment of contractility through RVEF, SVI, and calculation of RVSWI

- Intermittent calculation of oxygen delivery (DO₂) and consumption (VO₂)
Advantages of the advanced technology Swan-Ganz catheter as compared to the standard Swan-Ganz catheter

- Maximum amount of diagnostic information with same invasive procedure
- Continuous assessment of $\text{DO}_2/\text{VO}_2$ balance with $\text{SvO}_2$ monitoring
- Continuous assessment of adequacy of CO by assessing $\text{DO}_2/\text{VO}_2$ balance with $\text{SvO}_2$ monitoring
- Continuous assessment of components of stroke volume (preload, afterload, and contractility) ($\text{RVEDV}$, $\text{SVR}$, $\text{RVEF}$ and $\text{SVI}$)
- Mitigation of user error in association with wedge procedure/calculation through automated alternative preload parameter ($\text{RVEDV}$)
- Mitigation of pulmonary artery rupture possibility associated with wedge procedure by providing automated preload parameter ($\text{RVEDV}$)
- Mitigation of inappropriate therapy due to miscalculation of PAOP by using automated preload parameter ($\text{RVEDV}$)
- Mitigation of inappropriate preload assessment secondary to changes in ventricular compliance affecting PAD or PAOP
- Mitigation of iatrogenic infection risk from bolus injections
- Mitigation of cardiac output error with CCO automation through elimination of bolus cardiac output user error
- Increased accuracy of cardiac output calculations, elimination of ventilator cycle and thermal noise effect
Vigilance II Monitors

Vigilance II monitor is used with the advanced technology Swan-Ganz catheters to graphically and numerically display key flow parameters as well as the components of stroke volume. The Vigilance II monitor houses two distinct technologies: (1) continuous fiberoptic venous oximetry ($SvO_2$), and (2) continuous thermodilution cardiac output. CCO and RVEF are measured values while RVEDV, SVR, SVRI, and stroke volume are calculated when the Vigilance II monitor obtains heart rate (HR), mean arterial pressure (MAP), and central venous pressure (CVP) from the bedside physiologic monitor.

Applications and Contraindications

Clinical applications for Swan-Ganz pulmonary artery catheters:

- Intra-abdominal hypertension
- Patients at risk for acute right ventricular dysfunction
- ARDS
- Extensive burns
- Cardiac surgery
- Significant cardiac tamponade
- Significant cardiomyopathy
- Significant constrictive pericarditis
- Drug intoxication
- Severe eclampsia
- Significant intra- or extra-vascular fluid shifts
- At risk for hemorrhage
- Intra- and post-operative high-risk surgery management
- Patient on intra-aortic balloon counterpulsation
- Complex liver resections
- Liver transplantation
- Complex lung resection
- Complex myocardial infarctions
- Pulmonary edema
- Pulmonary embolism
- Pulmonary hypertension
- Acute renal failure
- Severe sepsis
- Presence of or at risk for cardiogenic shock
- Presence of or at risk for distributive shock
- Presence of or at risk for hemorrhagic shock
- Presence of or at risk for obstructive shock
- Shock of unknown etiology
- Shock unresponsive to attempts at resuscitation
- Severe trauma
- Ventilator affects on hemodynamics

Relative contraindications for Swan-Ganz pulmonary artery catheterization:

(There are no absolute contraindications to the use of a pulmonary artery catheter; risk-benefit must be assessed for each patient)

- Left bundle branch block
- Patients with tricuspid or pulmonic heart valve replacements
- Presence of endocardial pacing leads
- Lack of appropriate clinical skills or infrastructure to insert and/or support the use of a pulmonary artery catheter
- Heparin coated catheters in patients with known sensitivity to heparin
# Selected Swan-Ganz Catheter Specifications

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<thead>
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<th>Model Numbers</th>
<th>131</th>
<th>132</th>
<th>177</th>
<th>831/834</th>
<th>931/991</th>
<th>139</th>
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<td>33.2/2.8:1</td>
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<td>47/3.1:1</td>
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</table>
Advanced Swan-Ganz Catheters

Swan-Ganz CCOMbo – Volumetric
(SvO₂, CCO, RVEF, RVEDV)
Models 774, 777

These advanced technology Swan-Ganz catheters combine the same basic features of the original Swan-Ganz thermodilution catheter as well as advanced continuous monitoring parameters. The balance between oxygen delivery and consumption can be continuously assessed through fiberoptic measurements of mixed venous oxygen saturation (SvO₂) monitoring, as well as continuous thermodilution cardiac output measurements (CCO), a primary determinate of oxygen delivery (DO₂). It also allows the further evaluation of the components of stroke volume (SV) through continuous monitoring of right ventricular end diastolic volume (RVEDV) and continuous monitoring of right ventricular ejection fraction (RVEF). Advanced technology Swan-Ganz catheters must be used in conjunction with a Vigilance II series monitor. Systemic vascular resistance (SVR) can be continuously measured and displayed when the Vigilance II monitor is interfaced with the bedside monitor to obtain mean arterial pressure (MAP) and central venous pressure (CVP). The heart rate from the bedside monitor must be continuously sent to the Vigilance II monitors for volumetric measurements of RVEDV and RVEF.
Swan-Ganz CCOmbo and CCOmbo/VIP (SvO₂ and CCO) Models 744 and 746

These advanced technology Swan-Ganz catheters combine the same basic features of the original Swan-Ganz thermodilution catheter as well as advanced continuous monitoring parameters. The balance between oxygen delivery and consumption can be continuously assessed through fiberoptic measurements of mixed venous oxygen saturation (SvO₂) monitoring, as well as continuous thermodilution cardiac output measurements (CCO), a primary determinate of oxygen delivery (DO₂). Advanced technology Swan-Ganz catheters must be used in conjunction with a Vigilance II series monitor. Systemic vascular resistance (SVR) can be continuously measured and displayed when the Vigilance II monitor is interfaced with the bedside monitor to obtain mean arterial pressure (MAP) and central venous pressure (CVP). A venous infusion port (VIP) is also available for intravenous medication delivery.
Swan-Ganz Mixed Venous Oximetry ($SvO_2$) Models 741 and 780

These advanced technology Swan-Ganz catheters combine the same basic features of the original Swan-Ganz thermodilution catheter as well as advanced continuous monitoring parameters. The balance between oxygen delivery and consumption can be continuously assessed through fiberoptic measurements of mixed venous oxygen saturation ($SvO_2$). Advanced technology Swan-Ganz catheters must be used in conjunction with an Edwards oximetry monitor. The Paceport Oximetry TD catheter (780) is intended for use in patients who require hemodynamic monitoring when temporary transvenous pacing is anticipated.
Swan-Ganz Continuous Cardiac Output (CCO) Model 139

This advanced technology Swan-Ganz catheter combines the same basic features of the original Swan-Ganz thermodilution catheter as well as continuous thermodilution cardiac output measurements (CCO), a primary determinate of oxygen delivery ($\text{DO}_2$). Advanced technology Swan-Ganz catheters must be used in conjunction with a Vigilance II series monitor. Systemic vascular resistance (SVR) can be continuously measured and displayed when the Vigilance II monitor is interfaced with the bedside monitor to obtain mean arterial pressure (MAP) and central venous pressure (CVP).
Standard Swan-Ganz Catheters

Model 131

This standard Swan-Ganz thermodilution catheter provides assessment of a patient’s hemodynamic condition through direct intracardiac and pulmonary artery pressure monitoring. Intermittent determination of cardiac output by bolus thermodilution, a primary determinant of oxygen delivery, can be measured with this catheter. Sampling of mixed venous blood from the distal lumen in the pulmonary artery provides an assessment of oxygen utilization.
Swan-Ganz Thermodilution Catheter with Venous Infusion Port
Models 831 and 834

These standard Swan-Ganz thermodilution catheters provide assessment of a patient’s hemodynamic condition through direct intracardiac and pulmonary artery pressure monitoring. Intermittent determination of cardiac output by bolus thermodilution, a primary determinant of oxygen delivery, can be measured with this catheter. Sampling of mixed venous blood from the distal lumen in the pulmonary artery provides an assessment of oxygen utilization. In addition, venous infusion catheters provide additional lumens that exit either in the RA or both RA and RV, depending on the type of catheter. Clinical indications include those when central circulation access is needed for multiple volume and solution infusions. Intra-atrial or intra-ventricular pressure monitoring can also be obtained with these additional lumens.

**MODEL 831**

**MODEL 834**

Additional RA lumen and RV lumen exits at 19 cm from tip to assure precise RV pressure monitoring.
Swan-Ganz Paceport TD Catheters Models 931 and 991

In addition to traditional hemodynamic monitoring, the Paceport catheters provide either ventricular, atrial, or atrioventricular pacing on demand. Clinical conditions include those in which managing the patient’s ventricular heart rate is needed or optimizing cardiac output with synchronized AV pacing. Patients with known LBBB may be at risk for developing a complete heart block during PAC insertion. The Paceport catheter provides for rapid ventricular pacing if this occurs and the patient requires hemodynamic monitoring.

Temporary atrial, ventricular, or atrioventricular pacing can be instituted with the use of the Chandler Transluminal V-Pacing probe and atrial J pacing probe.

The additional lumens (RV lumen exits at 19 cm from the tip, RA exits at 27 cm) can also be used for pressure monitoring of their respective chambers or for additional fluid infusions.
Swan-Ganz Pacing Probe Catheters
Models 100 and 500

The 98-100H Chandler Transluminal V-Pacing probe can be used for standby ventricular pacing when the patient’s condition warrants. When the probe is not inserted, the lumen that exits at 19 cm from the distal catheter tip may be used for RV pressure monitoring or infusion of fluids or solutions.

These probes can also be used for intra-atrial or ventricular ECG monitoring.

The Flex-Tip Transluminal A-Pacing probe (model 98-500H) can be inserted into the A-Probe lumen of the A-V Paceport catheter for atrial pacing. The lumen exits at 27 cm from the distal tip.

For atrio-ventricular pacing, the 991H is used with both the 98-100H Chandler V-Pacing probe and the 98-500H. Clinical indications include patients who would benefit from AV sequential pacing for optimization of cardiac output.

To be used with the appropriate Swan-Ganz Paceport TD catheter.
Swan-Ganz Pacing Thermodilution Catheters Models 200 and 205

Atrial and ventricular pacing electrodes are placed on the catheter to provide on-demand atrial, ventricular, or AV sequential pacing. The 205 catheter is designed for patients with smaller anatomy to enhance capture for pacing. This catheter satisfies pacing indications previously stated with Paceport.

Temporary atrial, ventricular, or atrioventricular pacing can be instituted rapidly.
Physiological Basis for Pulmonary Artery Pressure Monitoring

Ventricles in Systole

In this figure the balloon is deflated and the ventricles are in systole. The tricuspid and mitral valves are closed, while the pulmonic and aortic valves are open. A higher pressure is generated by the right ventricle during contraction and is transmitted to the catheter tip located in the pulmonary artery. The catheter records pulmonary artery systolic pressure (PASP), which reflects right ventricular systolic pressure (RVSP) because there is now a common chamber with a common volume and pressure.

RVSP = PASP
Ventricles in Diastole

During diastole the tricuspid and mitral valves are open. The ventricles are filling with blood from their respective atria. At this time the tricuspid valve (TV) and mitral valve (MV) are open and the pulmonic valve (PV) and aortic valve (AoV) are closed.

With the balloon still deflated, pulmonary artery diastolic pressure (PADP) is recorded. After the closure of the pulmonic valve, the right ventricle continues to relax. This causes a lower diastolic pressure in the right ventricle than in the pulmonary artery. RVEDP is less than PADP.

Since there is normally no obstruction between the pulmonary artery and left atrium, the pressure recorded will be virtually the same as left atrial pressure. Left atrial pressure is also reflected as left ventricular end-diastolic pressure (LVEDP) when the mitral valve is open.

When transducing the proximal port, the right atrial pressure reflects right ventricular end-diastolic pressure when the tricuspid valve is open.

VENTRICULAR DIASTOLE

\[
\text{RAP} = \text{RVEDP} \\
\text{RVEDP} < \text{PADP} \\
\text{PADP} \approx \text{LAP} \approx \text{LVEDP}
\]
Ventricles in Diastole: Catheter Wedged

By inflating the balloon, the catheter floats downstream into a smaller branch of the pulmonary artery. Once the balloon lodges, the catheter is considered “wedged”. It is in this wedge position that right sided and PA diastolic pressures are effectively occluded.

Because there are no valves between the pulmonic and mitral valve, there is now an unrestricted vascular channel between the catheter tip in the pulmonary artery through the pulmonary vascular bed, the pulmonary vein, the left atrium, the open mitral valve and into the left ventricle. The distal lumen is now more closely monitoring left ventricular filling pressure or left ventricular end-diastolic pressure.

The importance of this pressure is that normally it closely approximates the pressure present in the left ventricle during end-diastole and provides an indirect means of assessing left ventricular preload.
Normal Insertion Pressures and Waveform Tracings

Right Atrial/Central Venous Pressure (RA/CVP)
2 to 6 mmHg
Mean 4 mmHg
a = atrial systole
c = backward bulging from tricuspid valve closure
v = atrial filling, ventricular systole

Right Ventricular
Systolic Pressure (RVSP)
15–25 mmHg
Diastolic Pressure (RVDP)
0–8 mmHg
Pulmonary Artery

Systolic Pressure (PASP)
15–25 mmHg

Diastolic Pressure (PADP)
8–15 mmHg

Mean Pressure (MPA)
10–20 mmHg

Pulmonary Artery Occlusion Pressure (PAOP)

Mean 6–12 mmHg

\( a = \text{atrial systole} \)

\( v = \text{atrial filling, ventricle systole} \)
### Abnormal Waveform Chart

#### RIGHT ATRIAL WAVEFORMS

<table>
<thead>
<tr>
<th>Waveform Description</th>
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<td>Hypovolemia</td>
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<td>Transducer zero level too high</td>
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<tr>
<td>Elevated mean pressure</td>
<td>Fluid overload states</td>
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<td>Right ventricular failure</td>
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<td>Left ventricular failure causing right ventricular failure</td>
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<td>Tricuspid stenosis or regurgitation</td>
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<td>Pulmonic stenosis or regurgitation</td>
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<td>Pulmonary hypertension</td>
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<td>Elevated “a” wave: atrial systole, increased resistance to ventricular filling</td>
<td>Tricuspid stenosis</td>
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<td>Decreased right ventricular compliance</td>
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<td>Pulmonary hypertension</td>
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<td>Absent “a” wave</td>
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<td>Functional regurgitation from right ventricular failure</td>
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<tr>
<td></td>
<td>Constrictive pericardial disease</td>
</tr>
<tr>
<td></td>
<td>Hypervolemia</td>
</tr>
</tbody>
</table>

#### RIGHT VENTRICULAR WAVEFORMS

<table>
<thead>
<tr>
<th>Waveform Description</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated systolic pressure</td>
<td>Pulmonary hypertension</td>
</tr>
<tr>
<td></td>
<td>Pulmonic valve stenosis</td>
</tr>
<tr>
<td></td>
<td>Factors that increase pulmonary vascular resistance</td>
</tr>
<tr>
<td>Decreased systolic pressure</td>
<td>Hypovolemia</td>
</tr>
<tr>
<td></td>
<td>Cardiogenic shock (RV failure)</td>
</tr>
<tr>
<td></td>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Increased diastolic pressure</td>
<td>Hypervolemia</td>
</tr>
<tr>
<td></td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td></td>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td></td>
<td>Pericardial constriction</td>
</tr>
<tr>
<td>Decreased diastolic pressure</td>
<td>Hypervolemia</td>
</tr>
</tbody>
</table>
### PULMONARY ARTERY WAVEFORMS

| Elevated systolic pressure | Pulmonary disease  
Increased blood flow, left to right shunt  
Increased pulmonary vascular resistance |
|---------------------------|------------------------------------------------|
| Elevated diastolic pressure | Left heart failure  
Intravascular volume overload  
Mitral stenosis or regurgitation |
| Reduced systolic and diastolic pressure | Hypovolemia  
Pulmonic stenosis  
Tricuspid stenosis |

