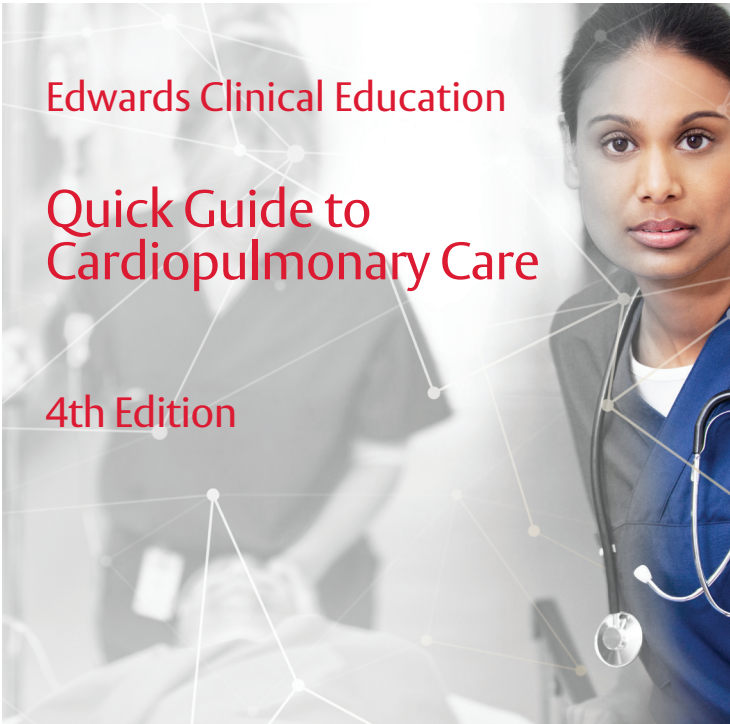

Edwards Clinical Education

Quick Guide to Cardiopulmonary Care

4th Edition

Edwards

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Quick Guide to Cardiopulmonary Care

4th Edition Editors

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Quick Guide to Cardiopulmonary Care

Pertinent clinical information dedicated to the critical care clinician

The intent of the Quick Guide is to provide a ready reference for hemodynamic monitoring and oxygenation assessment of the critically ill.

Critically ill patients are being cared for in many parts of the hospital beyond the intensive care unit. Minimally and noninvasive monitoring techniques have become part of routine assessment and care that can alert clinicians of changes in hemodynamic values and oxygenation status. Decision trees and algorithms using physiologic monitoring parameters have been published and are used in daily practice.

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 assist the clinician caring for critically ill patients.

Quick Guide to Cardiopulmonary Care
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Edwards Clinical Education

Anatomy and Physiology

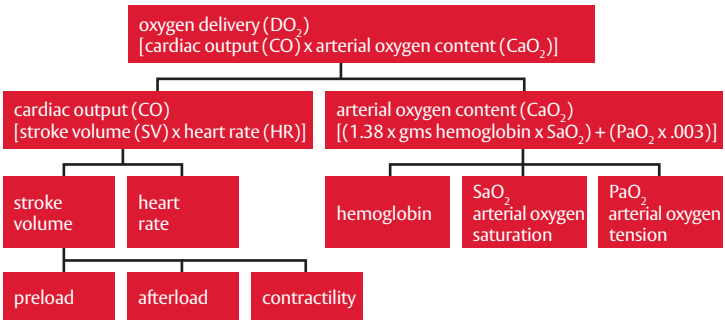
Anatomy and Physiology

Adequate oxygenation to the tissues is dependent upon oxygen delivery (DO_2) and the ability of the tissues to utilize the oxygen delivered at the cellular level; oxygen consumption (VO_2). Tissue hypoxia can occur when an imbalance exists between oxygen delivery and oxygen consumption. Insufficient oxygen delivery can be a result of poor pulmonary function, poor cardiac function or anemia.

Cardiopulmonary monitoring can assist clinicians when assessing critically ill patients at risk for tissue hypoxia related to decreased oxygen delivery. Continuous cardiopulmonary monitoring allows for prompt recognition with the goal for optimizing tissue oxygenation.

Oxygen Delivery ($DO_2 = CaO_2$)

DO_2 is the amount of oxygen delivered or transported to the tissues in one minute and is comprised of oxygen content and cardiac output. The adequacy of oxygen delivery is dependent upon appropriate pulmonary gas exchange, hemoglobin levels, sufficient oxygen saturation and cardiac output.



Oxygen content (CaO_2): amount of oxygen in the blood and can be calculated.
$(1.38 \times Hgb \times SaO_2) + (0.003 \times PaO_2)$

1.38: amount of O_2 that can combine with 1 gram of hemoglobin
0.003: solubility coefficient of O_2 in the plasma

$CaO_2 = (1.38 \times Hgb \times SaO_2) + (0.003 \times PaO_2)$ Normal 20.1 mL/dL†

Oxygen delivery (DO_2): amount of oxygen transported in the arterial blood.

Arterial oxygen delivery (DO_2): $CO \times CaO_2 \times 10$
5 L/min x 20.1 mL/dL x 10 = 1005 mL/min

† Assumes Hgb of 15gm/dL

Oxygen Consumption

Oxygen consumption refers to the amount of oxygen used by the tissues. 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 of oxygen returned on the venous side.

This is known as venous oxygen return (DvO_2).
$CvO_2 = (1.38 \times Hgb \times SvO_2) + (0.003 \times PvO_2)$
Normal 15.5 mL/dL
Venous oxygen return (DvO_2): $CO \times CvO_2 \times 10$
5 L/min x 15.5 mL/dL x 10 = 775 mL/min

oxygen consumption
oxygen consumption (VO_2) = oxygen delivery (DO_2) – venous oxygen return (DvO_2)

oxygen delivery (DO_2)
[cardiac output (CO) x arterial oxygen content (CaO_2)]
(CO) x (1.38 x 15 x SaO_2 + (PaO_2 x .003)
5 x 20.1 =
normal = 1005 mL O_2/min

—

venous oxygen return (DvO_2)
[cardiac output (CO) x venous oxygen content (CvO_2)]
(CO) x (1.38 x 15 x SvO_2) + (PvO_2 x .003)
5 x 15.5 =
normal = 775 mL O_2/min

$VO_2 - CO \times (CaO_2 - CvO_2) \times 10$
$VO_2 = CO \times Hgb \times 13.8 \times (SaO_2 - SvO_2)$
$VO_2 = 5 \times 15 \times 13.8 \times (.99 - .75)$
normal = 200 - 250 mL O_2/min

Oxygen consumption (VO_2)
Arterial oxygen transport – Venous oxygen transport
$VO_2 = (CO \times CaO_2) - (CO \times CvO_2)$
$= CO (CaO_2 - CvO_2)$
$= CO [(SaO_2 \times Hgb \times 13.8) - (SvO_2 \times Hgb \times 13.8)]$
$= CO \times Hgb \times 13.8 \times (SaO_2 - SvO_2)$
Normals: 200 – 250 mL/min 120 – 160 mL/min/m^2

Note: 13.8 = 1.38 x 10 (10 is used to convert dL to L)

Factors Affecting DO_2 and VO_2*

Decreased DO_2	Increased VO_2 or O_2ER
↓ Cardiac Output (CO) ↓ Hemoglobin/Anemia ↓ SaO_2 / Hypoxia	Seizures Shivering Pain Hyperthermia ↑ Work of breathing
Increased DO_2	**Decreased VO_2 or O_2ER**
↑ FiO_2 ↑ Cardiac Output (CO) Transfusion	Hypothermia Anesthesia ↓ Pain / Pain control measures

* Not an exhaustive list of factors

Diagnoses related to Increase in VO_2 or O_2ER	
Surgical trauma	(10-30%)
Severe sepsis	(50-100%)
Head injury	(89-138%)
Severe burns	(100%)

* Not an exhaustive list of related diagnoses

Other Assessment Parameters for Oxygen Utilization

Arterial-venous oxygen difference
$C(a\text{-}v)O_2$: normally 5mL/dL

Oxygen extraction ratio
O_2ER: normally 20-30%
O_2ER: $CaO_2 - CvO_2 / CaO_2 \times 100$
$CaO_2 = 20$
$CvO_2 = 15$
$O_2ER = 20 - 15 / 20 \times 100 = 25\%$

CO and SvO_2 correlations
Cardiac output (CO) can be measured by using the Oxygen Fick Principle that takes into account oxygen consumption (VO_2) and arteriovenous oxygen difference $C(a\text{-}v)O_2$. SvO_2 reflects the balance between oxygen delivery and oxygen consumption based upon the following equations.
$CO = VO_2 / C(a-v)O_2$
$VO_2 = C(a-v)O_2 \times CO \times 10$
$C(a-v)O_2 = VO_2 / (CO \times 10)$
$S(a-v)O_2 = VO_2 / (CO \times 10)$

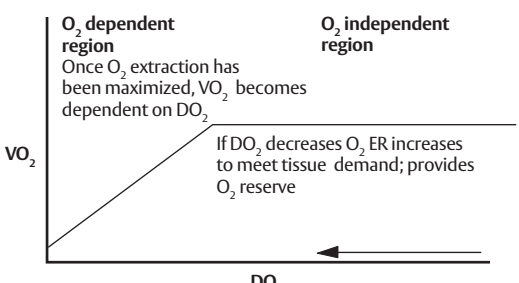
The Fick equation can be rearranged to reflect the balance between oxygen delivery and consumption:
When $SaO_2 = 1.0$, then $SvO_2 = CvO_2 / CaO_2$
$SvO_2 = 1 - [VO_2 / (CO \times 10 \times CaO_2)]$

As a result, SvO_2 reflects changes in oxygen extraction and the balance between DO_2 and VO_2.