### PULMONARY ARTERY WEDGE/LEFT ATRIAL WAVEFORM

| Decreased (mean) pressure | Hypovolemia  
Transducer level too high |
|---------------------------|------------------------------------------------|
| Elevated (mean) pressure  | Fluid overload states  
Left ventricular failure  
Mitral stenosis or regurgitation  
Aortic stenosis or regurgitation  
Myocardial infarction |
| Elevated “a” wave (any increased resistance to ventricular filling) | Mitral stenosis |
| Absent “a” wave | Atrial fibrillation  
Atrial flutter  
Junctional rhythms |
| Elevated “v” wave | Mitral regurgitation  
Functional regurgitation from left ventricular failure  
Ventricular septal defect |
| Elevated “a” and “v” waves | Cardiac tamponade  
Constrictive pericardial disease  
Left ventricular failure |
**Swan-Ganz Catheter Port Locations and Functions**

<table>
<thead>
<tr>
<th>Location</th>
<th>Color</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal</td>
<td>Yellow</td>
<td>Monitors PA pressures</td>
</tr>
<tr>
<td>Proximal</td>
<td>Blue</td>
<td>Monitors RA pressures, used for cardiac output injectate fluid</td>
</tr>
<tr>
<td>Balloon Gate Valve</td>
<td>Red</td>
<td>Syringe used to inflate balloon for placement and obtaining wedge values</td>
</tr>
<tr>
<td>Thermistor Connector</td>
<td>Yellow</td>
<td>Measures blood temperature 4 cm from distal tip</td>
</tr>
</tbody>
</table>

**ADDITIONAL SWAN-GANZ CATHETERS**

<table>
<thead>
<tr>
<th>Location</th>
<th>Color</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venous Infusion Port (VIP)</td>
<td>White</td>
<td>Additional RA lumen for fluid infusion</td>
</tr>
<tr>
<td>RV Infusion Port (VIP+)</td>
<td>Violet</td>
<td>Additional RV lumen for fluid infusion</td>
</tr>
<tr>
<td>RV Pacing Lumen (Paceport)</td>
<td>Orange</td>
<td>Additional lumen for RV pacing or fluid infusion</td>
</tr>
<tr>
<td>RA Pacing Lumen (AV Paceport)</td>
<td>Yellow</td>
<td>Additional lumen for RA pacing or infusion of fluids</td>
</tr>
</tbody>
</table>

Port exit locations may vary depending on catheter model. See Swan-Ganz Catheter Reference Section.

*Adult Catheters*
Insertion Techniques for the Swan-Ganz Catheter

1. Before insertion of the Swan-Ganz catheter, prepare the pressure monitoring system for use according to the institution’s policies and procedures.

2. Insert the catheter following recommended guidelines and advance the catheter towards the thorax.

3. Once the catheter tip has exited the introducer sheath (approximately 15 cm) and reached the junction of the superior or inferior vena cava and right atrium, the balloon is inflated with CO\textsubscript{2} or air to the full volume indicated on the catheter shaft and gate valve is locked (7 to 7.5 Fr 1.5 cc). This position can be noted when respiratory oscillations are seen on the monitor screen.

4. Catheter advancement to the PA should be rapid, since prolonged manipulation can result in loss of catheter stiffness. The Swan-Ganz catheter is made of polyvinyl chloride (PVC) material designed to soften in vivo. With prolonged insertion times, a “softer” catheter may cause coiling in the RV or difficulties in catheter advancement.

5. Once the wedge position has been identified, the balloon is deflated by unlocking the gate valve, removing the syringe and allowing the back pressure in the PA to deflate the balloon. After balloon deflation, reattach the syringe to the gate valve. The gate valve is typically only placed in the locked position during catheter insertion.

6. To reduce or remove any redundant length or loop in the right atrium or ventricle, slowly pull the catheter back 2–3 cm. Then reinflate the balloon to determine the minimum inflation volume necessary to obtain a wedge pressure tracing. The catheter tip should be in a position where the full or near-full inflation volume (1.5 cc for 7 to 8 Fr catheters) produces a wedge pressure tracing.
## Swan-Ganz Catheter Insertion Waveforms

![Waveforms of Swan-Ganz Catheters](image)

Tracings noted on insertion. Observe diastolic pressure on insertion as pressures will rise when pulmonary artery reached.

## Catheter Insertion Distance Markings*

<table>
<thead>
<tr>
<th>Location</th>
<th>Distance to VC/RA Junction</th>
<th>Distance to PA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal Jugular</td>
<td>15 to 20</td>
<td>40 to 55</td>
</tr>
<tr>
<td>Subclavian Vein</td>
<td>10 to 15</td>
<td>35 to 50</td>
</tr>
<tr>
<td>Femoral Vein</td>
<td>30</td>
<td>60</td>
</tr>
<tr>
<td>Right Antecubital Fossa</td>
<td>40</td>
<td>75</td>
</tr>
<tr>
<td>Left Antecubital Fossa</td>
<td>50</td>
<td>80</td>
</tr>
</tbody>
</table>

*(in cm)*

Note: Catheter markings occur every 10 cms and are denoted by a thin black ring. 50 cm markings are denoted by a thick black ring. Catheter must exit introducer sheath before inflating balloon, approximately 15 cm of catheter length.
Continuous Pulmonary Artery Pressure Monitoring

1. Optimize pressure monitoring systems according to manufacturers’ recommendations.
2. Maintain patency of inner lumens with heparinized solution or continuous flush systems.
3. Observe waveforms for proper placement.
4. Catheter migration may occur. Note any damping or loss of clarity of the PA tracing as catheter position may have changed.
5. Catheter may slip back to RV. Observe waveforms for spontaneous RV tracings from catheter slipping back into RV. Note changes in the diastolic pressure.
6. Wedge the catheter with the minimum balloon inflation volume required to obtain a wedge tracing. Note the inflation volume. If <1.25 cc of volume is required, the catheter position may have changed. Consider repositioning the catheter.
7. Never use more than the recommended balloon inflation volume marked on the catheter shaft.
8. Never inflate the balloon more than the minimum required to obtain a wedge tracing.

- Full inflation with 1.5 cc inflation volume. Appropriate “a” and “v” waves noted.
- Overinflation of balloon. Note waveform rise on screen.
- Catheter too distal. Overdamping of tracing.
- Catheter spontaneous wedging. Wedge type tracing with balloon deflated.
Summary Guidelines for Safe Use of Balloon-tipped Swan-Ganz Pulmonary Artery Catheters

1. Keep catheter tip centrally located in a main branch of the pulmonary artery
   - During insertion, inflate the balloon to the full recommended volume (1.5 mL) and advance the catheter to a pulmonary artery wedge position. Deflate the balloon.
   - To reduce or remove any redundant length or loop in the right atrium or ventricle, slowly pull the catheter back 2 to 3 cm.
   - Do not advance the catheter tip too far peripherally. Ideally, the catheter tip should be located near the hilum of the lungs. Remember, the tip migrates towards the periphery of the lungs during balloon inflation. Therefore, a central location before inflation is important.
   - Keep the tip at all times in a position where a full (1.5 mL) inflation volume is necessary to produce a "wedge" tracing.

2. Anticipate spontaneous catheter tip migration toward the periphery of the pulmonary bed
   - Reduce any redundant length or loop in the right atrium or ventricle at the time of insertion to prevent subsequent peripheral migration.
   - Monitor the distal tip pressure continuously to ensure that the catheter is not inadvertently wedged with the balloon deflated (this may induce pulmonary infarction).
   - Check catheter position daily by chest X-ray film to detect peripheral placement. If migration has occurred, pull the catheter back to a central pulmonary artery position, carefully avoiding contamination of the insertion site.
   - Spontaneous catheter tip migration towards the periphery of the lung occurs during cardiopulmonary bypass. Partial catheter withdrawal (3 to 5 cm) just before bypass should be considered, as withdrawal may help reduce the amount of distal migration and may prevent permanent catheter wedging in the post-bypass period. After termination of bypass, the catheter may require repositioning. Check the distal pulmonary artery tracing before inflating the balloon.
3. Exercise caution when inflating the balloon

- If “wedge” is obtained at volumes less than 1.5 mL, pull the catheter back to a position where the full volume (1.5 mL) produces a wedge pressure tracing.

- Check the distal pressure waveform before inflating the balloon. If the waveform appears dampened or distorted, do not inflate the balloon. The catheter may be wedged with the balloon deflated. Check catheter position.

- When the balloon is reinflated to record wedge pressure, add the inflation medium (CO₂ or air) slowly under continuous monitoring of the pulmonary artery pressure waveform. Stop inflating immediately when the pulmonary artery tracing is seen to change to pulmonary artery wedge pressure. Remove the syringe to allow rapid balloon deflation, and then reattach the syringe to the balloon lumen. Air should never be used for balloon inflation in any situation where air may enter the arterial circulation.

- Never over-inflate the balloon beyond the maximum volume printed on the catheter shaft (1.5 mL). Use the volume limited syringe provided with the catheter.

- Do not use liquids for balloon inflation; they may be irretrievable and may prevent balloon deflation.

- Keep the syringe attached to the balloon lumen of the catheter to prevent accidental injection of liquids into the balloon.

4. Obtain a pulmonary artery occlusion “wedge” pressure only when necessary

- If the pulmonary artery diastolic (PAD) and the wedge (PAOP) pressures are nearly identical, wedging the balloon may not be necessary: measure PAD pressure instead of PAOP as long as the patient’s heart rate, blood pressure, cardiac output and clinical state remain stable. However, in states of changing pulmonary arterial and pulmonary venous tone (i.e., sepsis, acute respiratory failure, and shock), the relationship between PAD and “wedge” may change with the patient’s clinical condition. PAOP measurement may be necessary.

- Keep “wedge” time to a minimum (two respiratory cycles or 10 to 15 seconds), especially in patients with pulmonary hypertension.
• Avoid prolonged maneuvers to obtain wedge pressure. If difficulties are encountered, give up the “wedge.”

• Never flush the catheter when the balloon is wedged in the pulmonary artery.

5. **Patients at highest risk of pulmonary artery rupture or perforation are elderly patients with pulmonary hypertension**

• These are usually elderly patients who are undergoing cardiac surgery with anticoagulation and hypothermia. Proximal catheter tip location near the hilum of the lungs may reduce the incidence of pulmonary artery perforation.

6. **Bedside physiologic monitor settings initiated and maintained**

• Pulmonary artery pressure systolic/diastolic/mean alarm settings must be initiated to alert clinicians to a spontaneous wedge or changes in the patient status.

• Appropriate scaling should be used in order to visualize the pulmonary artery pressure waveform. Scales set too low (0-20 mmHg) may result in clipping of all or part of the waveform. Scales set too high (0-150 mmHg) may result in a “damped” appearance due to waveform compression, leading to inappropriate troubleshooting or non-recognition catheter migration into a wedge position or into the right ventricle.

• Color coding (if available) for appropriate pressure channel identification. Pulmonary artery pressures = Yellow, Right atrial pressures = Blue or per institutional policy.
Lung Zone Placement

Catheter tip location in relationship to lung zones may impact the validity of pulmonary artery wedge readings, both under normal conditions and with the application of PEEP. Lung zones are identified by the relationships among the inflow pressure (pulmonary artery pressure, PaP) the outflow pressure (pulmonary venous pressure, PvP), and the surrounding alveolar pressure (PAP).

Zone 1: PaP < PAP > PvP. No blood flow occurs from the collapsed pulmonary capillary beds. The Swan-Ganz catheter is a flow-directed catheter and the tip will not usually flow to this lung region. PAOP readings will be inaccurate.

Zone 2: PaP > PAP > PvP. Some blood flow occurs since the arterial pressure is greater than the alveolar pressure. Under some conditions catheter tip may reside in Zone 2 placement. PAOP readings may be inaccurate.

Zone 3: PaP > PAP < PvP. Capillaries are open resulting in blood flow. Catheter tip is usually below the level of the left atrium and can be verified by a lateral chest x-ray. PAOP readings will be accurate.
Ventilatory Effects on Pulmonary Artery Tracings

Spontaneous Breathing

During normal respiration, inspiration results in decreased intrathoracic pressure and increased venous return resulting in increased cardiac filling. However, the waveforms on inspiration will be negative due to the greater inspiratory decrease in intrathoracic pressure than the inspiratory increase in the cardiac volumes. On expiration, the intrathoracic pressure is relatively higher than on inspiration and will result in positive deflections in the PA and PAOP waveforms. The values recorded should be obtained at end-expiration when the intrathoracic pressure influence is minimal.
Controlled Mechanical Ventilation

When a patient is ventilated and is not spontaneously breathing, the intrathoracic pressure during inspiration is at a positive level with ventilated breaths. On expiration, the values are negative due to the relative negative intrathoracic pressure at that phase. Again, the values, PA and PAOP, are to be read at end-expiration.

![Controlled Mechanical Ventilation Tracing]

Intermittent Mandatory Ventilation

When a form of intermittent mandatory ventilation is being applied, some breaths are controlled while others are spontaneous. The impact on the tracings is that during the controlled breaths, inspiration will produce elevated waves such as those during controlled mechanical ventilation. During a spontaneous breath the tracing will revert to normal with inspiration producing a negative wave. Observation of the patient’s breathing and noting if the breaths are controlled or spontaneous assists in the proper identification of end-expiration values of pulmonary artery pressures.

![Intermittent Mandatory Ventilation Tracing]
This is a tracing of a patient who is spontaneously breathing. Identification of PA pressures and PAOP pressures are influenced by the respiratory variations noted. Pressure values should be obtained at end-expiration. Possible causes for the respiratory variation includes hypovolemia or catheter tip in a non-zone 3 placement.
Cardiac Output Determinations

There are three common indirect methods for cardiac output determinations: Fick, dye indicator dilution, and the thermodilution indicator method. The first two are primarily performed in a controlled catheterization laboratory setting. Thermodilution is most readily used at the bedside.

**Fick Method**

The “gold standard” for cardiac output determinations is based on the principles developed by Adolph Fick in the 1870’s. Fick’s concept proposes that the uptake or release of a substance by an organ is the product of blood flow through that organ and the difference between the arterial and venous values of the same substance.

The Fick method utilizes oxygen as the substance and the lungs as the organ. Arterial and venous oxygen content are measured to obtain the difference (a - v O₂). Oxygen consumption (VO₂) can be calculated from the inspired minus expired oxygen content and ventilation rate. The cardiac output can then be determined using this formula:

\[
\text{Cardiac Output} = \frac{\text{Oxygen Consumption in mL/min}}{a - v \ O_2 \ \text{Difference in vol%}}
\]

(volume % = 1 mL oxygen/100 cc)

- Normal (CaO₂) arterial oxygen content: 20 volume %
- Normal (CvO₂) mixed venous oxygen content: 15 volume %
- Normal (VO₂) oxygen consumption: 250 mL/min

Inserting these values into the equation:

\[
\text{CO} = \frac{250}{(20-15)} \times 100
\]

\[
= \frac{250}{5} \times 100
\]

\[
= 5000 \text{ mL/min or 5 L/min}
\]
Calculating cardiac output with the Fick equation requires accurate measurement of the oxygenation variables. Slight errors in the content values may produce large errors in the oxygen consumption result. Normal oxygen consumption ranges between 200–250 mL/min. Indexed normal VO₂ values are 120–160 mL/min/m². Critically ill patients may not have normal oxygen consumption values; therefore, insertion of normal values into the above Fick equation may produce erroneous cardiac output values.

**Dye Indicator Dilution Method**

Principles for the indicator dilution method were first proposed in the 1890’s by Stewart, and later refined by Hamilton.

The basis of the dye indicator technique is that a known concentration of an indicator is added to a body of fluid. After allowing adequate mixing time, the dilution of that indicator will produce the amount of fluid it was added to. A densimeter records the dye or indicator concentration in the blood after a known sample was injected upstream.

By taking continuous blood samples, a time-concentration plot, called an indicator-dilution curve can be obtained. Once this is plotted, the cardiac output can be calculated using the Stewart-Hamilton Equation:

\[
CO = \frac{I \times 60 \times 1}{Cm \times t \times k}
\]

**WHERE:**

- CO = cardiac output (1/min)
- I = amount of dye injected (mg)
- 60 = 60 sec/min
- Cm = mean indicator concentration (mg/l)
- t = total curve duration (sec)
- k = calibration factor (mg/mL/mm deflection)
Thermodilution Method

In the early 1970’s, Drs. Swan and Ganz demonstrated reliability and reproducibility of the thermodilution method with a special temperature sensing pulmonary artery catheter. Since that time, the thermodilution method of obtaining cardiac output has become a gold standard for clinical practice.

The thermodilution method applies indicator dilution principles, using temperature change as the indicator. A known amount of solution with a known temperature is injected rapidly into the proximal injectate lumen of the catheter. This cooler than blood temperature solution mixes with the surrounding blood, and the temperature is measured downstream in the pulmonary artery by a thermistor bead embedded in the catheter. The resultant change in temperature is then plotted on a time-temperature curve. This curve is similar to the one produced by the indicator-dilution method.

A modified Stewart-Hamilton equation is used to calculate the cardiac output taking into consideration the change in temperature as the indicator. Modifications include the measured temperature of the injectate and the patient’s blood temperature, along with the specific gravity of the solution injected.

\[
CO = \frac{V \times (TB-TI) \times (SI \times CI) \times 60 \times CT \times K}{A \times (SB \times CB)}
\]

WHERE:

- \( CO \) = cardiac output
- \( V \) = volume of injectate (mL)
- \( A \) = area of thermodilution curve in square mm divided by paper speed (mm/sec)
- \( K \) = calibration constant in mm/°C
- \( TB, TI \) = temperature of blood (B) and injectate (I)
- \( SB, SI \) = specific gravity of blood and injectate
- \( CB, CI \) = specific heat of blood and injectate
- \( (SI \times CI) \) = 1.08 when 5% dextrose is used
- \( (SB \times CB) \) = 60 = 60 sec/min
- \( CT \) = correction factor for injectate warning
Thermodilution Curves

A normal curve characteristically shows a sharp upstroke from rapid injection of the injectate. This is followed by a smooth curve and slightly prolonged downslope back to the baseline. Since this curve represents a change from warmer temperature to cooler and then back to warmer temperature, the actual curve is in a negative direction. The area under the curve is inversely proportional to the cardiac output.

When cardiac output is low, more time is required for the temperature to return to baseline, producing a larger area under the curve. With high cardiac output, the cooler injectate is carried more quickly through the heart, and the temperature returns to baseline faster. This produces a smaller area under the curve.

![Diagram of Thermodilution Curves]

- **Normal Cardiac Output**
- **High Cardiac Output**
- **Low Cardiac Output**
- **Artifact Due to Noise Interference**
- **Improper Injection Technique**
# Troubleshooting Key Factors in Optimizing Bolus CO Determinations

The chart below describes factors that can influence the accuracy and reproducibility of bolus thermodilution cardiac output values.

<table>
<thead>
<tr>
<th>Factor Affecting Accuracy of Bolus CO Measurement</th>
<th>Potential Error</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inaccurate Injectate Temperature:</strong>&lt;br&gt;• 1°C error in iced injectate&lt;br&gt;• 1°C error in room temperature injectate&lt;br&gt;If injectate is removed from the ice bath for:&lt;br&gt;• 15 seconds&lt;br&gt;• 30 seconds</td>
<td>± 2.7%&lt;br&gt;± 7.7%&lt;br&gt;Mean increase of 0.34 ± 0.16°C&lt;br&gt;Mean increase of 0.56 ± 0.18°C</td>
</tr>
<tr>
<td><strong>Inaccurate Injectate Volume</strong></td>
<td>0.5 mL of error in 5 mL injection: ± 10%&lt;br&gt;0.5 mL of error in 10 mL injection: ± 5%</td>
</tr>
<tr>
<td><strong>Rapid Volume Infusion During Bolus Injections:</strong>&lt;br&gt;• Room temperature infusion&lt;br&gt;• Warmed infusion</td>
<td>CO decreased 30–80%&lt;br&gt;CO decreased 20–40%</td>
</tr>
<tr>
<td><strong>Respiratory Cycle Influences</strong></td>
<td>Normal variance of 20%&lt;br&gt;Maximum variance up to 70%</td>
</tr>
<tr>
<td><strong>Inaccurate Computation Constant</strong></td>
<td>1–100%</td>
</tr>
<tr>
<td><strong>Thermal Instability Post Cardiopulmonary Bypass (CPB):</strong>&lt;br&gt;• 1–10 minutes post&lt;br&gt;• 30 minutes post</td>
<td>10–20%&lt;br&gt;Up to 9%</td>
</tr>
</tbody>
</table>
Vigilance II Monitor and Advanced Technology
Swan-Ganz System

CCombo Monitoring Systems:
CCO and SvO₂ Continuous Display

*Digital display of SVR and dual oximetry parameters available if appropriate input variables provided.*
Continuous Mixed Venous Oxygen Saturation Monitoring

**Reflection Spectrophotometry**

**Swan-Ganz Oximetry TD Catheter**
Vigilance II Monitor
Abbreviated Instructions for Use

Continuous Cardiac Output (CCO) and Mixed Venous Oxygen Saturation (SvO₂)

To Begin Oximetry Monitoring (SvO₂, ScvO₂):

For In Vitro Calibration
1. Connect catheter to optics module.
2. Select SvO₂ (Swan-Ganz catheter) or ScvO₂ (PreSep catheter) in the Large Parameter frame.
3. Select In Vitro Calibration.
4. Select Calibrate and press the knob. Wait for calibration to complete.
5. Flush catheter; check balloon. Insert catheter in PA.
6. Select START, press knob and wait for Optical Module to update.
7. SvO₂ or ScvO₂ value will appear in the Large Parameter frame.

For In Vivo SvO₂ Calibration:
1. Turn Navigation Knob to select SvO₂ or ScvO₂. Press knob.
3. Select Draw, press knob, and slowly draw waste and laboratory blood sample for co-oximeter analysis.
4. Upon receipt of lab values from drawn sample, enter venous oximetry value and either HGB or Hct.
5. Select CALIBRATE and press knob. Wait for calibration to complete.
6. Confirm that SvO₂ or ScvO₂ is displayed in the Large Parameter frame and that the values are correct.
To Transport the Optical Module:

1. After reconnecting patient cable and optical module, turn knob to select $\text{SvO}_2$ or $\text{ScvO}_2$ in the Large Parameter frame. Press knob.

2. Select RECALL OM DATA and press knob.

3. If the data in the Optical Module is <24 hours old and appears correct, select YES and press knob.

To Begin Continuous Cardiac Output (CCO) Monitoring:

1. Connect thermal filament and thermistor connections on catheter to the patient cable.

2. Press the **START/STOP CCO BUTTON** to begin Continuous Cardiac Output (CCO) monitoring. A message will appear confirming the monitor is Collecting CCO Data.

3. The average CCO value will appear in the Large Parameter frame in 1 to 8 minutes.

To Configure the Computer Display Screen:

1. To change screen display:
   - Turn Navigation knob to select the SET UP icon to change display format (temperature units, international units, time format, alarm volume, and display language).
   - Select the desired action, press knob.
   - Rotate knob to select the desired change. Press knob.
   - Select RETURN and press knob to return to the display screen.

2. To change alarm settings:
   - Select desired Large Parameter frame with Navigation knob, and press the knob.
   - Select the Alarm limit value on the lower right side of the
drop-down window. Press the knob, then rotate knob to select the upper value. Press knob to set the value. Repeat this process for the lower value.

- Rotate knob to select RETURN. Press knob to exit the drop down menu.

3. To activate the Split Screen to see STAT DISPLAY:
   - Rotate Navigation Knob to select the SPLIT SCREEN icon at the bottom of the display.
   - Only CCO(I), RVEF and EDV(I) values can be shown here. To add one of these parameters to the STAT SCREEN, select that parameter in one of the Large Parameter Frames. See the Operators Manual for a description of the STAT Screen.
   - To remove the SPLIT SCREEN, rotate the knob to select the SPLIT SCREEN icon and press the knob.

**To Display the Cardiac/Oxygen Profile:**

1. To display the Cardiac or Oxygen Patient Profile:
   - Press the Patient Data button found to the right of the Display Screen.
   - Either the Oxygen Profile or the Cardiac Profile will appear.
   - Rotate the knob to select the alternate profile at the bottom of the drop down window and press the knob to change the profile.

2. To manually enter values in the Patient Profile screens:
   - Press the Patient Data button to activate the drop down window.
   - Select the appropriate Patient Profile.
   - Rotate the knob to select the desired parameter. Press the knob.
• Enter the desired value. An asterisk will appear by the value name to designate a manually inserted value.

• Rotate knob to select exit. Press Patient Profile button to exit the Patient Profile window.

• *Note: once an asterisk appears the value must be “cleared” to be auto – updated.

To Perform Bolus Cardiac Output (ICO):

1. Press the CCO/ICO button found to the right of the Display screen. The ICO screen will appear. To exit ICO mode, press the button again.
   • Rotate the Navigation Knob to select CO or CI in the Large Parameter frames. Press the knob.
   • Select any of the options shown to make adjustments to the ICO process.
   • For automatic ICO Bolus operation, select Automatic.
   • When the monitor has established a stable baseline temperature an INJECT message appears on the screen. Inject the solution at this time. Repeat this process up to 6 times. The monitor will display the cardiac output in the BOLUS frame for each injection in the series.
   • After completing the desired number of injections, rotate the knob to select the BOLUS frame (3rd Large Parameter frame showing the values for each injection). Press the knob. The average of the injections will be shown in the CO/CI Large Parameter frame and the Bolus Edit drop down screen will appear.

2. To delete individual CO/CI values from the average:
   • Rotate the Navigation Knob to select the 3rd Large Parameter BOLUS frame.
   • Press knob to open the BOLUS EDIT window.
• Rotate and press the knob to select one or more values to delete.

• Rotate and press the knob to select REDO SERIES. Values selected for deletion will be removed and the CCO/CCI average will be displayed.

3. To Exit BOLUS CO MODE

• From the BOLUS EDIT screen, rotate the knob and select EXIT. Press the knob.

• Press the CCO/ICO button found at the right of the Display screen.

• Answer the prompt to restart Continuous Cardiac Output (CCO) by rotating the knob, selecting the answer, and pressing the knob.

To Utilize Operational Pause (alarm silence mode for use during cardiopulmonary bypass):

1. To start operational pause:

• Press and hold the Alarm Silence button for at least 3 seconds.

• The yellow Operational Pause banner appears. Data collection and display in Large Parameter frames are paused and time stamped.

• Alarms associated with these parameters are silenced since monitoring is interrupted.

• Blood Temperature and Small Parameter frame parameters are monitored and displayed.

2. To discontinue operational pause:

• Push Navigation Knob to Exit Operational Pause

• Select Yes or No with Navigation Knob when asked if you want to restart CCO. If yes is selected, CCO will start and
a new average value will appear in the Large Parameter frame within approximately 1–8 minutes.

- With the Navigation Knob, select Yes or No when asked if you want to recalibrate SvO₂ or ScvO₂. If YES, the Calibration screen will appear. If NO, SvO₂ monitoring will begin using the calibration values at the time Operational Pause was begun.

**Note: Advanced Technology Swan-Ganz Catheter Tip Position**

Keep catheter tip centrally located in a main branch of the pulmonary artery near the hilum of the lungs. Do not advance tip too far peripherally. Tip should be kept where full or near full inflation volume is required to produce a wedge tracing. The tip migrates toward periphery during balloon inflation. The proximal end of thermal filament should be located after the Tricuspid valve and be free floating within the Right Ventricle. Insertion of the thermal filament beyond the pulmonic valve may result in erroneous continuous cardiac output measurements.

**Balloon Inflation Volume**
- Appropriate inflation volume is 1.5 cc

**VIP Port 777F8, 777HF8**
- 30 cm from tip
- Located in RA/SVC

**Proximal Injectate Port**
- 26 cm from tip
- Located in RA or SVC
- If incorrectly positioned in introducer sheath, Bolus CO measurement will be erroneously high due to reflux of injectate within introducer
- Transduce Proximal Injectate Lumen – proper waveform is RA or SVC

**PA Distal Port**
- Transduce distal lumen – proper waveform is PA

**Thermistor**
- 4 cm from tip
- In main body of PA

**Thermal Filament**
- 14 – 25 cm from tip
- Rests between RA and RV
- Should be free floating and avoid endocardial surface
- Erroneous CCO measurements may result if beyond pulmonic valve

**Note:** Assess patient physiology. Atypical physiology and heart size may require special handling.
### Vigilance II Monitor Troubleshooting

#### CCO/CCI Faults

<table>
<thead>
<tr>
<th>CCO/CCI Faults</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Temp Out of Range (&lt;31° or &gt;41°C)</td>
<td>Monitored blood temperature is &lt;31° or &gt;41°C</td>
<td>Verify proper catheter position in the pulmonary artery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Confirm wedge pressure balloon inflation volume of 1.5 mL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Confirm appropriate catheter placement for patient's height, weight and insertion site</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Consider chest x-ray for evaluation of proper placement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Resume CCO monitoring when blood temperature is within range</td>
</tr>
<tr>
<td>Catheter Memory, Use Bolus Mode</td>
<td>Poor catheter thermal filament connection</td>
<td>• Verify secure thermal filament connection</td>
</tr>
<tr>
<td></td>
<td>CCO cable malfunction</td>
<td>• Check catheter/CCO cable thermal filament connections for bent/missing pins</td>
</tr>
<tr>
<td></td>
<td>Catheter CCO error</td>
<td>• Perform Patient CCO Cable Test (see manual)</td>
</tr>
<tr>
<td></td>
<td>Catheter connected is not an Edwards CCO catheter</td>
<td>• Change CCO cable</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Use Bolus CO mode</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Verify catheter is an Edwards CCO catheter</td>
</tr>
<tr>
<td>Catheter Verification, Use Bolus Mode</td>
<td>CCO cable malfunction</td>
<td>• Perform Patient CCO Cable Test (see manual)</td>
</tr>
<tr>
<td></td>
<td>Catheter CCO error</td>
<td>• Change CCO cable</td>
</tr>
<tr>
<td></td>
<td>Catheter connected is not an Edwards CCO catheter</td>
<td>• Use Bolus CO mode</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Verify catheter is an Edwards CCO catheter</td>
</tr>
<tr>
<td>Check Catheter and Cable Connection</td>
<td>Catheter thermal filament and Thermistor connections not detected</td>
<td>• Verify CCO cable and catheter connections</td>
</tr>
<tr>
<td></td>
<td>CCO cable malfunction</td>
<td>• Disconnect Thermistor and thermal filament connections and check for bent/missing pins</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Perform Patient CCO Cable Test</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Change CCO cable</td>
</tr>
<tr>
<td>Check Thermal Filament Connection</td>
<td>Catheter thermal filament connection not detected</td>
<td>• Verify that catheter thermal filament is connected securely to CCO cable</td>
</tr>
<tr>
<td></td>
<td>CCO cable malfunction</td>
<td>• Disconnect thermal filament connection and check for bent/missing pins</td>
</tr>
<tr>
<td></td>
<td>Catheter connected is not an Edwards CCO catheter</td>
<td>• Perform Patient CCO Cable Test</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Use Bolus CO mode</td>
</tr>
<tr>
<td>Check Thermal Filament Position</td>
<td>Flow around thermal filament may be reduced</td>
<td>• Flush catheter lumens</td>
</tr>
<tr>
<td></td>
<td>Thermal filament may be against vessel wall</td>
<td>• Verify proper catheter positions in the pulmonary artery</td>
</tr>
<tr>
<td></td>
<td>Catheter not in patient</td>
<td>• Confirm wedge pressure balloon inflation volume of 1.5 mL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Confirm appropriate catheter placement for patient’s height, weight and insertion site</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Consider chest x-ray for evaluation of proper placement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Resume CCO monitoring</td>
</tr>
<tr>
<td>Check Thermistor Connection</td>
<td>Catheter Thermistor connection not detected</td>
<td>• Verify that catheter-Thermistor is connected securely to CCO cable</td>
</tr>
<tr>
<td></td>
<td>Monitored blood temperature is &lt;15°C or &gt;45°C</td>
<td>• Verify that blood temperature is between 15–45°C</td>
</tr>
<tr>
<td></td>
<td>CCO cable malfunction</td>
<td>• Disconnect Thermistor connection and check for bent/missing pins</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Perform Patient CCO Cable Test</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Change CCO cable</td>
</tr>
</tbody>
</table>
## CCO/CCI Faults