VO_2/DO_2 Relationships

Oxygen consumption (VO_2) is determined by the metabolic demands at the cellular level. Under normal conditions, oxygen delivery (DO_2) is approximately four times the amount of oxygen consumed. Normally VO_2 is about 25% of DO_2. This has been referred to as delivery-independent VO_2; meaning consumption is independent of delivery. If oxygen delivery decreases or cellular oxygen demands increase, a larger percentage of oxygen can be extracted (represented by O_2ER) from delivery to meet metabolic demands. O_2 extraction ratio (O_2ER) is the ratio of oxygen consumption to oxygen delivery. When oxygen consumption requirements equals or exceeds oxygen delivery, oxygen consumption becomes delivery-dependent; in which supply is inadequate to meet metabolic demands and anaerobic metabolism may take place.

Normal relation



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 heart

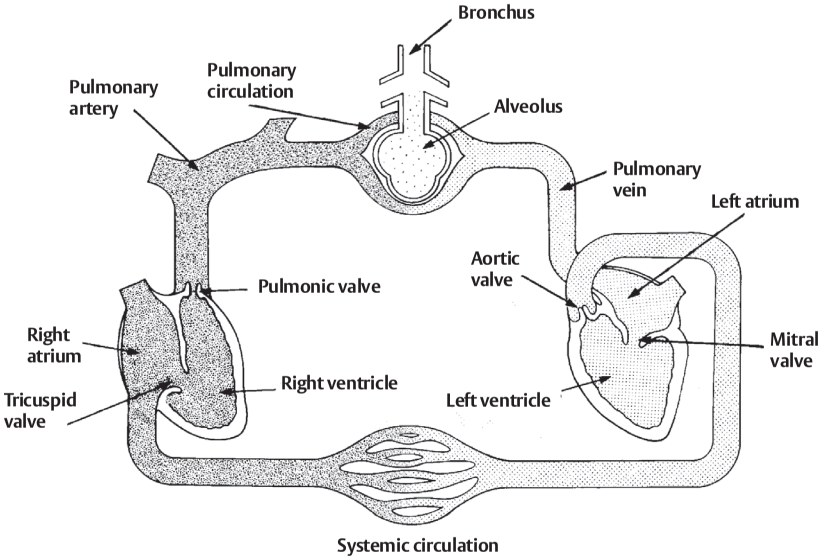
Receives deoxygenated blood

Low pressure system

Volume pump

Right ventricle thin and crescent shape

Coronary perfusion during systole and diastole



Left heart

Receives oxygenated blood

High pressure system

Pressure pump

Left ventricle thick and conical shape

Coronary perfusion during diastole

Coronary Arteries and Veins

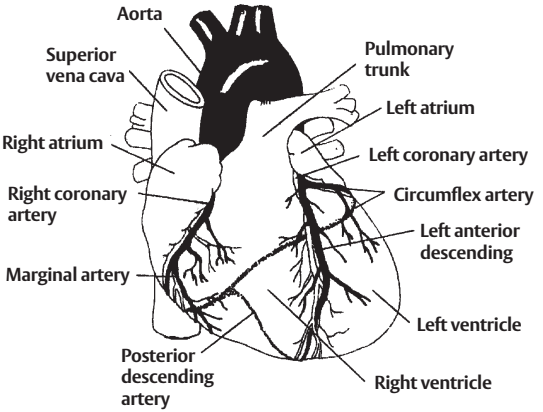
The two major branches of the coronary arteries arise from each side of the aortic root.

Major branches	Areas supplied
Right coronary artery (RCA)	Sinoatrial (SA) node, atrioventricular (AV) Node, bundle of His Right atrium (RA), right ventricle (RV) free wall Portion of interventricular septum (IVS)
Posterior descending branch (Provided by RCA)	Portion of interventricular septum (IVS) Diaphragmatic aspect of left ventricle (LV)
Left main coronary artery bifurcates	
Left anterior descending (LAD)	Left anterior wall Anterior portion of interventricular septum (IVS) Portion of right ventricle (RV)
Left circumflex (Provides posterior descending branch)	Sinoatrial (SA) node, left atrium (LA), atrioventricular (AV) node Lateral and posterior wall of left ventricle (LV)

Coronary veins	Location drains into
Thebesian veins	Directly into right and left ventricles
Great cardiac vein	Coronary sinus in the right atrium (RA)
Anterior cardiac veins	Right atrium (RA)

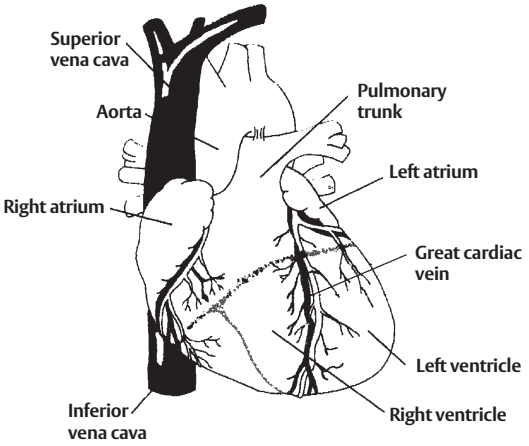
Coronary arteries

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



Coronary veins

Blood is drained by branches of the cardiac veins.

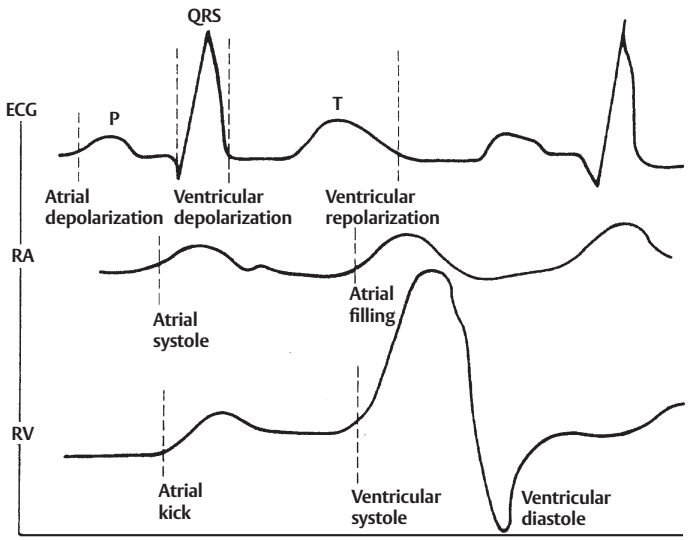


Cardiac Cycle: Electrical Correlation to Mechanical

The electrical cardiac cycle occurs prior to the mechanical cardiac cycle. Atrial depolarization begins from the sinoatrial (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.

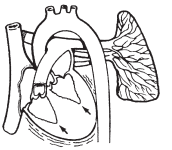
Electrical – mechanical cardiac cycle



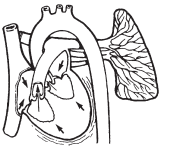
Mechanical Cardiac Cycle Phases

Systole

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



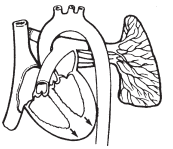
2. **Rapid ventricular ejection**
   Aortic valve opens
   Occurs during ST segment
   2/3 or more of blood volume ejected



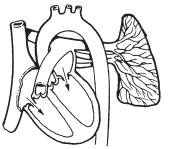
3. **Reduced ventricular ejection**
   Occurs during "T" wave
   Atria are in diastole
   Produces "v" wave in atrial tracing

Diastole

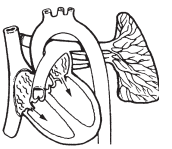
1. **Isovolumetric relaxation**
   Follows "T" wave
   All valves closed
   Ventricular pressure declines further
   LV pressure dips below LA pressure



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



Cardiac Output Definition

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

Cardiac output = heart rate x stroke volume
Heart rate = beats/min
Normal heart rate range: 60 – 100 BPM
Stroke volume = mL/beat; amount of blood ejected from ventricle in one beat
Normal stroke volume: 60 – 100 mL/beat
Stroke volume index = mL/m^2/beat; amount of blood ejected per beat based upon BSA
Normal SVI = 33 - 47 mL/m^2/beat
Normal cardiac output: 4 – 8 L/min
BSA = Body Surface Area (m^2)
Cardiac Index = CO/BSA
Normal cardiac index : 2.5 – 4 $L/min/m^2$

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].
SV = EDV – ESV

Left Ventricular Stroke Work Index	50 – 62 g/m^2/beat
LVSWI = SVI (MAP – PAOP) x 0.0136	
Right Ventricular Stroke Work Index	5 – 10 g/m^2/beat
RVSWI = SVI (PA mean – CVP) x 0.0136	

Ejection Fraction (EF) is measured as the percentage of volume of blood that is ejected from the ventricle at the end of systole. It is the ratio between SV and EDV. Normal EF for the left ventricle is ≥50% and normal EF for the right ventricle is ≥40%.
EF = (SV / EDV) x 100

Determinants of cardiac output

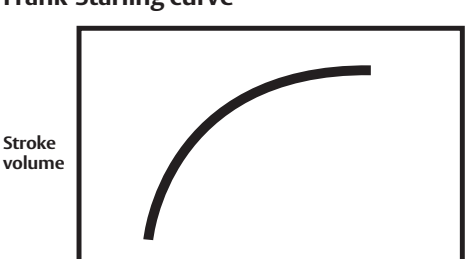
Preload Definition and Measurements

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. Pulmonary artery occlusion pressure (PAOP) or wedge pressure and left atrial pressures (LAP) have been used to evaluate left ventricular preload. Right atrial pressure (RAP) and central venous pressure (CVP) have been used to assess right ventricular preload. Volumetric parameters like right ventricular end-diastolic volume (RVEDV) and left ventricular end-diastolic volume (LVEDV) 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 identified the relationship between myocardial fiber length and force of contraction. The more the diastolic volume or fiber stretch at the end of diastole, the stronger the next contraction during systole and the greater the stroke volume.

Frank-Starling curve



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.

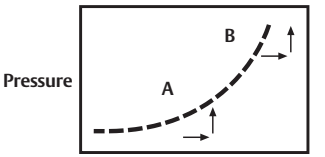
Effects of ventricular compliance

Normal compliance

Pressure/volume relationship is curvilinear:

a: Large increase in volume = small increase in pressure

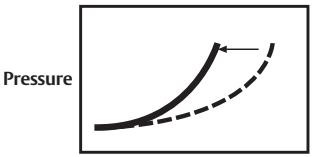
b: Small increase in volume = large increase in pressure



Decreased compliance

Stiffer, less elastic ventricle

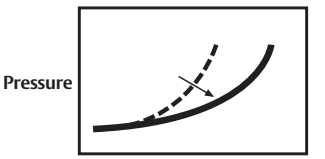
Ischemia
Increased afterload
Hypertension
Inotropes
Restrictive cardiomyopathies
Increased intrathoracic pressure
Increased abdominal pressure



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 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}

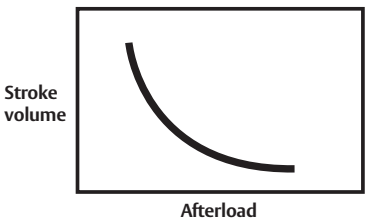
$$PVR = \frac{MPAP - PAOP}{CO} \times 80$$

Systemic vascular resistance (SVR): 800-1200 dynes-sec/cm^{-5}

$$SVR = \frac{MAP - RAP}{CO} \times 80$$

Afterload has an inverse relationship to stroke volume. As resistance to ejection increases (e.g. increased SVR or PVR), an increase in myocardial oxygen consumption also occurs.

Ventricular function



Contractility Definition and Measurements

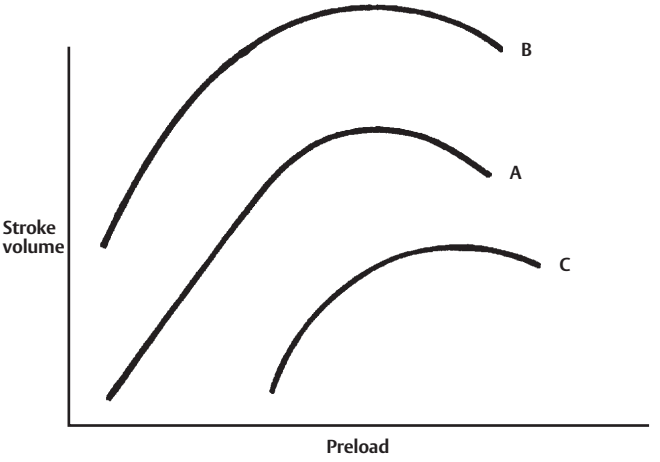
Inotropy or contractility refers to the inherent property of the myocardial muscle fibers to shorten independent of preload and/or afterload. Stroke volume is determined by myocardial fiber shortening and ventricular compliance.

Contractility changes can be plotted on a curve known as ventricular function curves. Ventricular function curves represent the Frank-Starling mechanism of the heart.

Clinical assessment of contractility include determinants of preload and afterload, and can be inferred through ventricular function curves.

Ventricular function curves

Ventricular function can be represented by plots on the ventricular curves. The performance characteristics of cardiac contractility from these curves can be implied depending upon the state of preload, afterload, or ventricular compliance.



A: Normal contractility
B: Increased contractility
C: Decreased contractility

Acid Base Balance

Arterial blood gas analysis
Values obtained from blood gas analysis determine the disorder (acidemia or alkalemia) present.

Definitions:

pH: Indicates the acidity or alkalinity of the blood. Acid-base disorders may have a metabolic or respiratory component

Acidemia: An acid condition of the blood with increased hydrogen ions and pH < 7.35

Alkalemia: An alkalosis (base) condition of the blood with decreased hydrogen ions and pH > 7.45

PCO_2: Respiratory component

Normal 35 – 45 mmHg
Hypoventilation > 45 mmHg
Hyperventilation < 35 mmHg

HCO_3: 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

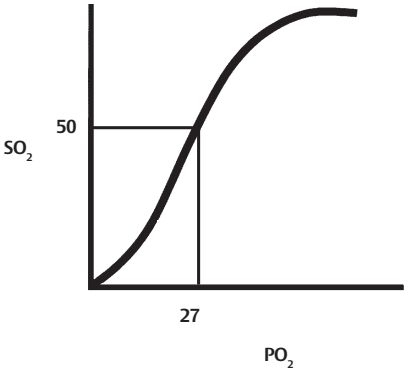
Normal blood gas values

Component	Arterial	Venous
pH	7.40 (7.35 – 7.45)	7.36 (7.31 – 7.41)
PO_2 (mmHg)	80 – 100	35 – 45
SO_2 (%)	≥ 95	60 – 80
PCO_2 (mmHg)	35 – 45	42 – 55
HCO_3 (mEq/L)	22 – 26	
Base excess/deficit	-2 – +2	-2 – +2

Oxyhemoglobin Dissociation Curve

The oxyhemoglobin dissociation curve graphically illustrates the relationship that exists between the partial pressure (PO_2) of oxygen and oxygen saturation (SO_2) which is the percentage of hemoglobin saturated with oxygen. Under normal conditions, the point at which the hemoglobin is 50% saturated with oxygen is called the P50. At a PO_2 of 27 mmHg, hemoglobin is 50% saturated.

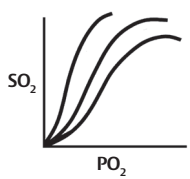
Normal oxyhemoglobin dissociation curve



Factors shifting oxyhemoglobin dissociation curve

The affinity of oxygen for hemoglobin depends upon pH, 2,3 DPG and temperature. A shift to the right of the oxyhemglobin dissociation curve represents a decrease affinity or an increase release of oxygen from the hemoglobin to the dissolved state in order to meet an increase in cellular O_2 demands. A leftward shift of the oxyhemglobin dissociation curve represents a greater affinity of oxygen or a decrease release of oxygen from the hemoglobin. This may indicate a decrease in O_2 demands, but may also indicate an inability of oxygen to be released from the hemoglobin in certain pathophysiologic states (i.e., sepsis), leading to tissue hypoxia despite normal or even high saturation values.

Leftward shift:
Increased affinity O_2 to Hgb, ↑ pH, Alkalosis, Hypothermia, ↓ 2-3 DPG



Rightward shift:
Decreased affinity O_2 to Hgb, ↓ pH, Acidosis, Hyperthermia, ↑ 2-3 DPG

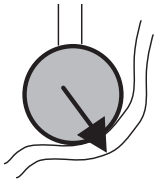
Pulmonary gas exchange equations

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: PAO_2 is the alveolar PO_2 and is calculated based on barometric pressure. Barometric pressure (PB) is based on atmospheric pressure and FiO_2.

$PAO_2 = [(PB - PH_2O) \times FiO_2] - PaCO_2 / 0.8$

Alveolar–arterial oxygen gradient
(A–a Gradient or P(A–a)O_2)



PB: Atmospheric pressure at sea level: 760
PH_2O: Pressure of water: 47 mmHg
FiO_2: Fraction of inspired oxygen
$PaCO_2$: Partial pressure of CO_2
0.8: Respiratory quotient (VCO_2 / VO_2)

P(A-a)O_2: Assesses the amount of diffusion across the alveolar capillary unit.

$PAO_2 = [(PB - PH_2O) \times FiO_2] - PaCO_2 \times [FiO_2 + (1 - FiO_2) / 0.8]$
PAO_2 is measured from the arterial blood gas
Normal: < 15 mmHg on room air

Abnormal: > 15 mmHg on room air indicative of lung disease

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 (Qs represents shunt and Qt represents flow). 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 patients.

A pathologic shunt occurs when there are either collapsed alveolar units (i.e., atelectasis) or other conditions where the venous blood is not oxygenated and essentially bypasses the lung.

Qs/Qt is calculated as follows:

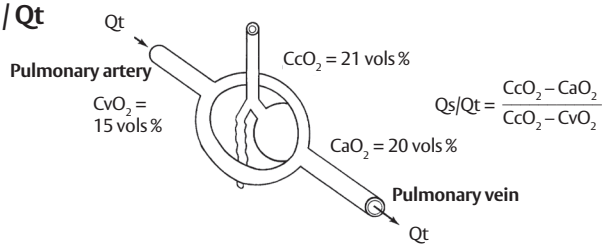
$$Qs/Qt = \frac{CcO_2 - CaO_2}{CcO_2 - CvO_2}$$

CcO_2 = Capillary oxygen content
$(1.38 \times Hgb \times 1) + (PAO_2 \times 0.003)$

CaO_2 = Arterial oxygen content
$(1.38 \times Hgb \times SaO_2) + (PaO_2 \times 0.003)$

CvO_2 = Venous oxygen content
$(1.38 \times Hgb \times SvO_2) + (PvO_2 \times 0.003)$

Qs / Qt



Idealized version of the heart and lungs depicting shunt: Depicts a shunt of deoxygenated blood from the right heart that remains deoxygenated as it flows past atelectatic lung that does not participate in gas exchange.