<table>
<thead>
<tr>
<th>CCO/CCI Faults</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| Cardiac Output <1.0 L/min | • Measured CO <1.0 L/min | • Follow hospital protocol to increase CO 
• Resume CCO monitoring |
| Thermal Signal Loss | • Thermal signal detected by monitor is too small to process 
• Sequential compression device interference | • Verify proper catheter position in the pulmonary artery 
■ Confirm wedge pressure balloon inflation volume of 1.25–1.50 mL 
■ Confirm appropriate catheter placement for patient’s height, weight and insertion site 
■ Consider chest x-ray for evaluation of proper placement 
• Minimizing patient discomfort may reduce temperature variations 
• Temporarily turn off sequential compression device per hospital procedure |

## CCO/CCI Alerts

<table>
<thead>
<tr>
<th>CCO/CCI Alert Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| Signal Adapting – Continuing | • Large pulmonary artery blood temperature variations detected 
• Sequential compression device interference 
• Catheter thermal filament not properly positioned | • Allow more time for monitor to measure and display CCO 
• Verify proper catheter position in the pulmonary artery 
■ Confirm wedge pressure balloon inflation volume of 1.5 mL 
■ Confirm appropriate catheter placement for patient’s height, weight and insertion site 
■ Consider chest x-ray for evaluation of proper placement 
• Minimizing patient discomfort may reduce temperature variations 
• Temporarily turn off sequential compression device per hospital procedure |
| Unstable Blood Temp – Continuing | • Large pulmonary artery blood temperature variations detected 
• Sequential compression device interference | • Wait for CO measurement to be updated 
• Minimizing patient discomfort may reduce temperature variations 
• Temporarily turn off sequential compression device per hospital procedure |
| SV: Heart Rate Signal Loss | • Patient’s time-averaged heart rate out of range (HR <30 or >200 bpm) 
• No heart rate detected 
• ECG interface cable connection not detected | • Wait until average heart rate is within range 
• Select appropriate lead configuration to maximize heart rate triggers 
• Verify cable connection between Vigilance II monitor and bedside monitor is secure 
• Change ECG interface cable |

## CCO/CCI General Troubleshooting

<table>
<thead>
<tr>
<th>CCO/CCI Topic</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| CCI > CCO | • Incorrect patient BSA 
• BSA < 1 | • Verify units of measure and values for patient’s height and weight |
| CCO ≠ BOLUS CO | • Incorrectly configured bolus information 
• Faulty Thermistor or injectate probe 
• Unstable baseline temperature affecting bolus CO measurements | • Verify that computation constant, injectate volume, and catheter size have been correctly selected 
• Use “iced” injectate and/or 10 mL injectate volume to create a large thermal signal 
• Verify correct injection technique 
• Change injectate temperature probe |
## SVR/SVRI Messages and Troubleshooting

### SVR/SVRI Alerts and General Troubleshooting

<table>
<thead>
<tr>
<th>SVR/SVRI Alert Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVR: Slaved-In Pressures Signal Loss</td>
<td>• Vigilance II analog input port not configured to accept MAP and CVP</td>
<td>• Verify correct voltage range and low/high voltage values on the Vigilance II monitor for external monitor</td>
</tr>
<tr>
<td></td>
<td>• Analog input interface cable connections not detected</td>
<td>• Verify cable connection between the Vigilance II monitor and bedside monitor is secure</td>
</tr>
<tr>
<td></td>
<td>• Inaccurate input signal</td>
<td>• Verify correct height/weight entries and units of measure for patient’s BSA</td>
</tr>
<tr>
<td></td>
<td>• External monitor malfunction</td>
<td>• Check for signal at external monitor’s analog output device</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Change external device module, if used</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SVR/SVRI Topic</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVR &gt; SVRI</td>
<td>• Incorrect patient BSA</td>
<td>• Verify units of measure and values for patient’s height and weight</td>
</tr>
<tr>
<td>Vigilance II MAP and CVP ≠ External Monitor</td>
<td>• Vigilance II monitor configured incorrectly</td>
<td>• Verify correct voltage range and low/high voltage values on the Vigilance II monitor for external monitor</td>
</tr>
<tr>
<td></td>
<td>• Inaccurate input signal</td>
<td>• Confirm correct units of measure for analog input port voltage values (mmHg or kPa)</td>
</tr>
<tr>
<td></td>
<td>• External monitor malfunction</td>
<td>• Verify correct height/weight entries and units of measure for patient’s BSA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Check for signal at external monitor’s analog output device</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Change analog input interface cable</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Change external device module, if used</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Clear asterisk (*) from Cardiac Profile screen for MAP and CVP if slaving from an external device</td>
</tr>
</tbody>
</table>
### Oximetry Messages and Troubleshooting

#### OXIMETRY FAULTS AND ALERTS

<table>
<thead>
<tr>
<th>Oximetry Fault Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| **Light Range**         | • Poor optical module/catheter connection  
                          • Debris or film obstructing optical module/catheter connection lens  
                          • Optical module malfunction  
                          • Catheter kinked or damaged | • Verify secure optical module/catheter connection  
                          • Clean optical module/catheter connectors with 70% isopropyl alcohol and swab, let air dry and recalibrate  
                          • Replace catheter if damage is suspected and recalibrate |
| **OM Disconnected**     | • Optimal module connection at monitor not detected  
                          • Bent or missing optical module connector pins | • Verify secure optical module/catheter connection  
                          • Check optical module cable connector for bent/missing pins |
| **OM Memory**           | • Optimal module memory malfunction | • Change optical module and recalibrate |
| **Value Out of Range**  | • Incorrectly entered oximetry, HGB or Hct values  
                          • Incorrect HGB units of measure  
                          • Calculated oximetry value is outside of the 0–99% range | • Verify correctly entered oximetry, HGB and Hct values  
                          • Verify correct HGB unit of measure  
                          • Obtain updated oximetry lab values and recalibrate |
| **Red/IR Transmit**     | • Debris or film obstructing optical module/catheter connection lens  
                          • Optical module malfunction | • Clean module/catheter connections with 70% isopropyl alcohol and swab, let air dry and recalibrate  
                          • Change optical module and recalibrate |
| **OM Temperature**      | • Optical module malfunction | • Change optical module and recalibrate |
| **Oximetry Not Available** | • Internal system malfunction | • Power monitor off and on to restore system  
                          • If problem persists, contact Edwards Technical Support |

<table>
<thead>
<tr>
<th>Oximetry Alert Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| **SQI = 4**             | • Low blood flow at catheter tip or catheter tip against vessel wall  
                          • Significant changes in HGB/Hct values  
                          • Catheter tip clotted  
                          • Catheter kinked or damaged | • Verify proper catheter position. For $SvO_2$, verify proper catheter position in the pulmonary artery  
                          • Confirm wedge pressure balloon inflation volume of 1.5 mL (for $SvO_2$ only)  
                          • Confirm appropriate catheter placement for patient’s height, weight and insertion site  
                          • Consider chest x-ray for evaluation of proper placement  
                          • Aspirate then flush distal lumen per hospital protocol  
                          • Update HGB/Hct values using Update function  
                          • Check catheter for kinking and recalibrate  
                          • Replace catheter if damage is suspected and recalibrate |
### Oximetry Warnings

<table>
<thead>
<tr>
<th>Oximetry Warning Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| In Vitro Calibration Error | • Poor optical module and catheter connection  
• Calibration cup wet  
• Catheter kinked or damaged  
• Optical module malfunction  
• Catheter tip is not in catheter package calibration cup | • Verify secure optical module/catheter connection  
• Straighten any visible kinks; replace catheter if damage is suspected  
• Change optical module and recalibrate  
• Verify catheter tip is securely seated in calibration cup  
• Perform in vivo calibration |
| Unstable Signal            | • Changing oximetry, HGB/Hct, or unusual hemodynamic values                     | • Stabilize patient per hospital protocol and perform in vivo calibration          |
| Wall Artifact or Wedge Detected | • Low blood flow at catheter tip  
• Catheter tip clotted  
• Catheter tip wedged in vessel or against vessel wall | • Aspirate then flush distal lumen per hospital protocol  
• Verify proper catheter position. For SvO₂, verify proper catheter position in the pulmonary artery  
  ▪ Confirm wedge pressure balloon inflation volume of 1.5 mL (for SvO₂ only)  
  ▪ Confirm appropriate catheter placement for patient’s height, weight and insertion site  
  ▪ Consider chest x-ray for evaluation of proper placement  
• Perform in vivo calibration |
| Optical Module Not Calibrated – Select oximetry to calibrate | • Optical module has not been calibrated (in vivo or in vitro)  
• Recall OM data function has not been performed  
• Optical module malfunction | • Perform in vivo or in vitro calibration  
• Perform Recall OM Data function if module was previously calibrated  
• Change optical module and recalibrate |
| Patient Data in Optical Module more than 24 hours old | • Last optical module calibration > 24 hours old  
• Date and time on Vigilance II monitors at facility are incorrect | • Perform in vivo calibration  
• Synchronize date and time on all monitors at facility |

### Oximetry General Troubleshooting

<table>
<thead>
<tr>
<th>Oximetry Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| Optical Module Not Calibrated – Select oximetry to calibrate | • Optical module has not been calibrated (in vivo or in vitro)  
• Recall OM data function has not been performed  
• Optical module malfunction | • Perform in vivo or in vitro calibration  
• Perform Recall OM Data function if module was previously calibrated  
• Change optical module and recalibrate |
| Patient Data in Optical Module more than 24 hours old | • Last optical module calibration > 24 hours old  
• Date and time on Vigilance II monitors at facility are incorrect | • Perform in vivo calibration  
• Synchronize date and time on all monitors at facility |
### CEDV Messages and Troubleshooting

#### CEDV Alerts

<table>
<thead>
<tr>
<th>CEDV Alert Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| Heart Rate Signal Loss | • Patient’s time-averaged heart rate out of range (HR < 30 or >200 bpm)  
• No heart rate detected  
• ECG interface cable connection not detected | • Wait until average heart rate is within range  
• Select appropriate lead configuration to maximize heart rate triggers  
• Verify cable connection between the Vigilance II monitor and bedside monitor is secure  
• Change ECG interface cable |
| Irregular ECG Pattern | • Physiological change in patient’s status  
• Unsecured leads/connections of ECG signal  
• Double-sensing due to atrial or atrial-ventricular (AV) pacing | • Follow standard hospital protocol to stabilize patient’s status  
• Reposition leads or reconnect ECG interface cable  
• Reposition reference lead to minimize atrial spike sensing  
• Select appropriate lead configuration to maximize heart rate triggers and minimize atrial spike sensing  
• Assess correct milliamperage (mA) for pacing level |
| Signal Adapting – Continuing | • Patient’s respiratory pattern may have changed  
• Sequential compression device interference  
• Catheter thermal filament not properly positioned | • Allow more time for monitor to measure and display EDV  
• Temporarily turn off sequential compression device per hospital procedure  
• Verify proper catheter position in the pulmonary artery  
  ▪ Confirm wedge pressure balloon inflation volume of 1.5 mL  
  ▪ Confirm appropriate catheter placement for patient’s height, weight and insertion site  
  ▪ Consider chest x-ray for evaluation of proper placement |
| Vigilance II HRAVG ≠ External Monitor HR | • External monitor not optimally configured for ECG signal output  
• External monitor malfunction  
• ECG interface cable malfunction | • Stop CCO and verify heart rate is the same for the Vigilance II monitor and external monitor  
• Select appropriate lead configuration to maximize heart rate triggers and minimize atrial spike sensing  
• Verify signal output from external monitoring device; if necessary, change module  
• Change ECG interface cable |

#### ICO (Bolus) Messages and Troubleshooting

#### ICO Faults

<table>
<thead>
<tr>
<th>ICO Fault Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
</table>
| Check Thermistor Connection | • Catheter Thermistor connection not detected  
• Monitored blood temperature is < 15°C or > 45°C  
• CCO cable malfunction | • Verify that catheter Thermistor is connected securely to CCO cable  
• Verify that blood temperature is between 15 – 45°C  
• Disconnect Thermistor connection and check for bent/missing pins  
• Change CCO cable |
| IT out of range, Check Probe | • Injectate temperature < 0°C, > 30°C or > BT  
• Injectate temperature probe malfunction  
• CCO cable malfunction | • Verify injectate fluid temperature  
• Check injectate probe connections for bent/missing pins  
• Change injectate temperature probe  
• Change CCO cable |
### ICO Faults [Cont]

<table>
<thead>
<tr>
<th>ICO Fault Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Check Injectate Probe Connection</td>
<td>• Injectate temperature probe not detected&lt;br&gt;• Injectate temperature probe malfunction&lt;br&gt;• CCO cable malfunction</td>
<td>• Verify connection between CCO cable and injectate temperature probe&lt;br&gt;• Change injectate temperature probe&lt;br&gt;• Change CCO cable</td>
</tr>
<tr>
<td>Injectate Volume not valid</td>
<td>• In-line probe injectate volume must be 5 mL or 10 mL</td>
<td>• Change injectate volume to 5 mL or 10 mL&lt;br&gt;• Use a bath type probe for an injectate volume of 3 mL</td>
</tr>
</tbody>
</table>