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 $SaO_2 < 100\%$
2. Poor agreement with Qs/Qt if $PaO_2 > 99$ mmHg
3. Good correlation when Qs/Qt > 15%

Equation derivations

$$Qs/Qt = \frac{100 \times [(1.38 \times Hgb) + (0.003 \times PAO_2) - CaO_2]}{[(1.38 \times Hgb) + (0.003 \times PAO_2) - CvO_2]}$$

$$VQI = \frac{100 \times [1.38 \times Hgb \times (1 - SaO_2 / 100) + (0.003 \times PAO_2)]}{[(1.38 \times Hgb) + (0.003 \times PAO_2) - CvO_2]}$$

Dual oximetry, simplifies the shunt equation

$$VQI = \frac{SAO_2 - SaO_2 = 1 - SaO_2}{SAO_2 - SvO_2 = 1 - SvO_2} \quad \text{or} \quad \frac{1 - SpO_2}{1 - SvO_2}$$

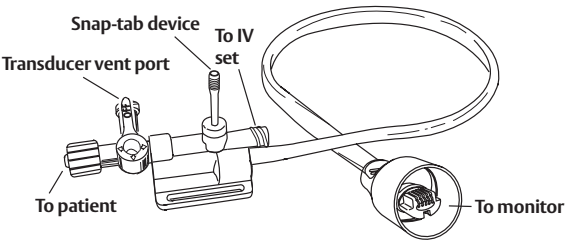
Edwards Clinical Education

Basic Monitoring

Physiologic Pressure Monitoring

Pressure monitoring is a basic tool for clinicians 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.

TruWave disposable pressure tranducer components



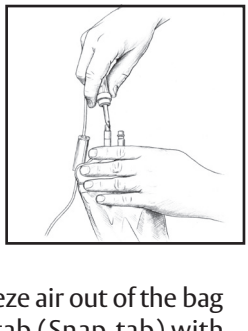
Components of a physiologic pressure measurement system

- Invasive catheter
- Edwards transducer kit
  - Non-compliant pressure tubing
  - Stopcocks
  - Transducer housing
  - 3 mL/hr flush device
  - Cable connection
  - Fluid administration set
- Flush solution (e.g., saline) (500 or 1000 mL) (Heparin per institutional policy)
- Pressure infusion bag (appropriately sized for flush solution bag)
- Reusable pressure cable specific to TruWave transducer
- Bedside physiologic monitor

Correct set-up, calibration, and maintenance based upon manufacturer's instructions of a physiologic pressure transducer system is crucial in obtaining the most accurate pressure readings.

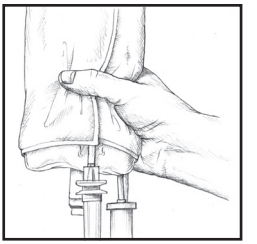
Setting up a physiologic pressure measurement system for intravascular monitoring

1. Wash hands.
2. Open TruWave disposable pressure transducer packaging and inspect contents. Ensure that all connections are tight.
3. Remove the TruWave transducer from its packaging and insert into a TruClip holder 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. Remove remaining air from the flush bag per institutional policy or while keeping IV bag inverted, gently squeeze air out of the bag with one hand while pulling flush tab (Snap-tab) with the other hand until air is emptied from IV bag and drip chamber is filled to at least 1/2 full.

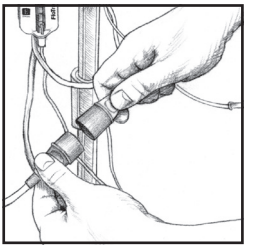


5. Insert flush bag into pressure infuser bag (**do not inflate**) and hang on IV pole approximately 2 feet (60 cm) above the transducer.

6. With gravity only (no pressure in pressure bag), flush TruWave transducer holding pressure tubing in upright position. Flushing under pressure creates turbulence and increased occurrence of bubbles.



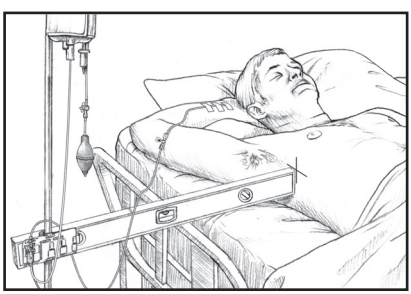
7. Pressurize the pressure bag until it reaches 300 mmHg.
8. Fast-flush transducer tubing while tapping on tubing and stopcocks to remove any residual bubbles.
9. After all air has been removed and the entire system has been completely flushed, replace all vented caps on the sides of the stopcocks with non-vented caps.
10. Connect non-disposable pressure cable that is compatible with bedside monitor to disposable pressure transducer and bedside monitor.



11. Connect tubing to arterial or venous catheter, and then aspirate and flush system to ensure catheter is intra-vascular and remove residual bubbles.
12. Level the stopcock just above the TruWave transducer to the phlebostatic axis.
13. Open the stopcock to atmospheric air. Zero pressure per bedside monitor's instructions for use.
14. Inspect pressure trace on bedside monitoring screen to confirm appropriate pressure scale, alarm settings, pressure label, color coding, and a physiologic waveform is present.

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. Leveling by visual estimation is not recommended.



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.

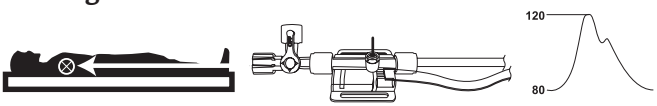
Maintaining physiologic pressure transducer system

- **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 should be performed per institutional policy
- **Check pressure infuser bag**
  Maintain a pressure of 300 mmHg to ensure constant flow of flush solution and system fidelity
- **Check flush bag volume**
  Change flush bag when < ¼ full to ensure constant flow of flush solution and system fidelity
- **Check system integrity**
  Ensure 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, or per institutional policy
- **Changing system frequency**
  Centers for Disease Control recommends changing disposable transducer flush systems, tubing and flush solution at 96 hour intervals

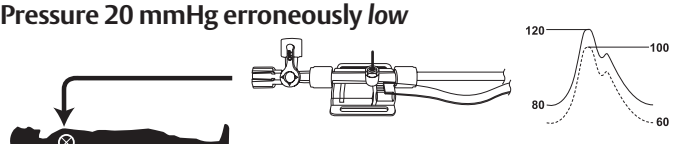
Impact of improper leveling on pressure readings
Intravascular pressure readings may have error introduced if alignment with the phlebostatic axis is not maintained. The amount of error introduced is dependent upon the degree of offset.

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.

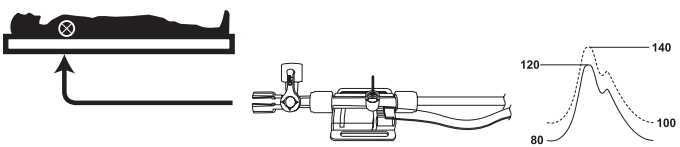
Heart aligned with transducer =
0 mmHg error



Heart 10" (25 cm) *lower* than transducer =
Pressure 20 mmHg erroneously *low*



Heart 10" (25 cm) *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.

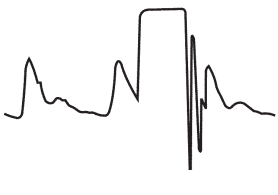
An underdamped physiologic pressure system will result in an overestimation of systolic pressure and an under estimation of diastolic pressure.

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

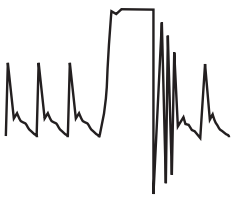
Determining dynamic response
Optimal pressure monitoring requires a pressure system that accurately reproduces the physiologic signals applied to it. A simple evaluation of dynamic response can be obtained by performing a square wave test and by observing the resultant oscillations. In order to perform 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

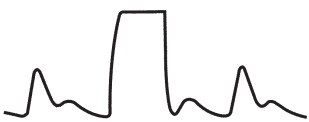
Optimally damped
1.5 – 2 oscillations before returning to tracing. Values obtained are accurate.



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



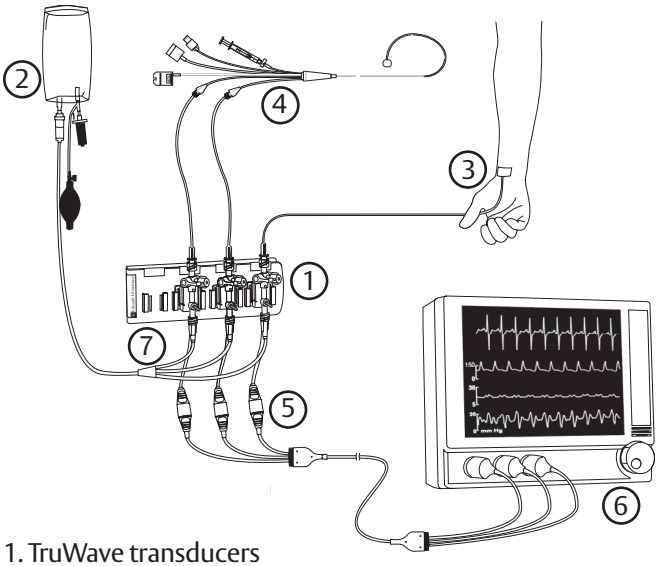
Overdamped
< 1.5 oscillations. Underestimation of systolic pressures, diastolic may not be affected.



Pressure monitoring systems

This schematic identifies the components of a standard pressure monitoring system. The Edwards oximetry central venous or 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 flush solution (at 300 mmHg). An integral flush device with a restrictor limits the flow rate to approximately 3 mL/hour for adults. Typically normal saline is used as the flush solution or per institutional protocol.

Pressure system shown with Swan-Ganz catheter



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

Measuring technique

Hydrostatic zero reference

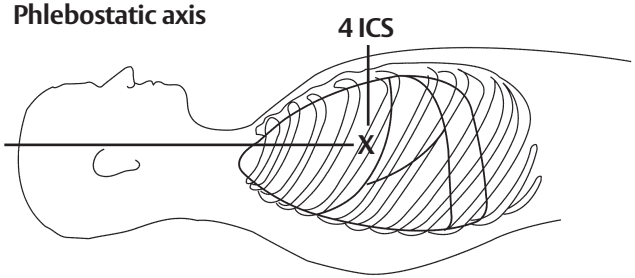
To obtain accurate pressure measurements, the level of the air-fluid interface must be aligned with the chamber or vessel being measured.

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.

Phlebostatic axis



Intra-arterial monitoring
Components of the arterial pulse

Peak systolic pressure: begins with opening of the 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

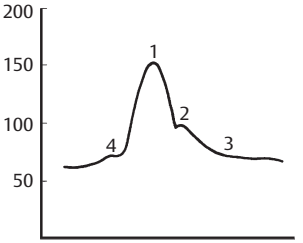
Diastolic pressure: relates to the level of vessel recoil or amount of vasoconstriction in the arterial system. May be termed the descending limb

Anacrotic notch: the anacrotic notch will occur before the opening of the aortic valve if present. (May be present in aortic insufficiency)

Pulse pressure: difference between systolic and diastolic pressure

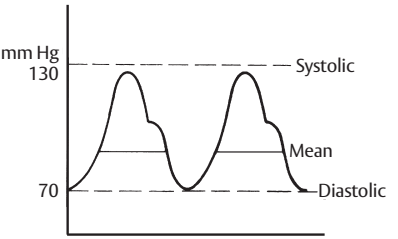
Mean arterial pressure (MAP): average pressure in the arterial system during a complete cardiac cycle. Systole requires one-third of the cardiac cycle, diastole normally during two-thirds. This timing relationship is reflected in the equation for calculating MAP. MAP can be written as MAP = DP + 1/3PP or as MAP = (SP +2(DP))/3

Components of arterial pulse



1. Peak systolic pressure
2. Dicrotic notch
3. Diastolic pressure
4. Anacrotic notch

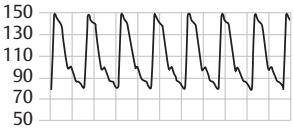
Mean arterial pressure



Bedside physiologic monitors use various algorithms to incorporate the area under the curve for determining the mean pressure.

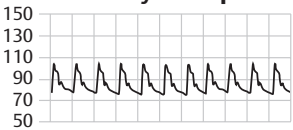
Abnormal arterial pressure waveforms

Elevated systolic pressure*



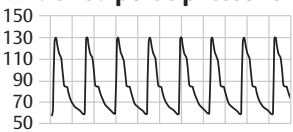
Systemic hypertension
Arteriosclerosis
Aortic insufficiency

Decreased systolic pressure*



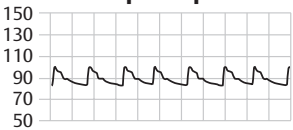
Aortic stenosis
Heart failure
Shock

Widened pulse pressure*



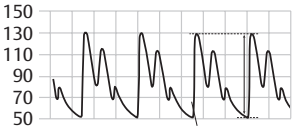
Vasodilation
Aortic insufficiency
Shock

Narrowed pulse pressure*



Cardiac tamponade
Cardiogenic shock

Pulsus bisferiens*



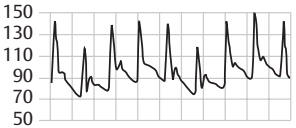
Aortic insufficiency
Obstructive hypertrophic cardiomyopathy

*note: y axis represents mmHg and x axis represents time

Note: this is not an exhaustive list of causes

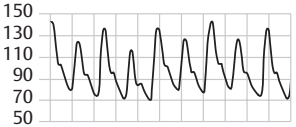
Abnormal arterial pressure waveforms (continued)

Pulsus paradoxus*



Cardiac tamponade
Pulmonary embolis

Pulsus alternans*



Congestive heart failure
Cardiomyopathy cardiac dysrhythmia

*note: y axis represents mmHg and x axis represents time

Note: this is not an exhaustive list of causes

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 nontunneled / 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 utilized 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, thus 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.

General applications of central venous access devices

- Fluid administration – for example, in cases of, or at high risk of, high blood loss
  - Cardiovascular and vascular surgery
  - Heart failure
  - Shock
  - Respiratory failure
  - Severe burns
  - Sepsis
  - Multisystem organ dysfunction
  - Head injury
  - Trauma
  - Acute respiratory distress system
  - Mechanical ventilation (adjustment of settings, weaning)
- 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
- Measurement of oxygen saturation levels in blood returning to the heart ($ScvO_2$)
- Monitoring and access for either pre- or post-pulmonary artery catheter insertion (same insertion site)

Relative contraindications may include patients with

- 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 pleural space collapsing lung): internal jugular IJ approach has a lower incidence of a pneumothorax than a subclavian approach. Patients with overinflated lungs (i.e., COPD or PEEP) may have an elevated risk of pneumothorax with a subclavian approach
- Hemorrhage from insertion site
- Hemothorax (blood in pleural space collapsing lung), secondary to artery puncture or laceration
- 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, extravascular space, line related infection.

Central venous catheter specifics

Lumens and functionality

- More than one lumen increases the functionality of the CVC
- 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
- Triple lumen 8.5 French (Fr) catheters have more functional ports, but usually have smaller lumens (i.e., 8.5 Fr 18/18/15 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, lumen sizes are 18 gauge and 15 gauge

8.5 Fr double lumen catheter cross section

8.5 Fr triple lumen catheter cross section

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)

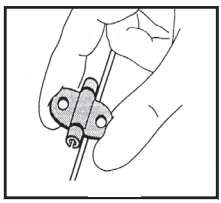
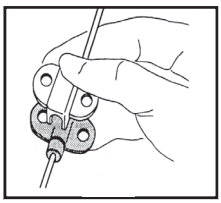
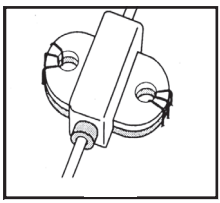
Flow rates are usually calculated with normal saline at a head height of 40" (101.6 cm).

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 but there exists excess catheter between the backform and site of insertion, a box-clamp can be utilized to anchor and secure the catheter at the insertion site.

Introducers as a central line

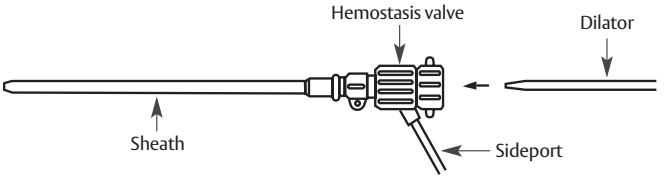
Sometimes an introducer is used for central venous access when rapid volume resuscitation is needed or is left in place following the removal of a pulmonary artery catheter. Components of the introducer system usually include:

- Flexible polyurethane sheath
- Guidewire and dilator
- Sideport
- Hemostasis valve

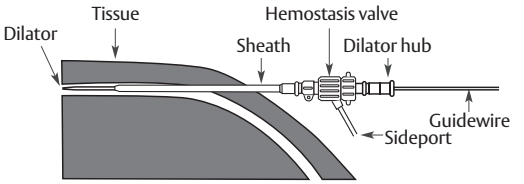
Fluids may be administered through the sideport, while the hemostasis valve prevents bleedback and / or air embolization.

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.

Automatic hemostasis



Tuohy-Borst value introducer (inserted)



Catheter tip placement
Central venous catheters should be inserted so that the tip is proximal to the right atrium (for rightsided 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.

A critical component 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 extra-pericardial 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 dysrhythmias and tamponade, the tip of a CVC should lie above this reflection and not in the right atrium.

Tips to ensure catheter tip is 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

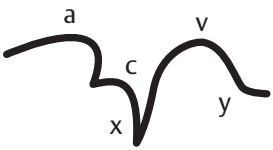
Central venous catheters are used to measure the pressure under which the blood is returned to the right atrium and to give an assessment of the intraventricular volume and right heart function. The CVP is a useful monitor if the factors affecting it are recognized and its limitations are understood. Serial measurement and changes in response to volume infusion are more useful than individual values. 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.

There are many factors that influence CVP values: cardiac performance, blood volume, vascular tone, intrinsic venous tone, increased intra-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 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, the c wave, resulting from closure of the tricuspid valve, may be seen. The x descent represents the continually decreasing atrial presure. 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.

Right atrium

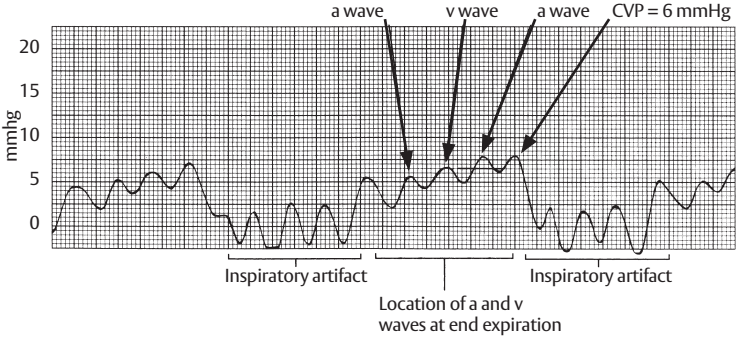


"a" = atrial contraction
"c" = closure of tricuspid valve
"v" = passive atrial filling
"x" = atrial diastole
"y" = atrial emptying

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



Reading CVP waveforms with spontaneous inspiratory artifact.