### ICO Alerts

<table>
<thead>
<tr>
<th>ICO Alert Messages</th>
<th>Possible Cause(s)</th>
<th>Suggested Action(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Curve Not Detected</td>
<td>• No bolus injection detected for &gt; 4 minutes (Automatic mode) or 30 seconds (Manual mode)</td>
<td>• Restart Bolus CO monitoring and proceed with injections</td>
</tr>
<tr>
<td>Extended Curve</td>
<td>• Thermodilution curve slow to return to baseline&lt;br&gt;• Injectate port in introducer sheath&lt;br&gt;• Possible cardiac shunt</td>
<td>• Verify correct insertion technique&lt;br&gt;• Verify proper catheter position in the pulmonary artery&lt;br&gt;• Confirm wedge pressure balloon inflation volume of 1.5 mL&lt;br&gt;• Confirm appropriate catheter placement for patient’s height, weight and insertion site&lt;br&gt;• Consider chest x-ray for evaluation of proper placement&lt;br&gt;• Ensure injectable port location is outside of the introducer sheath&lt;br&gt;• Use “iced” injectate and/or 10 mL injectate volume to create a large thermal signal</td>
</tr>
<tr>
<td>Irregular Curve</td>
<td>• Thermodilution curve has multiple peaks</td>
<td>• Verify correct injectate technique&lt;br&gt;• Verify proper catheter position in the pulmonary artery&lt;br&gt;• Confirm wedge pressure balloon inflation volume of 1.5 mL&lt;br&gt;• Confirm appropriate catheter placement for patient’s height, weight and insertion site&lt;br&gt;• Consider chest x-ray for evaluation of proper placement&lt;br&gt;• Use “iced” injectate and/or 10 mL injectate volume to create a large thermal signal</td>
</tr>
<tr>
<td>Unstable Baseline</td>
<td>• Large pulmonary artery blood temperature variations detected</td>
<td>• Allow time for blood temperature baseline to stabilize&lt;br&gt;• Use manual mode</td>
</tr>
<tr>
<td>Warm Injectate</td>
<td>• Injectate temperature within 8°C of blood temperature&lt;br&gt;• Injectate temperature probe malfunction&lt;br&gt;• CCO cable malfunction</td>
<td>• Use cooler injectate fluid&lt;br&gt;• Change injectate temperature probe&lt;br&gt;• Change CCO cable</td>
</tr>
</tbody>
</table>
RVEDV Quick Reference

1. Parameters Attained with Vigilance II Monitor

- **CARDIAC OUTPUT (CO)** = 4 – 8.0 L/min

- **CARDIAC INDEX (CI)** = 2.5 – 4.0 L/min/m²

- **STROKE VOLUME (SV)**: The volume of blood ejected from the ventricle in each beat.
  
  \[ SV = \frac{CO}{HR \times 1000} \]

  Normal SV: 60 – 100 mL/beat
  Normal SVI: 33 – 47 mL/beat/m²

- **END-DIASTOLIC VOLUME (EDV)**: The volume of blood in the ventricle at the end of the diastole.
  
  \[ EDV = \frac{SV}{EF} \]

  Normal RV EDV: 100 – 160 mL
  Normal RV EDVI: 60 – 100 mL/m²

- **END-SYSTOLIC VOLUME (ESV)**: The volume of blood in the ventricle at the end of systole.
  
  \[ ESV = EDV - SV \]

  Normal RV ESV: 50 – 100 mL
  Normal RV ESVI: 30 – 60 mL/m²

- **EJECTION FRACTION (EF)**: The percentage of blood ejected from the ventricle each beat.
  
  \[ EF = \frac{EDV - ESV}{EDV} \quad \text{or} \quad \frac{SV}{EDV} \]

  Normal RVEF: 40 – 60%

*(Note: As with all measurements in hemodynamic monitoring, the absolute number is not as important as trends and changes in response to therapy.)*
2. Goal of RV Volumetric Measurements

- Optimize RV Efficiency
- Optimize the relationship between EDV and SV
  a. In an efficient state, an increase in PRELOAD (EDV) will result in an INCREASE in STROKE VOLUME (SV).
  b. Prior to reaching the FLAT PART of the curve, an increase in PRELOAD (EDV) will increase SV while not causing a decrease in Ejection Fraction.
  c. On the FLAT PART of the curve, a further increase in PRELOAD (EDV) will not result in an increase in SV.

At this point, a further increase in volume may:

- Decrease oxygen supply
- Increase oxygen demand
- Decrease left ventricular compliance

Therapy should be directed at increasing contractility or reducing afterload.
Idealized Ventricular Function Curves

<table>
<thead>
<tr>
<th>Ventricular Function Indicators</th>
<th>SV mL/beat</th>
<th>LVSWI gm·m⁻²·beat⁻¹</th>
<th>CI L/Min·m⁻²</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>60-75</td>
<td>&gt;3.5</td>
<td></td>
</tr>
</tbody>
</table>

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>60</td>
<td>50</td>
<td>2.2-2.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.0</td>
</tr>
</tbody>
</table>

I. **Normal Perfusion**
   No Pulmonary Congestion

II. **Normal Perfusion**
   Pulmonary Congestion

III. **Hypoperfusion**
    No Pulmonary Congestion

IV. **Hypoperfusion**
   Pulmonary Congestion

**Possible Interventions**

1 = ↑ Preload; moves along same curve, volume
2 = ↓ Preload; moves along same curve, diuretic/venodilator
3 = ↑ Contractility; shifts to higher curve, minimal change in preload, positive inotrope
4 = ↓ Afterload; shifts to a higher curve at a lower preload, afterload reducters, vasodilators
Swan-Ganz Reference Chart

The chart below describes the wide breadth of line of the Swan-Ganz catheters manufactured by Edwards Lifesciences.

### Advanced Technology Catheters – Continuous Hemodynamic Monitoring

<table>
<thead>
<tr>
<th>Catheter Model Number</th>
<th>Lumens</th>
<th>Length (cm)</th>
<th>PAP/PAOP</th>
</tr>
</thead>
<tbody>
<tr>
<td>CCOMbo/CEDV/VIP</td>
<td>777HF8</td>
<td>7</td>
<td>110</td>
</tr>
<tr>
<td>CCOMbo/CEDV</td>
<td>774HF75</td>
<td>6</td>
<td>110</td>
</tr>
<tr>
<td>CCO/CEDV</td>
<td>177HF75</td>
<td>6</td>
<td>110</td>
</tr>
<tr>
<td>CCOMbo/VIP</td>
<td>746HF8</td>
<td>7</td>
<td>110</td>
</tr>
<tr>
<td>CCOMbo</td>
<td>744HF75</td>
<td>6</td>
<td>110</td>
</tr>
<tr>
<td>CCO</td>
<td>139HF75(P)</td>
<td>6</td>
<td>110</td>
</tr>
<tr>
<td>SvO2</td>
<td>741HF75</td>
<td>6</td>
<td>110</td>
</tr>
</tbody>
</table>

### Standard Thermodilution Catheters (some models available in S-Tip, T-Tip, C-Tip and various stiffness characteristics to facilitate femoral approach)

<table>
<thead>
<tr>
<th>Catheter</th>
<th>Model Number</th>
<th>Lumens</th>
<th>Length (cm)</th>
<th>PAP/PAOP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Base TD</td>
<td>131HF7</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>VIP</td>
<td>831HF75(P)</td>
<td>5</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>VIP+</td>
<td>834HF75</td>
<td>6</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>Pediatric TD</td>
<td>132F5</td>
<td>4</td>
<td>75</td>
<td>•</td>
</tr>
<tr>
<td>Adults with Small Vessels TD</td>
<td>096F6P</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>Base TD Hi-Shore</td>
<td>141HF7P</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>Base TD S-Tip</td>
<td>151F7</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>CardioCath</td>
<td>143HTF7</td>
<td>5</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>ControlCath C tip (non PVC) (non-latex)</td>
<td>K9FC146F7</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>ControlCath C tip (non PVC)</td>
<td>C144F7</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>ControlCath S tip (non PVC)</td>
<td>S144HF7</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
</tbody>
</table>

### Pacing Catheters and Thermodilution Paceport Catheters

(Use with models D98100 – Chandler Transluminal V-Pacing Probe and/or D98500 – Flex-Tip Transluminal A-Pacing)

<table>
<thead>
<tr>
<th>Catheter</th>
<th>Model Number</th>
<th>Lumens</th>
<th>Length (cm)</th>
<th>PAP/PAOP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paceport</td>
<td>931HF75</td>
<td>5</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>A-V Paceport</td>
<td>991HF8</td>
<td>6</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>Pacing TD-A, V, or A-V Pacing</td>
<td>D200HF7</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>Bipolar Pacing (Femoral)</td>
<td>D97130F5</td>
<td>1</td>
<td>90</td>
<td>•</td>
</tr>
<tr>
<td>Bipolar Pacing</td>
<td>D97120F5</td>
<td>1</td>
<td>90</td>
<td>•</td>
</tr>
<tr>
<td>VIP Bipolar Pacing</td>
<td>D97140HF5</td>
<td>2</td>
<td>100</td>
<td>•</td>
</tr>
</tbody>
</table>

### Monitoring Catheters

<table>
<thead>
<tr>
<th>Catheter</th>
<th>Model Number</th>
<th>Lumens</th>
<th>Length (cm)</th>
<th>PAP/PAOP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Double Lumen Monitoring</td>
<td>110F5</td>
<td>2</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td></td>
<td>111F7</td>
<td>2</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td></td>
<td>123F6P</td>
<td>2</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>Triple Lumen Monitoring</td>
<td>114F7P</td>
<td>4</td>
<td>110</td>
<td>•</td>
</tr>
<tr>
<td>Pediatric Double Lumen Monitoring</td>
<td>116F4</td>
<td>2</td>
<td>60</td>
<td>•</td>
</tr>
<tr>
<td>Small French Oximetry</td>
<td>040HF4</td>
<td>2</td>
<td>40</td>
<td>•</td>
</tr>
</tbody>
</table>
This chart can be used as a quick ready reference guide to choose a catheter specific to the needs of the patient.

<table>
<thead>
<tr>
<th>Distance from Tip</th>
<th>Recommended Introducer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Proximal</strong></td>
<td><strong>Injectate Port</strong></td>
</tr>
<tr>
<td>26 cm</td>
<td>30 cm</td>
</tr>
<tr>
<td>26 cm</td>
<td></td>
</tr>
<tr>
<td>26 cm</td>
<td>30 cm</td>
</tr>
<tr>
<td>26 cm</td>
<td>30 cm</td>
</tr>
<tr>
<td>26 cm</td>
<td></td>
</tr>
<tr>
<td>30 cm</td>
<td>30 cm</td>
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<tr>
<td>30 cm</td>
<td></td>
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<tr>
<td>30 cm</td>
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<tr>
<td>30 cm</td>
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<tr>
<td>30 cm</td>
<td></td>
</tr>
<tr>
<td>30 cm</td>
<td></td>
</tr>
<tr>
<td>19 cm</td>
<td></td>
</tr>
<tr>
<td>20 cm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>12 cm</td>
<td></td>
</tr>
<tr>
<td>12 cm</td>
<td></td>
</tr>
<tr>
<td>15 cm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This is a reference chart only and is not a complete list of catheters. All model numbers with an "H" contain AMC Thromboshield, an antibacterial heparin coating which decreases viable microbe count on surface of product during handling and placement. Many catheters are available with or without heparin coating.
### COMPUTATION CONSTANTS FOR THERMODILUTION (BATH) CARDIAC OUTPUTS

<table>
<thead>
<tr>
<th>Swan-Ganz Catheter Model</th>
<th>Cold Injectate (0-5°C)</th>
<th>Room Injectate (19-22°C)</th>
<th>Room Injectate (23-25°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10cc</td>
<td>5cc</td>
<td>3cc</td>
</tr>
<tr>
<td>096F6</td>
<td>0.547</td>
<td>0.259</td>
<td>0.144</td>
</tr>
<tr>
<td>131F7</td>
<td>0.542</td>
<td>0.247</td>
<td>0.132</td>
</tr>
<tr>
<td>132F5</td>
<td>--</td>
<td>0.270</td>
<td>0.154</td>
</tr>
<tr>
<td>141HF7</td>
<td>0.542</td>
<td>0.247</td>
<td>0.132</td>
</tr>
<tr>
<td>143HTF7</td>
<td>0.554</td>
<td>0.259</td>
<td>--</td>
</tr>
<tr>
<td>C144F7 / S144F7</td>
<td>0.547</td>
<td>0.252</td>
<td>0.134</td>
</tr>
<tr>
<td>C145HF6</td>
<td>0.547</td>
<td>0.252</td>
<td>0.134</td>
</tr>
<tr>
<td>151F7</td>
<td>0.542</td>
<td>0.247</td>
<td>0.132</td>
</tr>
<tr>
<td>139F75 / 177F75</td>
<td>0.564</td>
<td>0.257</td>
<td>0.143</td>
</tr>
<tr>
<td>746F8 / 777F8</td>
<td>0.550</td>
<td>0.256</td>
<td>--</td>
</tr>
<tr>
<td>831HF75</td>
<td>0.564</td>
<td>0.262</td>
<td>0.139</td>
</tr>
<tr>
<td>834HF75</td>
<td>0.564</td>
<td>0.257</td>
<td>0.143</td>
</tr>
<tr>
<td>931HF75</td>
<td>0.564</td>
<td>0.262</td>
<td>0.139</td>
</tr>
<tr>
<td>991HF8</td>
<td>0.568</td>
<td>0.268</td>
<td>0.147</td>
</tr>
</tbody>
</table>

### COMPUTATION CONSTANTS EDWARDS CO-SET+ CLOSED INJECTATE DELIVERY SYSTEM

<table>
<thead>
<tr>
<th>Swan-Ganz Catheter Model</th>
<th>Cold Injectate</th>
<th>Room Injectate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10cc (6-12°C)</td>
<td>5cc (8-16°C)</td>
</tr>
<tr>
<td>096F6</td>
<td>0.558</td>
<td>0.277</td>
</tr>
<tr>
<td>131F7</td>
<td>0.561</td>
<td>0.259</td>
</tr>
<tr>
<td>132F5</td>
<td>--</td>
<td>0.285</td>
</tr>
<tr>
<td>141HF7</td>
<td>0.561</td>
<td>0.259</td>
</tr>
<tr>
<td>143HTF7</td>
<td>0.569</td>
<td>0.266</td>
</tr>
<tr>
<td>C144F7</td>
<td>0.570</td>
<td>0.271</td>
</tr>
<tr>
<td>C145HF6 / S145HF6</td>
<td>0.570</td>
<td>0.271</td>
</tr>
<tr>
<td>151F7</td>
<td>0.561</td>
<td>0.259</td>
</tr>
<tr>
<td>139F75 / 177F75</td>
<td>0.574</td>
<td>0.287</td>
</tr>
<tr>
<td>746F8 / 777F8</td>
<td>0.559</td>
<td>0.263</td>
</tr>
<tr>
<td>831HF75</td>
<td>0.578</td>
<td>0.272</td>
</tr>
<tr>
<td>834HF75</td>
<td>0.574</td>
<td>0.287</td>
</tr>
<tr>
<td>931HF75</td>
<td>0.578</td>
<td>0.272</td>
</tr>
<tr>
<td>991HF8</td>
<td>0.553</td>
<td>0.277 (8-12°C)</td>
</tr>
</tbody>
</table>
Perioperative Goal-Directed Therapy

Advancing Critical Care Through Science-Based Education Since 1972
Perioperative Goal-Directed Therapy

A growing body of evidence suggests that conventional care using static hemodynamic parameters may increase the risk of postoperative complications. Hemodynamic optimization through Perioperative Goal-Directed Therapy (PGDT) in high-risk surgery has been shown to reduce postoperative complications, including acute kidney injury (AKI), surgical site infections (SSI), urinary tract infection (UTI), pneumonia, and major/minor GI complications, and may help improve your patients’ postoperative recovery.

Both hypo- and hypervolemia may deleteriously affect perioperative organ function. The graphic below describes the goal of PGDT. Exceeding or falling short of this range may result in increased morbidity. Hemodynamic optimization through PGDT using advanced hemodynamic parameters can ensure the adequacy of resuscitation.

**Perioperative Goal-Directed Therapy:** A clinician-directed treatment protocol, which defines and treats to a goal, using dynamic and flow-based parameters (such as SVV, SV, ScvO₂ and DO₂) to improve patient outcomes and reduce complications.
**PGDT Benefits**

**Improves clinical and economic outcomes**

PGDT has shown statistically significant clinical and economic benefits across a wide range of surgical procedures. These studies include 5 meta-analyses spanning 27 randomized controlled trials, which have shown important benefits for PGDT in high-risk surgeries with large fluid shifts.