Edwards Clinical Education

Minimally-Invasive Monitoring

Minimally-Invasive Monitoring

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 (σ_{AP}) around the mean arterial pressure (MAP) value, measured in mmHg. 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 σ_{AP} is calculated.

Traditional: CO = HR * SV

FloTrac system:

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

Where χ = M (HR, σ_{AP}, C (P), BSA, MAP, μ_{1ap}, μ_{2ap}, μ_{3ap}, μ_{4ap}...)

APCO = arterial based pressure cardiac output

σ_{AP} = standard deviation of arterial pulse pressure in mmHg is proportional to pulse pressure

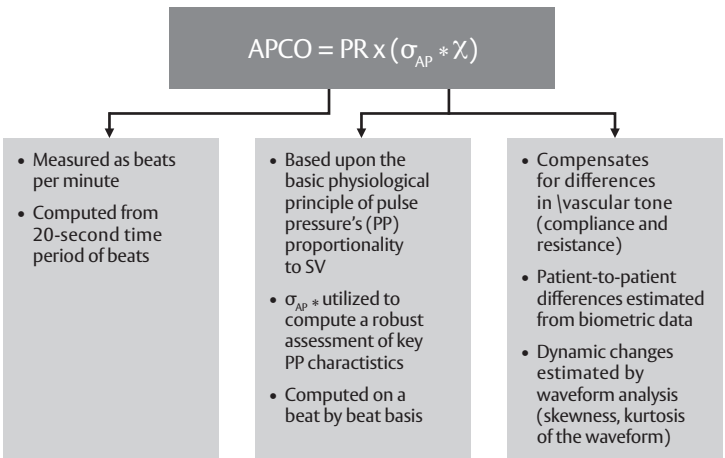
Khi (χ) = scaling multivariate parameter proportional to the effects of vascular tone on pulse pressure

M = multivariate polynomial equation

BSA = body surface area calculated by Dubois' equation

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.



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

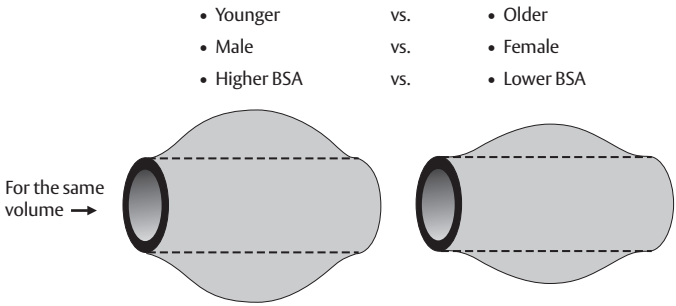
The conversion of standard deviation of arterial pressures (mmHg) into mL/beat is performed by multiplying it by a conversion factor known as *Khi* (χ). *Khi* is a multivariate polynomial equation which assesses the impact of the patient's ever-changing vascular tone on pulse pressure. *Khi* is calculated 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, whereas a decrease often represents a decrease in resistance
- **Standard deviation of arterial pressure (σ_{AP}):** Pulse pressure is proportional to σ_{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 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:

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

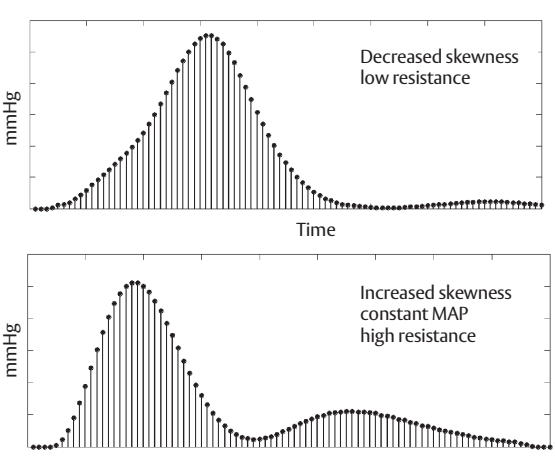
L = estimated aortic length
A_{max} = maximal aortic cross-sectional area
P = arterial pressure
P_0 = pressure at which compliance reaches its maximum
P_1 = pressure where arterial compliance is half of maximum value

Additional measures of weight, height and the BSA were also found to correlate with vascular tone and were added to enhance the calculation of aortic compliance.



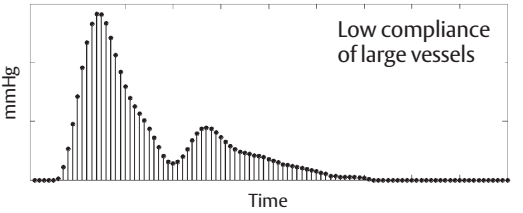
- Compliance inversely affects PP
- The algorithm compensates for the effects of compliance on PP base, age, gender, and BSA

- **Skewness (a measure for lack of symmetry, μ_{3ap}):** 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

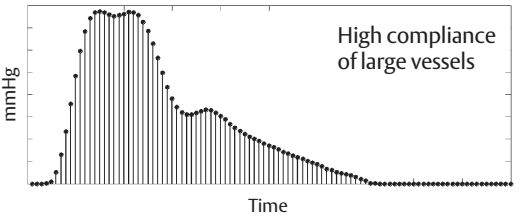


- **Kurtosis (a measure of how peaked or flat the pressure data points are distributed from normal distribution, $\mu_{4a}p$):** Pressure data with high kurtosis has the pressure rise and fall very quickly relative to the normal pulse pressure and can be directly associated with large vessel compliance

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



Khi **(χ) 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* (χ). *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) = σ_{AP} (mmHg)∗χ (mL/mmHg)

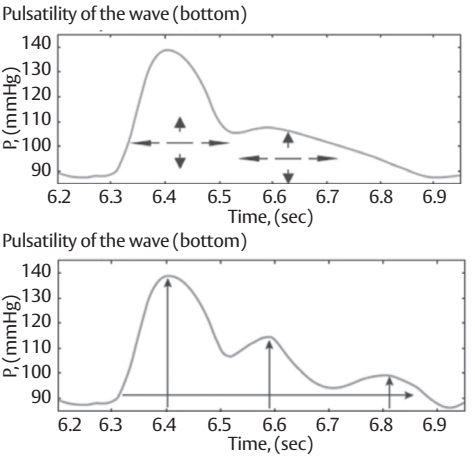
No manual calibration needed
The FloTrac system algorithm continuously adjusts for the patient's ever-changing vascular tone, and does not require manual calibration. As a component of the calibration, *Khi* auto corrects for changes in vascular tone through a complex waveform analysis. This feature 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 technique in pressure monitoring is important by priming with gravity, keeping pressure bag at 300 mmHg, ensure 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. Modifying FloTrac sensor kits may reduce dynamic response resulting in compromised hemodynamic monitoring.

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. The following high-risk surgical patients were added to the database including, but not limited to, gastrointestinal, esophageal, pancreaticoduodenectomy (whipple) and esophagectomy. The expanded patient database has informed the algorithm to recognize and adjust for more patient conditions.

Additional physiologically-based variables (see image on next page) 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.



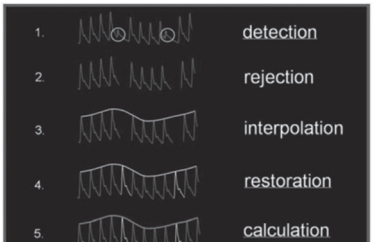
In addition to a broader database the FloTrac system 4.0 algorithm adjusts for rapid changes in pressure that occur during vasopressor administration through *Khi*-fast. *Khi*-fast is assessed every 20 seconds and is inversely affected by pressure. *Khi* continues to assess vascular tone every 60 seconds and *Khi*-fast every 20 seconds resulting in a more physiologic response to changes in resistance.

The SVVxtra Algorithm

Expanding clinical use with the FloTrac system algorithm

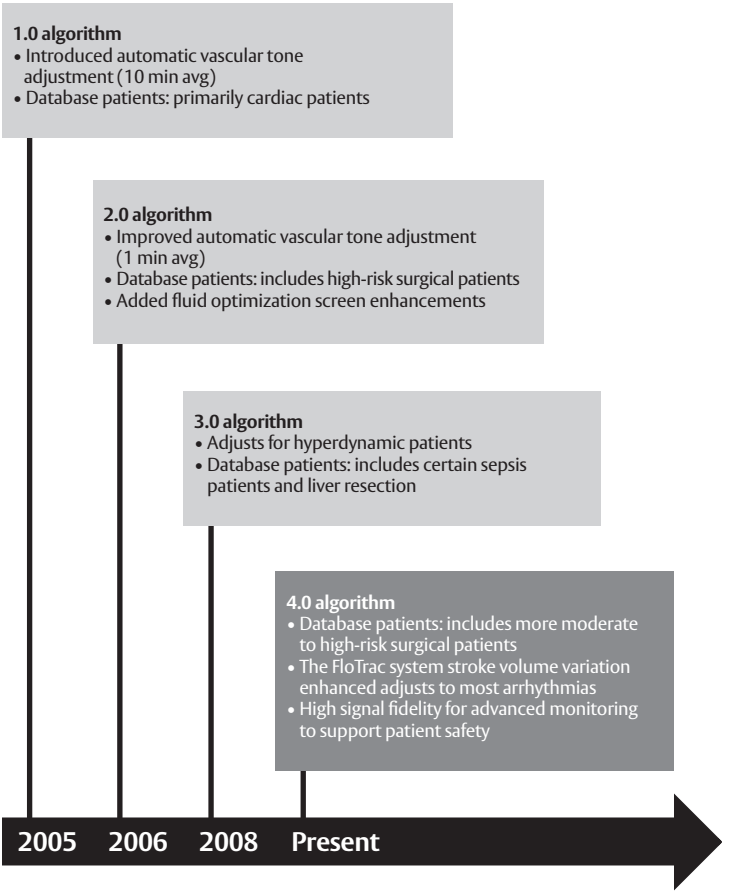
Historically dysrhythmias have been considered a contraindication for the application of SVV to guide fluid resuscitation. The SVVxtra algorithm within the FloTrac system algorithm, allows the clinician to continue to use SVV despite the presence of premature atrial or premature ventricular contractions. The SVVxtra algorithm 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:



If the frequency of dysrhythmias has exceeded the algorithms ability to filter these dysrhythmias then a “yellow heart” icon will appear.