In multiple studies, Perioperative Goal-Directed Therapy showed a dramatic reduction in postoperative complications and costs.

<table>
<thead>
<tr>
<th>Surgical Procedure</th>
<th>Patient Complications</th>
<th>COST SAVINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>Acute Kidney Injury (AKI)</td>
<td>Four studies explore the overall costs associate with acute kidney injury (AKI); costs are $19211 higher in patients with AKI</td>
</tr>
<tr>
<td>GI (upper/lower)</td>
<td>Hospital-Associated Pneumonia</td>
<td>Eight studies extrapolate the costs of hospital-associated pneumonia; in an 8-year nationwide analysis, postoperative pneumonia generates $46400 in incremental costs, in trauma patients that excess cost rises to $64544</td>
</tr>
<tr>
<td>Hepatobiliary</td>
<td>Major Gastrointestinal</td>
<td>Four studies explore the costs of one or more major gastrointestinal (GI) complications; across all general surgeries the attributable excess cost of major GI complications is $77483</td>
</tr>
<tr>
<td>Hip Replacement</td>
<td>Minor Gastrointestinal</td>
<td>Three studies explore the costs of one or more minor gastrointestinal complications; across all general surgeries the attributable excess cost of minor GI complications is $8296</td>
</tr>
<tr>
<td>Neuro</td>
<td>Urinary Tract Infection (UTI)</td>
<td>Five studies provide cost estimates for urinary tract infections (UTI) in a hospital setting; surgical patients who acquire UTIs generate $12828 in excess costs</td>
</tr>
<tr>
<td>Trauma</td>
<td>Surgical Site Infection (SSI)</td>
<td>Seven studies provide estimates of the cost of surgical site infections (SSI); median costs for patients with SSIs are $27979 higher than for controls</td>
</tr>
<tr>
<td>Urological Vascular</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Perioperative Goal-Directed Therapy Improves Outcomes vs Conventional Care

Hemodynamic optimization through PGDT has been shown to reduce post-surgical complications in moderate- and high-risk surgery, and reduce hospital length of stay, when compared to conventional care using static parameters such as Heart Rate (HR), Mean Arterial Pressure (MAP), Central Venous Pressure (CVP), and urine output. By comparison, PGDT protocols use dynamic flow-based parameters such as Stroke Volume (SV) and Stroke Volume Variation (SVV), which are more sensitive and specific.
**PGDT Protocols**

The following algorithm is provided to help you select the most appropriate Perioperative Goal-Directed Therapy (PGDT) protocol for your patient. Your selection deserves careful consideration.

Is my patient at risk* of developing post-op complications?

- **YES**
  - Consider PGDT
    - Any limitation† to the use of SVV?
      - **YES**
        - Use an SV-based treatment protocol (NHS, Cecconi)
      - **NO**
        - Use an SVV-based treatment protocol (Benes, Ping, Ramsingh)
  - **NO**
    - Conventional care (eg BP, CVP, HR, urine output)

* At risk because of comorbidities or the surgical procedure itself.

† Limitations to the use of SVV: spontaneous breathing, tidal volume <7 mL/kg, open chest, atrial fibrillation, right ventricular failure, and laproscopic surgery.
**BENES PROTOCOL**

**STUDY DESIGN**
Randomized controlled trial

**PATIENT POPULATION**
Undergoing elective abdominal surgery >2 h with expected blood loss >1000 ml

**INCLUSION CRITERIA**
One or more of the following: Ischemic heart disease or severe heart dysfunction, moderate to severe chronic obstructive pulmonary disease, aged 70+, ASA III or more

**TARGET PARAMETERS**
Central Venous Pressure, Stroke Volume Variation, Cardiac Index

**INTERVENTION**
Fluid (Colloid), Dobutamine

**PRIMARY OUTCOMES**
Decrease in 30-day postoperative complications (56%), decrease in hospital length of stay (10%)

BENES PROTOCOL (continued)

Measure and record SVV, CI

SVV ≥10% and CVP <15 mmHg

NO

Repeat monitoring of SVV, CI during next 5 minutes

NO

YES

Colloid bolus 3 ml/kg over 5 minutes

YES

CVP rise ≤3 mm Hg

NO

NO

NO

Dobutamine infusion to reach CI ≥2.5 l/min/m²

YES

SVV <10% and no change or decrease of CI

YES

Cl <2.5 l/min/m²

YES
CECCONI PROTOCOL

STUDY DESIGN
Randomized controlled trial

PATIENT POPULATION
Undergoing elective total hip replacement under regional anesthesia

INCLUSION CRITERIA
ASA II

TARGET PARAMETERS
Stroke Volume, Oxygen Delivery

INTERVENTION
Fluid (Colloid), Dobutamine

PRIMARY OUTCOMES
Decrease in postoperative complications (20%)
CECCONI PROTOCOL (continued)

Keep:

- $\text{SaO}_2 > 95\%$
- $\text{Hb} > 8 \text{ mg/dl}$
- $\text{HR} < 100 \text{ bpm}$
- MAP between 60 and 100 mmHg

Achieve SV max and then target $\text{DO}_2\text{I}$ to 600 ml/min·m²

250 ml HES bolus

Increase of SV $> 10\%$ or blood loss $> 250$ ml during fluid challenge

NO

SV stable $> 20$ min

NO

See oxygen delivery

YES

$\text{DO}_2\text{I}^+$

$\geq 600 \text{ ml/min·m}^2$

NO

Dobutamine:
Increase by 3 mcg/kg·min
Decrease or STOP if $\text{HR} > 100 \text{ bpm}$
or signs of cardiac ischemia

YES

Check every 10 minutes
If $\text{DO}_2\text{I}$ falls below 600 ml/min·m², restart algorithm

† Resuscitation to achieve a $\text{DO}_2\text{I}$ value of 600 is presented as a goal and not intended to be a hard target. This protocol is intended as guidance, and healthcare professionals should use sound clinical judgment and individualize therapy to each specific patient care situation.

NHS-NICE/KUPER PROTOCOL

STUDY DESIGN
Quality improvement program (before-after comparison)

PATIENT POPULATION
Undergoing emergency and elective abdominal, orthopedic, gynecologic, urologic, and vascular surgery

INCLUSION CRITERIA
Three cohorts of patients aged ≤60, 61-71, and ≥71 years with ASA >I

TARGET PARAMETERS
Stroke Volume

INTERVENTION
Fluid

PRIMARY OUTCOMES
3.7-day decrease in hospital length of stay (25%)


(continued on next page)
Measure SV

200-250 ml fluid over 5-10 minutes

SV increase >10%?

NO

Monitor SV for clinical signs of fluid loss

SV reduction >10%

YES

NO


**Wang Protocol**

**Study Design**
Randomized controlled trial

**Patient Population**
Undergoing radical gastrectomy, colon cancer resection, rectal cancer, and Whipple surgery

**Inclusion Criteria**
ASA I or ASA II

**Target Parameters**
Stroke Volume Variation

**Intervention**
Fluid

**Primary Outcomes**
Faster recovery time to normal diet (16%), decrease in hospital length of stay (19%)

WANG PROTOCOL (continued)

Maintain SVV 11%-13%

Fluid to maintain the patient in the range

BP drops >30% from baseline

HR <50 beats/min

NO

YES

Atropine .5 mg

Ephedrine 6 mg

YES
**Ramsingh Protocol**

**Study Design**
Randomized, single-blinded controlled trial

**Patient Population**
Undergoing major abdominal surgery, urologic, gastrointestinal or gynecologic cancer resection, and Whipple surgery

**Inclusion Criteria**
P-POSSUM mean predicted mortality rate of 1.4*

**Target Parameters**
Stroke Volume Variation

**Intervention**
Fluid (Colloid)

**Primary Outcomes**
Faster return of GI function (3 vs 4 days), faster return of PO intake (4 vs 5 days), and a 2.5-day decrease in hospital length of stay (33%)

---


* No differences other than age were statistically significant. P-POSSUM scores predicted mortality and showed no difference between the groups.
Ramsingh Protocol (continued)

GDT Group
(ventilate 8 ml/kg)

SVV >12% NO Monitor SVV and CO

YES

250 ml Albumin bolus
(may repeat to max of 20 ml/kg)

NO

>20 ml/kg Albumin?

NO

SVV >12%

YES

Crystalloid 3:1 replacement
(consider PRBCs, monitor ABGs)

YES
DONATI PROTOCOL

STUDY DESIGN
Multicenter randomized controlled trial

PATIENT POPULATION
Undergoing elective abdominal extensive surgery or abdominal aortic surgery

INCLUSION CRITERIA
ASA II

TARGET PARAMETERS
Central Venous Pressure, Oxygen Extraction Ratio

INTERVENTION
Fluid (Colloid), Dobutamine

PRIMARY OUTCOMES
Decrease in postoperative complications (60%), decrease in hospital length of stay (16%)

**DONATI PROTOCOL (continued)**

Pre-op (T0):
- Arterial and central venous line
  - Check SaO₂—ScvO₂—calculate O₂ER†

Intra-op (T1):
- Group A
  - O₂ER† (hourly)
  - ≤27%
  - No change
    - (or decrease Dobutamine)
  - >27%
  - CVP
    - CVP <10 mmHg or SVV >12%
    - Fluid challenge
      - Colloids (when Hb >10 g/dl)
      - PBC (when Hb <10 g/dl)
      - If O₂ER† still >27%
    - CVP >10 mmHg or SVV < 12%
    - Dobutamine

- Group B
  - Standard management
    - (MAP, urine output, CVP)

Post-op (T2):
- Similar management to intra-op
  - Checks of O₂ER† at the end of anesthesia, 0.5, 1, 2, and 6 hours, and day +1

† O₂ER is estimated based on use of ScvO₂.
Quick Reference

ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972

Note: The following algorithms and protocols are for educational reference only. Edwards does not endorse or support any one specific algorithm or protocol. It is up to each individual clinician and institution to select the treatment that is most appropriate.
Advanced Technology Swan-Ganz Catheter Algorithm

**SvO₂**

60–80%

- **CCO**
  - 4–8 lpm

- **HR**
  - 60–100 bpm
  - Optimal HR
  - Pacing
  - Optimal R-R
  - Optimal P-R

- **SV**
  - 60–100 mL/beat
  - Preload
  - Afterload
  - Contractility
  - RVEDVI
    - 60–100 mL/m²
  - SVR
    - 800–1200 dynes-sec-cm⁻²
  - RVEF
    - 40–60%
  - SVRI
    - 1970–2390 dynes-sec⁻¹-m²
  - RVSWI
    - 5–10 Gm-m/m²/beat
  - PADP
    - 8–15 mmHg
  - PVR
    - <250 dynes-sec-cm⁻²
  - SVI
    - 33–47 mL/beat/m²

- **Hemoglobin**
  - Hb 12–16 g/dL
  - Hct 35–45%

- **Oxygenation**
  - SaO₂ 98%
  - PaO₂ >80 mmHg

- **Metabolic Demand**
  - VO₂ 200–250 mL/min

- **Bleeding**
  - SaO₂
  - PaO₂
  - FiO₂

- **Hemodilution**
  - Ventilation
  - PEEP

- **Anemia**
  - Work of Breathing

**Oxygen Delivery**

- DO₂=CaO₂ x CO x 10
- 950–1150 mL/min

**Oxygen Consumption**

- VO₂=200–250 mL/min
Advanced Minimally-Invasive Algorithm

**Quick Reference**

- **Advanced Minimally-Invasive Algorithm**

- **FloTrac CCO**
  - 4–8 lpm

- **HR**
  - 60–100 bpm
  - Optimal HR
  - Pacing
  - Optimal R-R
  - Optimal P-R

- **SV**
  - 60–100 mL/beat
  - Preload
  - Afterload
  - Contractility
  - SVV 13%
  - SVR 800–1200 dynes-sec-cm⁻¹
  - SVI 33–47 mL/beat/m²
  - CVP 2–6 mmHg
  - SVRI 1970–2390 dynes-sec-cm⁻¹-m²

- **Hemoglobin**
  - Hb 12–16 g/dL
  - Hct 35–45%
  - Bleeding
  - Hemodilution
  - Anemia

- **Oxygenation**
  - SaO₂ 98%
  - PaO₂ >80 mmHg
  - SaO₂
  - PaO₂
  - FiO₂
  - Ventilation
  - PEEP

- **Metabolic Demand**
  - VO₂ 200–250 mL/min
  - Shivering
  - Fever
  - Anxiety
  - Pain
  - Muscle Activity
  - Work of Breathing

- **Oxygen Delivery**
  - DO₂=CaO₂ x CO x 10
  - 950–1150 mL/min

- **Oxygen Consumption**
  - VO₂=200–250 mL/min

- **ScvO₂ 70%**
Advanced Swan-Ganz Catheter Goal-Directed Protocol

Resuscitate to a mean arterial pressure of >65 mmHg

**SvO₂**
- Normal (60–80%)
  - Do Nothing
- Low (<60%)
- High (>80%)
  - Evaluate Tissue Oxygenation
  - Lactate levels
  - Base deficit

**SaO₂**
- Low (Hypoxemia)
  - Oxygen therapy, Increase PEEP
- Normal (>95%) (Increased O₂ER)

**Cardiac Output**
- High Cl (>2.5 L/min/m²)
- Low Cl (<2.0 L/min/m²)

**Hemoglobin**
- >8 gm/dL stress, anxiety, pain (high VO₂)
  - Analgesia
  - Sedation

**PAOP/RVEDVI**
- <8 gm/dL anemia
  - Blood Transfusion
- PAOP >18mmHg RVEDVI >140 mL/m² myocardial dysfunction
  - Dobutamine
- PAOP <10 mmHg RVEDVI <80 mL/m² hypovolemia
  - Fluid Challenge

Advanced Minimally-Invasive
Goal-Directed Protocol

Resuscitate to a mean arterial pressure of >65 mmHg

**ScvO₂**
- Normal (>70%)
- Low (<70%)
- High (>80%)

Do Nothing

**SaO₂**
- Low (Hypoxemia)
- Normal (>95%) (Increased O₂ER)

Oxygen therapy, Increase PEEP

**FloTrac**
Cardiac Output
- High Cl (>2.5 L/min/m²)
- Low Cl (<2.0 L/min/m²)

Hemoglobin
- >8 gm/dL stress, anxiety, pain (high VO₂)
- <8 gm/dL anemia

Analgesia Sedation

Blood Transfusion

SVV
- **SVV <10%** myocardial dysfunction
- **SVV >15%** hypovolemia

Dobutamine

Fluid Challenge


* Used within the limitations of SVV as a guide for fluid responsiveness.

** Cardiac Output response to fluid challenge or passive leg raising when SVV cannot be used.
Protocol for Early Goal-Directed Therapy

Supplemental oxygen ± endotracheal intubation & mechanical ventilation

Central venous & arterial catheterization

Sedation, paralysis (if intubated), or both

CVP

<8 mmHg

Crystalloid

≥8 mmHg

Colloid

MAP

<65 mmHg

Vasoactive agents

≥65 mmHg

Transfusion of red cells until hematocrit ≥30%

≥90 mmHg

Inotropic agents

ScvO₂

<70%

No

<65 mmHg and ≤90 mmHg

Goals achieved

≥70%

Yes

Hospital admission

Physiologic Algorithm Using SVV, SVI and ScvO₂

Volume Responsive: SVV >13%

- No
  - SVI Normal
  - SVI Low

ScvO₂ evaluate O₂ extraction

- ? Pressor**
- ? Inotrope*

Re-evaluate DO₂, O₂ extraction, SVV & SVI

* If O₂ extraction is high, an inotrope may be required to provide perfusion support.

** As individual organ perfusion may also depend on blood pressure, a MAP target > 60-65 mmHg may require a vasopressor even when O₂ extraction is normal.
Physiologic Optimization Program
Using SVV and SVI In Hypotensive and/or Oliguric Patients

Volume responsive patients: SVV > 10-15% receive volume therapy titrated against both SVV and SVI.

For non-volume responsive patients, SVV < 10-15% the physiology is interrogated at the level of cardiac performance on a beat to beat basis. Ultimately with this approach, many patients will develop a SVI ≥ normal (pathway 1). This represents resuscitated septic shock, and these patients may be safely placed on a vasopressor knowing that volume resuscitation has been accomplished and additional volume is not helpful.

Pathway 2 patients typically have poor cardiac performance related to either systolic or diastolic heart failure. Echocardiography is important in defining appropriate therapy in this subset of patients. Inotropes are not indicated in those with good ejection fraction.

In Pathway 3 volume therapy is stopped and diuretics will be beneficial for those who go on to develop ALI / ARDS typically after the initial resuscitation phase. (McGee, 2009).

---

Early Goal-Directed Therapy in Moderate to High-Risk Cardiac Surgery Patients

<table>
<thead>
<tr>
<th>Condition</th>
<th>HR</th>
<th>MAP</th>
<th>CO/CI</th>
<th>CVP/RAP</th>
<th>PAP/PAOP</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Ventricular Failure</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>Pulmonary Edema (Cardiogenic)</td>
<td>↑</td>
<td>N,↓</td>
<td>↓</td>
<td>↑</td>
<td>↑PAOP &gt;25 mmHg</td>
<td></td>
</tr>
<tr>
<td>Massive Pulmonary Embolism</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↑ N</td>
<td>↑PAD &gt; PAOP by &gt;5 mmHg</td>
<td>↑PVR</td>
</tr>
<tr>
<td>Acute Ventricular Septal Defect</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑giant “v” waves on PAOP tracing</td>
<td>O₂ step up noted in SvO₂</td>
</tr>
<tr>
<td>Acute Mitral Valve Regurgitation</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑giant “v” waves on PAOP tracing</td>
<td>No O₂ step up noted in SvO₂</td>
</tr>
<tr>
<td>Cardiac Tamponade</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑CVP, PAD and PAOP equalized</td>
<td>↓RVEDVI</td>
</tr>
<tr>
<td>Right Ventricular Failure</td>
<td>↑,V</td>
<td>↓,V</td>
<td>↓</td>
<td>↑</td>
<td>PAP ↑, PAOP N/↓</td>
<td>↑RVEDVI</td>
</tr>
<tr>
<td>Hypovolemic Shock</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑Oxygen extraction ↑SVR</td>
</tr>
<tr>
<td>Cardiogenic Shock</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>N,↑</td>
<td>↑</td>
<td>↑Oxygen extraction ↑SVR</td>
</tr>
<tr>
<td>Septic Shock</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↓,N</td>
<td>↓,N</td>
<td>SVR changes, ↓Oxygen extraction ↓SVR</td>
</tr>
</tbody>
</table>
# New York Heart Classification of Cardiovascular Disease

<table>
<thead>
<tr>
<th>Class</th>
<th>Subjective Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal cardiac output without systemic or pulmonary congestion; asymptomatic at rest and on heavy exertion</td>
</tr>
<tr>
<td>II</td>
<td>Normal cardiac output maintained with a moderate increase in pulmonary systemic congestion; symptomatic on exertion</td>
</tr>
<tr>
<td>III</td>
<td>Normal cardiac output maintained with a marked increase in pulmonary-systemic congestion; symptomatic on mild exercise</td>
</tr>
<tr>
<td>IV</td>
<td>Cardiac output reduced at rest with a marked increase in pulmonary-systemic congestion; symptomatic at rest</td>
</tr>
</tbody>
</table>

# Forrester Classification Hemodynamic Subsets of Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>Subset Clinical Description</th>
<th>Cardiac index L/min/m²</th>
<th>PAOP mmHg</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>I  No Failure</td>
<td>2.7 ± 0.5</td>
<td>12 ± 7</td>
<td>Sedate</td>
</tr>
<tr>
<td>II Isolated Pulmonary Congestion</td>
<td>2.3 ± 0.4</td>
<td>23 ± 5</td>
<td>Normal BP: Diuretics ↑ BP: Vasodilators</td>
</tr>
<tr>
<td>III Isolated Peripheral Hypoperfusion</td>
<td>1.9 ± 0.4</td>
<td>12 ± 5</td>
<td>↑ HR: Add volume ↓ HR: Pacing</td>
</tr>
<tr>
<td>IV Both Pulmonary Congestion and Hypoperfusion</td>
<td>1.6 ± 0.6</td>
<td>27 ± 8</td>
<td>↓ BP: Inotropes Normal BP: Vasodilators</td>
</tr>
</tbody>
</table>
### GLASGOW COMA SCALE

<table>
<thead>
<tr>
<th>Neurological Function</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye Opening</td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To sound</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>Never</td>
<td>1</td>
</tr>
<tr>
<td>Best Motor Response</td>
<td></td>
</tr>
<tr>
<td>Obeys commands</td>
<td>6</td>
</tr>
<tr>
<td>Localizes pain</td>
<td>5</td>
</tr>
<tr>
<td>Flexion (withdraws)</td>
<td>4</td>
</tr>
<tr>
<td>Flexion (abnormal)</td>
<td>3</td>
</tr>
<tr>
<td>Extension</td>
<td>2</td>
</tr>
<tr>
<td>None (flaccid)</td>
<td>1</td>
</tr>
<tr>
<td>Best Verbal Response</td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused conversation</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
</tbody>
</table>

### ATLS CHART

#### Estimated Fluid and Blood Requirements in a 70kg Male

<table>
<thead>
<tr>
<th>INITIAL PRESENTATIONS</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood loss (mL)</td>
<td>&lt;750</td>
<td>750–1500</td>
<td>1500–2000</td>
<td>&gt;2000</td>
</tr>
<tr>
<td>Blood loss (% blood volume)</td>
<td>&lt;15%</td>
<td>15%–30%</td>
<td>30%–40%</td>
<td>&gt;40%</td>
</tr>
<tr>
<td>Pulse rate (bpm)</td>
<td>&lt;100</td>
<td>&gt;100</td>
<td>&gt;120</td>
<td>&gt;140</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure (mmHg)</td>
<td>Normal or increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Respiratory rate (bpm)</td>
<td>14–20</td>
<td>20–30</td>
<td>30–40</td>
<td>&gt;35</td>
</tr>
<tr>
<td>Urine output (mL/hr)</td>
<td>30 or more</td>
<td>20–30</td>
<td>5–15</td>
<td>Negligible</td>
</tr>
<tr>
<td>CNS-Mental status</td>
<td>Slightly anxious</td>
<td>Mildly anxious</td>
<td>Anxious and confused</td>
<td>Confused and lethargic</td>
</tr>
<tr>
<td>Fluid replacement</td>
<td>Crystalloid</td>
<td>Crystalloid</td>
<td>Crystalloid + blood</td>
<td>Crystalloid + blood</td>
</tr>
</tbody>
</table>
**Fluid Challenge Guideline Chart**

<table>
<thead>
<tr>
<th>PAOP* mmHg</th>
<th>Challenge Volume Amount/10 Minutes</th>
<th>CVP* mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;12 mmHg</td>
<td>200 mL or 20 cc/minute</td>
<td>&lt;6 mmHg</td>
</tr>
<tr>
<td>12–16–18 mmHg</td>
<td>100 mL or 10 cc/minute</td>
<td>6–10 mmHg</td>
</tr>
<tr>
<td>&gt;16–18 mmHg</td>
<td>50 mL or 5 cc/minute</td>
<td>&gt;10 mmHg</td>
</tr>
</tbody>
</table>

- Re-profile at the end of 10 minutes or fluid challenge
- Discontinue challenge if PAOP increased >7 mmHg or CVP increased >4 mmHg
- Repeat challenge if PAOP increased <3 mmHg or CVP increased <2 mmHg
- Observe patient for 10 minutes and re-profile if PAOP increased >3 mmHg, but <7 mmHg or CVP increased >2 mmHg or <4 mmHg
- Observe SVI and RVEDVI if RV volume values are available
- Discontinue challenge if: SVI fails to increase by at least 10% and RVEDVI increases by 25% or RVEDVI is >140 mL/m² and PAOP increases >7 mmHg

**Optional Baseline RVEDVI Value Guidelines:**
- If RVEDVI <90 mL/m² or mid-range 90-140 mL/m², administer fluid challenge
- If RVEDVI >140 mL/m², do not administer fluid challenge

*References differ on PAOP and CVP ranges*
### APACHE II SEVERITY OF DISEASE CLASSIFICATION SYSTEM

<table>
<thead>
<tr>
<th></th>
<th>High Abnormal Range</th>
<th>Low Abnormal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+4</td>
<td>+3</td>
</tr>
<tr>
<td><strong>Temperature-rectal (°C)</strong></td>
<td>≥41°</td>
<td>39–40.9°</td>
</tr>
<tr>
<td><strong>Mean Arterial Pressure - mmHg</strong></td>
<td>≥160</td>
<td>130–159</td>
</tr>
<tr>
<td><strong>Heart Rate</strong> (ventricular response)</td>
<td>≥180</td>
<td>140–179</td>
</tr>
<tr>
<td><strong>Respiratory Rate (bpm)</strong> (non-ventilated or ventilated)</td>
<td>≥50</td>
<td>35–49</td>
</tr>
<tr>
<td><strong>Oxygenation</strong> A-aDO₂ or PaO₂ (mmHg) a. FIO₂ ≥0.5 record A-aDO₂, b. FIO₂ ≤0.5 record only PaO₂</td>
<td>≥500</td>
<td>350–499</td>
</tr>
<tr>
<td><strong>Arterial pH</strong></td>
<td>≥7.7</td>
<td>7.6–7.69</td>
</tr>
<tr>
<td><strong>Serum Sodium</strong> (mMol/L)</td>
<td>≥180</td>
<td>160–179</td>
</tr>
<tr>
<td><strong>Serum Potassium</strong> (mMol/L)</td>
<td>≥3.5</td>
<td>2–3.4</td>
</tr>
<tr>
<td><strong>Serum Creatinine</strong> (mg/100 mL) (Double point score for acute renal failure)</td>
<td>≥60</td>
<td>50–59.9</td>
</tr>
<tr>
<td><strong>Hematocrit (%)</strong></td>
<td>≥40</td>
<td>20–39.9</td>
</tr>
<tr>
<td><strong>White Blood Count</strong> (total/ mm³) (in 1,000s)</td>
<td>≥52</td>
<td>41–51.9</td>
</tr>
</tbody>
</table>

### A. Acute Physiology Score (APS):

Sum of the 12 individual variable points from the chart above.
B. Age Points:
Assign points to age as shown in chart at right:

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤44</td>
<td>0</td>
</tr>
<tr>
<td>45–54</td>
<td>2</td>
</tr>
<tr>
<td>55–64</td>
<td>3</td>
</tr>
<tr>
<td>65–74</td>
<td>5</td>
</tr>
<tr>
<td>≥75</td>
<td>6</td>
</tr>
</tbody>
</table>

C. Chronic Health Points:
If the patient has a history of severe organ system insufficiency or is immunocompromised, assign points as follows:

a. for nonoperative or emergency postoperative patients - 5 points
or
b. for elective postoperative patient - 2 points

Definitions
Organ insufficiency or immunocompromised state must have been evident prior to this hospital admission and conform to the following criteria:

Liver: Biopsy-proven cirrhosis and documented portal hypertension; episodes of past upper GI bleeding attributed to portal hypertension; or prior episodes of hepatic failure/encephalopathy/coma.

Cardiovascular: New York Heart Association Class IV.

Respiratory: Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction, i.e., unable to climb stairs or perform household duties; or documented chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension (>40 mmHg), or respiratory dependency.

Renal: Receiving chronic dialysis.

Immunocompromised: Immunosuppression, chemotherapy, radiation, long-term or recent high-dose steroids, or has a disease that is sufficiently advanced to suppress resistance to infection, e.g., leukemia, lymphoma, AIDS.

APACHE II Score
Sum of A + B + C

A. APS points
B. Age points
C. Chronic health points

Total Apache II
ACC/AHA 2004 Guidelines
Pulmonary Artery Catheter and Arterial Pressure Monitoring

Recommendations for Pulmonary Artery Catheter Monitoring:

Class I

1. Pulmonary artery catheter monitoring should be performed for the following:
   a. Progressive hypotension, when unresponsive to fluid administration or when fluid administration may be contraindicated
   b. Suspected mechanical complications of STEMI, (i.e., VSR, papillary muscle rupture, or free wall rupture with pericardial tamponade) if an echocardiogram has not been performed

Class IIa

1. Pulmonary artery catheter monitoring can be useful for the following:
   a. Hypotension in a patient without pulmonary congestion who has not responded to an initial trial of fluid administration
   b. Cardiogenic shock
   c. Severe or progressive CHF or pulmonary edema that does not respond rapidly to therapy
   d. Persistent signs of hypoperfusion without hypotension or pulmonary congestion
   e. Patients receiving vasopressor/inotropic agents
Class III
1. Pulmonary artery catheter monitoring is not recommended in patients with STEMI without evidence of hemodynamic instability or respiratory compromise.

Recommendations for Intra-arterial Pressure Monitoring:

Class I
1. Intra-arterial pressure monitoring should be performed for the following:
   a. Patients with severe hypotension (systolic arterial pressure less than 80 mmHg)
   b. Patients receiving vasopressor/inotropic agents
   c. Cardiogenic shock

Class II
1. Intra-arterial pressure monitoring can be useful for patients receiving intravenous sodium nitroprusside or other potent vasodilators.

Class IIb
1. Intra-arterial pressure monitoring might be considered in patients receiving intravenous inotropic agents.