FloTrac system algorithm evolution

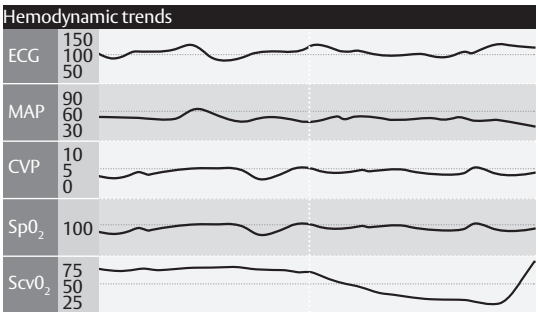


Venous Oximetry Physiology and Clinical Applications

Physiology and Venous Oximetry

Maintaining the balance between oxygen delivery (DO_2) and consumption (VO_2) to the tissues is essential for cellular homeostasis and preventing tissue hypoxia and subsequent organ failure. Traditional monitoring parameters (HR, blood pressure, CVP, and SpO_2) have been proven to be poor indicators of oxygen delivery. Moreover, patients have demonstrated continued signs of tissue hypoxia (increased lactate, low $ScvO_2$) even after they have been resuscitated to normalized vital signs.

$ScvO_2$ = early warning and prevention

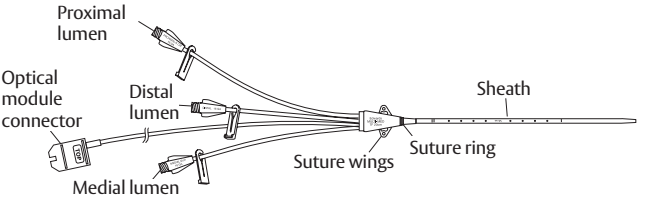


Traditional monitoring parameters failed to alert clinicians to cardiac tamponade in this case

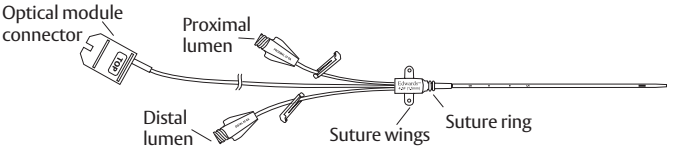
Continuous venous oximetry is a sensitive real-time indicator of the balance between oxygen delivery and consumption, which can be applied as a global (mixed venous oxygen saturation as SvO_2) or regional (central venous oxygen saturation as $ScvO_2$) indicator. SvO_2 is a global indicator of the balance between oxygen delivery and consumption. It is measured in the pulmonary artery before the blood is directed to the lungs to be oxygenated. SvO_2 consists of mixed blood from the superior vena cava (SVC), inferior vena cava (IVC) and the coronary sinus (CS) combined.

$ScvO_2$ is a regional reflection of the balance between oxygen delivery and oxygen consumption of the venous blood returning to the right side of the heart and measured in the SVC. Venous oximetry has been extensively studied and used clinically to monitor the balance between DO_2 and VO_2.

Edwards oximetry central venous catheter
8.5 Fr 20cm 18/18/15 gauge



PediaSat oximetry catheter
4.5 Fr 5cm 20/23 gauge



Difference between SvO_2 and $ScvO_2$
Since SvO_2 and $ScvO_2$ are affected by the same four factors (cardiac output, hemoglobin, oxygenation, and oxygen consumption), and trend together clinically, they are often considered clinically interchangeable.

SvO_2 is a global indicator of the balance between DO_2 and VO_2 as it is a reflection of all venous blood; IVC, SVC, and CS. $ScvO_2$ is a regional reflection (head and upper body) of that balance. Under normal conditions $ScvO_2$ is slightly lower than SvO_2. In hemodynamically unstable patients, this relationship changes with $ScvO_2$ being higher than SvO_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.

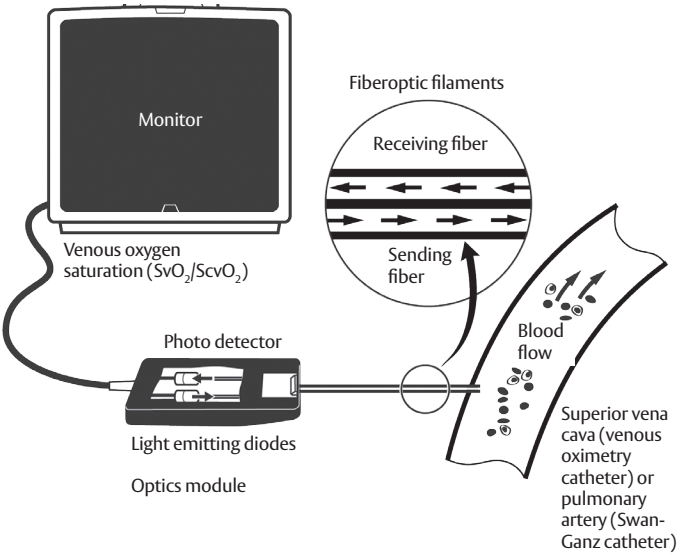
Global venous oximetry
SvO_2 – global mixed venous oximetry

Regional venous oximetry
$ScvO_2$ – head and upper extremities
$SpvO_2$ – peripheral venous oximetry

Organ specific venous oximetry
$SjvO_2$ – cranial jugular bulb oximetry
$ShvO_2$ – hepatic venous oximetry
$ScsO_2$ – coronary sinus oximetry

Continuous $ScvO_2$ 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 few seconds as a percent value.

Fiberoptic venous oximetry system



Accuracy of Edwards fiberoptic continuous $ScvO_2$ compared to co-oximetry

In a laboratory bench environment continuous fiberoptic venous oximetry monitoring accuracy is approximately ± 2% at oximetry range between 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 ($r^2 = 0.88$, $p < 0.001$). Difference of means (bias) was - 0.03% with a ± 4.41% precision per Liakopoulos et al.

Interference with $ScvO_2$ 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 the signal quality indicator (SQI) and readings if the tip is positioned against a vessel wall. Fluids infused through the distal lumen may also influence the SQI and readings (e.g., lipids such as TPN or propofol, and crystalloid infusions at high flow rates). Catheter kinking may also result in a high SQI.

Interpreting venous oximetry (SvO_2 and $ScvO_2$) values
Normal range values for SvO_2 are 60-80% and 60%-70% for $ScvO_2$. $ScvO_2$ usually runs slightly higher than SvO_2 in critically ill patients. Low oximetry readings usually indicate either low oxygen delivery (DO_2) or an increase in oxygen consumption (VO_2).

When change is significant
$ScvO_2$ and SvO_2 values are not static and may vary. These values may show significant changes with activities or interventions such as suctioning. When monitoring $ScvO_2$, 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 $ScvO_2$:

- Cardiac output (CO)
- Hemoglobin (HgB)
- Arterial oxygen saturation (SaO_2)
- Oxygen consumption (VO_2)

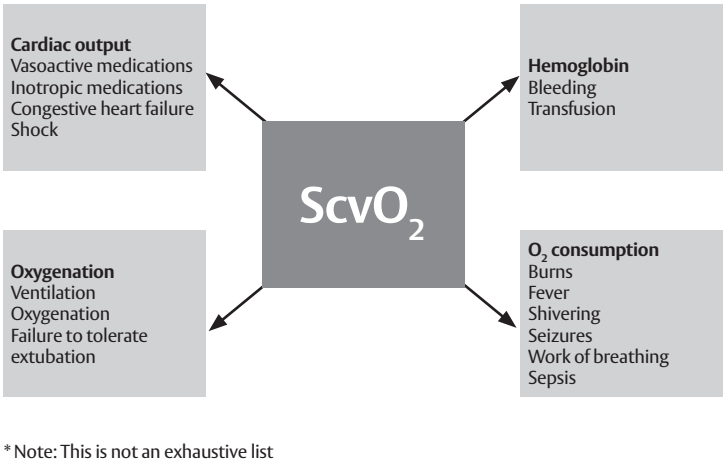
Note: CO, hemoglobin and SaO_2 are indicators of oxygen delivery (DO_2).

Clinical applications of $ScvO_2$

$ScvO_2$ and SvO_2 are affected by the same four factors and trend together more than 90% of the time.

The figure below provides examples of clinical situations where $ScvO_2$ monitoring may be helpful in identifying imbalances between DO_2 and VO_2.