Class III
1. Intra-arterial pressure monitoring is not recommended for patients with STEMI who have no pulmonary congestion and have adequate tissue perfusion without use of circulatory support measures.
## Normal Hemodynamic Parameters and Laboratory Values

### Normal Hemodynamic Parameters – Adult

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Equation</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial Blood Pressure (BP)</td>
<td>Systolic (SBP) Diastolic (DBP)</td>
<td>100–140 mmHg 60–90 mmHg</td>
</tr>
<tr>
<td>Mean Arterial Pressure (MAP)</td>
<td>SBP + (2 x DBP)/3</td>
<td>70–105 mmHg</td>
</tr>
<tr>
<td>Right Atrial Pressure (RAP)</td>
<td></td>
<td>2–6 mmHg</td>
</tr>
<tr>
<td>Right Ventricular Pressure (RVP)</td>
<td>Systolic (RVSP) Diastolic (RVDP)</td>
<td>15–30 mmHg 0–8 mmHg</td>
</tr>
<tr>
<td>Pulmonary Artery Pressure (PAP)</td>
<td>Systolic (PASP) Diastolic (PADP)</td>
<td>15–30 mmHg 8–15 mmHg</td>
</tr>
<tr>
<td>Mean Pulmonary Artery Pressure (MPAP)</td>
<td>PASP + (2 x PADP)/3</td>
<td>9–18 mmHg</td>
</tr>
<tr>
<td>Pulmonary Artery Occlusion Pressure (PAOP)</td>
<td></td>
<td>6–12 mmHg</td>
</tr>
<tr>
<td>Left Atrial Pressure (LAP)</td>
<td></td>
<td>4–12 mmHg</td>
</tr>
<tr>
<td>Cardiac Output (CO)</td>
<td>HR x SV/1000</td>
<td>4.0–8.0 L/min</td>
</tr>
<tr>
<td>Cardiac Index (CI)</td>
<td>CO/BSA</td>
<td>2.5–4.0 L/min/m²</td>
</tr>
<tr>
<td>Stroke Volume (SV)</td>
<td>CO/HR x 1000</td>
<td>60–100 mL/beat</td>
</tr>
<tr>
<td>Stroke Volume Index (SVI)</td>
<td>CI/HR x 1000</td>
<td>33–47 mL/m²/beat</td>
</tr>
<tr>
<td>Stroke Volume Variation (SVV)</td>
<td>SVmax–SVmin/SVmean x 100</td>
<td>&lt;10–15%</td>
</tr>
<tr>
<td>Systemic Vascular Resistance (SVR)</td>
<td>80 x (MAP–RAP)/CO</td>
<td>800–1200 dynes-sec-cm⁻³</td>
</tr>
<tr>
<td>Systemic Vascular Resistance Index (SVRI)</td>
<td>80 x (MAP–RAP)/CI</td>
<td>1970–2390 dynes-sec-cm⁻³-m²</td>
</tr>
<tr>
<td>Pulmonary Vascular Resistance (PVR)</td>
<td>80 x (MPAP–PAOP)/CO</td>
<td>&lt;250 dynes-sec-cm⁻⁵</td>
</tr>
<tr>
<td>Pulmonary Vascular Resistance Index (PVRI)</td>
<td>80 x (MPAP–PAOP)/CI</td>
<td>255–285 dynes-sec-cm⁻³-m²</td>
</tr>
<tr>
<td>Left Ventricular Stroke Work Index (LVSWI)</td>
<td>SVI x (MAP–PAOP) x 0.0136</td>
<td>50–62 g/m²/beat</td>
</tr>
<tr>
<td>Right Ventricular Stroke Work Index (RVSWI)</td>
<td>SVI x (MPAP–CVP) x 0.0136</td>
<td>5–10 g/m²/beat</td>
</tr>
<tr>
<td>Coronary Artery Perfusion Pressure (CPP)</td>
<td>Diastolic BP–PAOP</td>
<td>60–80 mmHg</td>
</tr>
<tr>
<td>Right Ventricular End-Diastolic Volume (RVEDV)</td>
<td>SV/EF</td>
<td>100–160 mL</td>
</tr>
<tr>
<td>Right Ventricular End-Diastolic Volume Index (RVEDVI)</td>
<td>RVEDV/BSA</td>
<td>60–100 mL/m²</td>
</tr>
<tr>
<td>Right Ventricular End-Systolic Volume (RVESV)</td>
<td>EDV–SV</td>
<td>50–100 mL</td>
</tr>
<tr>
<td>Right Ventricular Ejection Fraction (RVEF)</td>
<td>SV/EDV x 100</td>
<td>40–60%</td>
</tr>
<tr>
<td>Parameter</td>
<td>Equation</td>
<td>Normal Range</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Partial Pressure of Arterial Oxygen (PaO₂)</td>
<td>(0.0138 x Hgb x SaO₂) + 0.0031 x PaO₂</td>
<td>75–100 mmHg</td>
</tr>
<tr>
<td>Partial Pressure of Arterial CO₂ (PaCO₂)</td>
<td></td>
<td>35–45 mmHg</td>
</tr>
<tr>
<td>Bicarbonate (HCO₃⁻)</td>
<td></td>
<td>22–26 mEq/L</td>
</tr>
<tr>
<td>pH</td>
<td></td>
<td>7.34–7.44</td>
</tr>
<tr>
<td>Arterial Oxygen Saturation (SaO₂)</td>
<td></td>
<td>95–100%</td>
</tr>
<tr>
<td>Mixed Venous Saturation (SvO₂)</td>
<td></td>
<td>60–80%</td>
</tr>
<tr>
<td>Central Venous Oxygen Saturation (ScvO₂)</td>
<td></td>
<td>70%</td>
</tr>
<tr>
<td>Arterial Oxygen Content (CaO₂)</td>
<td>(0.0138 x Hgb x SaO₂) + 0.0031 x PaO₂</td>
<td>16–22 mL/dL</td>
</tr>
<tr>
<td>Venous Oxygen Content (CvO₂)</td>
<td>(0.0138 x Hgb x SvO₂) + 0.0031 x PvO₂</td>
<td>15 mL/dL</td>
</tr>
<tr>
<td>A-V Oxygen Content Difference (Cₐ-v)O₂</td>
<td>CaO₂ – CvO₂</td>
<td>4–6 mL/dL</td>
</tr>
<tr>
<td>Oxygen Delivery (DO₂)</td>
<td>CaO₂ x CO x 10</td>
<td>950–1150 mL/min</td>
</tr>
<tr>
<td>Oxygen Delivery Index (DO₂/I)</td>
<td>CaO₂ x CI x 10</td>
<td>500–600 mL/min/m²</td>
</tr>
<tr>
<td>Oxygen Consumption (VO₂)</td>
<td>Cₐ-vO₂ x CO x 10</td>
<td>200–250 mL/min</td>
</tr>
<tr>
<td>Oxygen Consumption Index (VO₂/I)</td>
<td>Cₐ-vO₂ x CI x 10</td>
<td>120–160 mL/min/m²</td>
</tr>
<tr>
<td>Oxygen Extraction Ratio (O₂ER)</td>
<td>(CaO₂ – CvO₂)/CaO₂ x 100</td>
<td>22–30%</td>
</tr>
<tr>
<td>Oxygen Extraction Index (O₂EI)</td>
<td>(SaO₂ – SvO₂)/SaO₂ x 100</td>
<td>20–25%</td>
</tr>
<tr>
<td>Extra Vascular Lung Water (EVLW)</td>
<td>CO x DST - 0.25 GEDV</td>
<td>3-7 mL/kg</td>
</tr>
<tr>
<td>Extra Vascular Lung Water Index (ELWI)</td>
<td>EVLW/PBW</td>
<td></td>
</tr>
<tr>
<td>Predicted Body Weight (PBW):</td>
<td>Female: 45.5 = 0.91 x (Height-152.4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male: 50 = 0.91 x (Height-152.4)</td>
<td></td>
</tr>
<tr>
<td>Global End Diastolic Volume (GEDV)</td>
<td>CO x MTt x f(S1/S2)</td>
<td>680–800 mL/m²</td>
</tr>
<tr>
<td>Global End Diastolic Volume Index (GEDI)</td>
<td>CI x MTt x f(S1/S2)</td>
<td></td>
</tr>
<tr>
<td>Global Ejection Fraction (GEF)</td>
<td>SV x 4 / GEDV</td>
<td>&gt;20%</td>
</tr>
<tr>
<td>Cardiac Function Index (CFI)</td>
<td>1000 x CO / GEDV</td>
<td>4.5-6.6 1/min</td>
</tr>
<tr>
<td>Intra Thoracic Blood Volume (ITBV)</td>
<td>ITBV = 1.25 x GEDV</td>
<td>850-1000 mL/m³</td>
</tr>
<tr>
<td>Intra Thoracic Blood Volume Index (ITBI)</td>
<td>ITBI = 1.25 x GEDI</td>
<td></td>
</tr>
<tr>
<td>Pulmonary Vascular Permeability Index (PVPI)</td>
<td>EVLW/0.25 x GEDV</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Cardiac Power (CPO)</td>
<td>CO x MAP x K</td>
<td>0.5-0.7 W/m²</td>
</tr>
<tr>
<td>Cardiac Power Index (CPI)</td>
<td>CI x MAP x K</td>
<td></td>
</tr>
</tbody>
</table>
### Normal Blood Laboratory Values

#### Chemistry Studies

<table>
<thead>
<tr>
<th>Test</th>
<th>Conventional Units (Reference Values)</th>
<th>SI Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (Na)</td>
<td>135–145 mEq/L</td>
<td>135–145 mmol/L</td>
</tr>
<tr>
<td>Potassium (K)</td>
<td>3.5–5.0 mEq/L</td>
<td>3.5–5.0 mmol/L</td>
</tr>
<tr>
<td>Chloride (Cl)</td>
<td>100–108 mEq/L</td>
<td>100–108 mmol/L</td>
</tr>
<tr>
<td>Carbon Dioxide (CO₂)</td>
<td>22–26 mEq/L</td>
<td>22–26 mmol/L</td>
</tr>
<tr>
<td>Glucose (BS)</td>
<td>70–100 mg/dL</td>
<td>3.9–6.1 mmol/L</td>
</tr>
<tr>
<td>Blood Urea Nitrogen (BUN)</td>
<td>8–20 mg/dL</td>
<td>2.9–7.5 mmol/L</td>
</tr>
<tr>
<td>Creatine kinase (CK)</td>
<td>Males: 55–170 U/L</td>
<td>Males: 0.94–2.89 μkat/L</td>
</tr>
<tr>
<td></td>
<td>Females: 30–135 U/L</td>
<td>Females: 0.51–2.3 μkat/L</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.6–1.2 mg/dL</td>
<td>53–115 μmol/L</td>
</tr>
<tr>
<td>Calcium (Ca)</td>
<td>8.2–10.2 mEq/L</td>
<td>2.05–2.54 mmol/L</td>
</tr>
<tr>
<td>Magnesium (Mg)</td>
<td>1.3–2.1 mg/dL</td>
<td>0.65–1.05 mmol/L</td>
</tr>
<tr>
<td>Bilirubin (direct/indirect)</td>
<td>&lt;0.5–1.1 mg/dL</td>
<td>&lt;6.8–19 μmol/L</td>
</tr>
<tr>
<td>Amylase</td>
<td>25–85 U/L</td>
<td>0.39–1.45 μkat/L</td>
</tr>
<tr>
<td>Lipase</td>
<td>&lt;160 U/L</td>
<td>&lt;2.72 μkat</td>
</tr>
<tr>
<td>Anion Gap</td>
<td>8–14 mEq/L</td>
<td>8–14 mmol/L</td>
</tr>
<tr>
<td>Lactate</td>
<td>0.93–1.65 mEq/L</td>
<td>0.93–1.65 mmol/L</td>
</tr>
<tr>
<td>Alanine Aminotransferase</td>
<td>8–50 IU/L</td>
<td>0.14–0.85 μkat/L</td>
</tr>
<tr>
<td>(ALT, GPT)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspartate Aminotransferase</td>
<td>7–46 U/L</td>
<td>0.12–0.78 μkat/L</td>
</tr>
<tr>
<td>(AST, GOT)</td>
<td></td>
<td></td>
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</table>

#### Hemotologic Studies

<table>
<thead>
<tr>
<th>Test</th>
<th>Conventional Units (Reference Values)</th>
<th>SI Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red Blood Cells</td>
<td>Males: 4.5–5.5 million/μL</td>
<td>4.5–5.5 x 10¹²/L</td>
</tr>
<tr>
<td></td>
<td>Females: 4–5 million/μL</td>
<td>4–5 x 10¹²/L</td>
</tr>
<tr>
<td>White Blood Cells (WBC)</td>
<td>4,000–10,000/μL</td>
<td>4–10 x 10⁹/L</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>Males: 12.4–17.4 g/dL</td>
<td>124–174 g/L</td>
</tr>
<tr>
<td></td>
<td>Females: 11.7–16 g/dL</td>
<td>117–160 g/L</td>
</tr>
<tr>
<td>Hematocrit (Hct)</td>
<td>Males: 42%–52%</td>
<td>0.42–0.52</td>
</tr>
<tr>
<td></td>
<td>Females: 36%–48%</td>
<td>0.36–0.48</td>
</tr>
<tr>
<td>Test</td>
<td>Conventional Units (Reference Values)</td>
<td>SI Units</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>---------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Lipids/Lipoproteins Studies</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Cholesterol: Desirable Range</td>
<td>Males: &lt;205 mg/dL  Females: &lt;190 mg/dL</td>
<td>&lt;5.3 mmol/L  &lt;4.9 mmol/L</td>
</tr>
<tr>
<td></td>
<td><strong>LDL Cholesterol: Desirable Range</strong></td>
<td><strong>&lt;130 mg/dL</strong></td>
</tr>
<tr>
<td></td>
<td>Males: 37–70 mg/dL  Females: 40–85 mg/dL</td>
<td>0.96–1.8 mmol/L  1.03–2.2 mmol/L</td>
</tr>
<tr>
<td></td>
<td><strong>HDL Cholesterol: Desirable Range</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Males: 37–70 mg/dL  Females: 40–85 mg/dL</td>
<td>0.96–1.8 mmol/L  1.03–2.2 mmol/L</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>Males: 44–180 mg/dL  Females: 11–190 mg/dL</td>
<td>0.44–2.01 mmol/L  0.11–2.21 mmol/L</td>
</tr>
<tr>
<td><strong>Coagulation Studies</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platelet Count</td>
<td>150,000–400,000/mm³</td>
<td></td>
</tr>
<tr>
<td>Prothrombin Time (PT)</td>
<td>10–13 sec</td>
<td></td>
</tr>
<tr>
<td>International Normalized Ratio (INR)</td>
<td>2.0–3.0 for pts. on warfarin therapy; 2.5–3.5 for pts. with mech. prosthetic heart valves</td>
<td></td>
</tr>
<tr>
<td>Plasma Thrombin Time (PTT)</td>
<td>60–70 sec</td>
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</tr>
<tr>
<td>Activated Partial Thromboplastin Time (APTT)</td>
<td>35–45 sec</td>
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</tr>
<tr>
<td>Activated Clotting Time (ACT)</td>
<td>107 ± 13 sec</td>
<td></td>
</tr>
<tr>
<td>Fibrin Split Product (FSP)</td>
<td>&lt;10 μg/mL</td>
<td>&lt;10 mg/L</td>
</tr>
<tr>
<td>D-dimer</td>
<td>Neg. or &lt;250 μg/L</td>
<td></td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>200–400 mg/dL</td>
<td>2–4 g/L</td>
</tr>
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</table>

SI Units = International Units
<table>
<thead>
<tr>
<th>Test</th>
<th>Conventional Units (Reference Values*)</th>
<th>SI Units</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac Biomarkers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatine kinase (CK)</td>
<td>Males: 55–170 U/L Females: 30–135 U/L</td>
<td>0.94–2.89 μkat/L 0.51–2.3 μkat/L</td>
</tr>
<tr>
<td>CK isoenzymes:</td>
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<tr>
<td>CK-MM (muscle)</td>
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<td></td>
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<tr>
<td>CK-MB (myocardial)</td>
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<td></td>
</tr>
<tr>
<td>With AMI CK-MB:</td>
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<td></td>
</tr>
<tr>
<td>Onset: 4–6 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak: 12–24 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration: 2 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>95–100%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Troponin I</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With AMI:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset: 4–6 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak: 10–24 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration: 7–10 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–0.2 ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Myoglobin</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With AMI:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset: 2–4 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak: 8–12 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration: 24–30 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males: 20–90 ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females: 10–75 ng/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Other Cardiac Tests</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High sensitivity C-reactive Protein (hs-CRP)</td>
<td>Low: &lt;1.0 mg/L Average: 1.0–3.0 mg/L High: &gt;3.0 mg/L</td>
<td></td>
</tr>
<tr>
<td>B-type natiuretic peptide (BNP)</td>
<td>&lt;100 pg/mL</td>
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</tr>
</tbody>
</table>

SI Units = International Units

*Reference Values vary by regional laboratory techniques and methods.*
References

ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972
References

ANATOMY AND PHYSIOLOGY


ADVANCED NONINVASIVE MONITORING


REFERENCES


BASIC MONITORING

Pressure Monitoring


Central Venous Access


ADVANCED MINIMALLY-INVASIVE MONITORING


Biais M, Nouette-Gaulain K, Quinart A, Roullet S, Revel P, Sztark F. Uncalibrated stroke volume variations are able to predict the hemodynamic effects of positive end-expiratory pressure in patients with acute lung injury or acute respiratory distress syndrome after liver transplantation. Anesthesiology 2009; 111:855-862.


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PERIOPERATIVE GOAL-DIRECTED THERAPY


QUICK REFERENCE SECTION