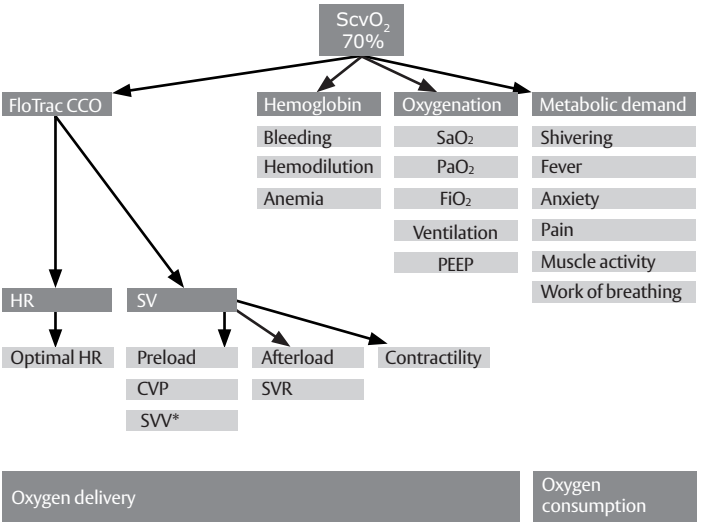
Clinical uses of $ScvO_2$ monitoring*



* Note: This is not an exhaustive list

$ScvO_2$ when used adjunctively with cardiac output monitoring, assists the clinician to determine the adequacy of oxygen delivery and to differentiate between issues of oxygen delivery vs. oxygen consumption.

Minimally-invasive algorithm
$DO_2 = CO \times CaO_2$



* SVV is an indicator of preload responsiveness.

VolumeView System

The VolumeView system expands the application of thermodilution technology through transpulmonary thermodilution (TPTD). It can 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.

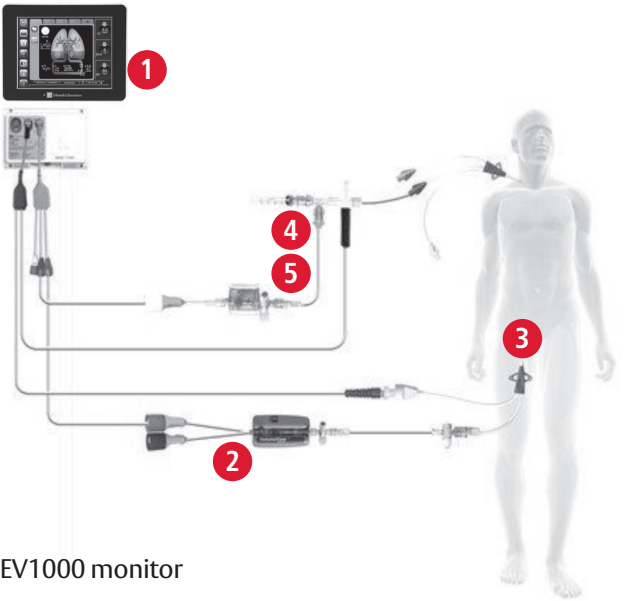
Transpulmonary thermodilution with the VolumeView system allows for the measurement of:

- 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

Additional capabilities:

- Extra-vascular lung water
- Pulmonary vascular permeability index

VolumeView system setup

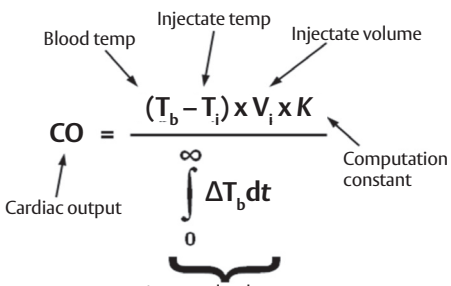


1. EV1000 monitor
2. VolumeView sensor
3. VolumeView femoral arterial catheter
4. VolumeView thermistor (CVC) manifold
5. TruWave pressure transducer

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 with each bolus. A computation constant is derived from an injectate solution of a known temperature, volume, and specific weight. The clinician enters the injectate volume into the monitor.

Stewart-Hamilton equation



After injection, the area under the transpulmonary thermodilution curve is analyzed 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 monitor by the clinician
- The area under the curve is calculated and analyzed 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), and intermittent systemic vascular resistance (iSVR) along with the globes which indicate where the values are within the target ranges.

Continuous cardiac output with VolumeView system
VolumeView system 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 system algorithm adjusts the calculated continuous cardiac output display by a percent change based on its proprietary algorithm against the measured cardiac output.

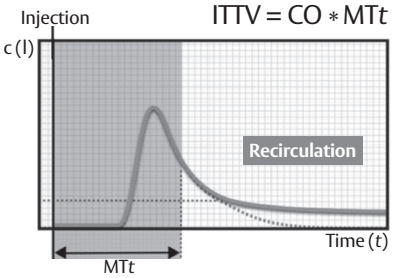
Calculating global end diastolic volume
The transpulmonary thermodilution measurement used to calculate cardiac output can also be used to calculate other physiologic parameters such as global end diastolic volume, global ejection fraction, and extra vascular lung water. These parameters are useful in evaluating and guiding volume resuscitation, ventricular performance, and changes in lung water as a result 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.

GEDI 680 – 800 mL/m²

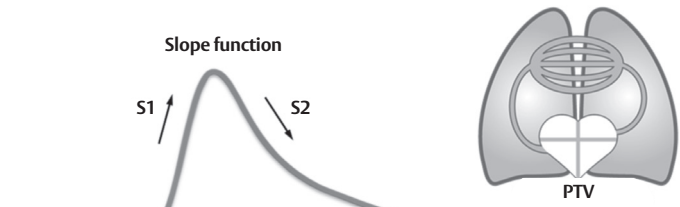
In order to calculate GEDV, intra-thoracic thermal volume (ITTV) is calculated by identifying the beginning of the injection cycle from a pressure spike measured in the central venous pressure from the VolumeView system CVC manifold. The VolumeView system's TPTD algorithm then identifies the peak indicator 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.

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.



ITTV = iCO x MTt

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.



Slope function * ITTV = GEDV

Global end diastolic volume (GEDV) is indexed against body surface area to give the global end diastolic volume index (GEDI).

Global ejection fraction
Global ejection fraction (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.

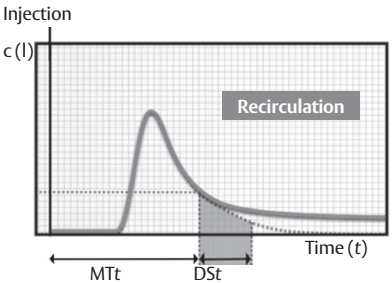
Stroke volume = cardiac output /pulse rate

Global ejection fraction = stroke volume x 4 / GEDV

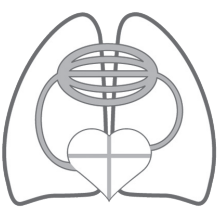
Global ejection fraction normal range is between 25-35%

Extra vascular lung water
VolumeView system can calculate the amount of extra vascular lung water (EVLW), which is an assessment of pulmonary edema.

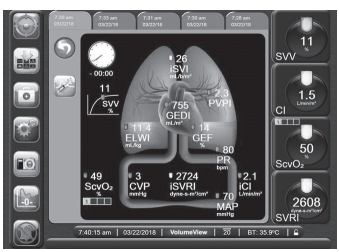
Extra vascular lung water is calculated by subtracting the pulmonary blood volume from the pulmonary thermal volume. EVLW can be used to assess the level of pulmonary edema. This is indexed against the patient's predicted body weight to obtain extra vascular lung water index (EVLWI). EVLW can be used to assess the level of pulmonary edema.



PTV
PTV = CO x Dst



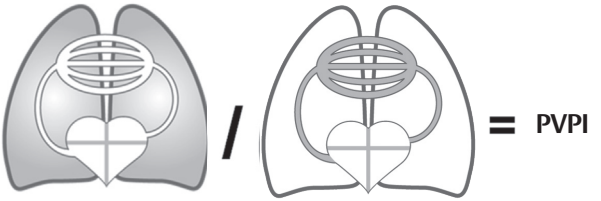
PBV = 0.25 * GEDV



The “normal” value for EVLWI is reported to be 3–7 mL/kg. Values above 10 mL/kg indicate pulmonary edema and values up to 30 mL/kg indicate severe pulmonary edema.

Pulmonary vascular permeability index

Pulmonary vascular permeability index (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.



PVPI helps the clinician to differentiate which mechanisms are responsible for increased EVLW. PVPI is often increased (>3) in patients with increased pulmonary permeability due to lung injury and usually normal in patients with hydrostatic and cardiogenic pulmonary edema.

Edwards Clinical Education

Noninvasive Monitoring

Noninvasive Monitoring

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 noninvasive systems that utilize volume clamp technology and pulse contour analysis provide the opportunity to measure blood pressure, cardiac output and other hemodynamic parameters without the need for an arterial line.

The ClearSight system is 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. By leveraging proven Nexfin technology, the ClearSight system provides clinicians clarity without the barriers of complexity or invasiveness. This technology has also been used as a standard for hemodynamic monitoring in space.

The Nexfin technology has been validated extensively throughout the years. The blood pressure measurement has performed well against both intermittent noninvasive and continuous invasive methods. Studies conclude that the Nexfin technology measures blood pressure according to the AAMI standard. When compared to invasive measurements in patients undergoing orthopedic surgery, the clinical data demonstrate that the Nexfin technology blood pressure is more reliable than a traditional upper arm blood pressure cuff.

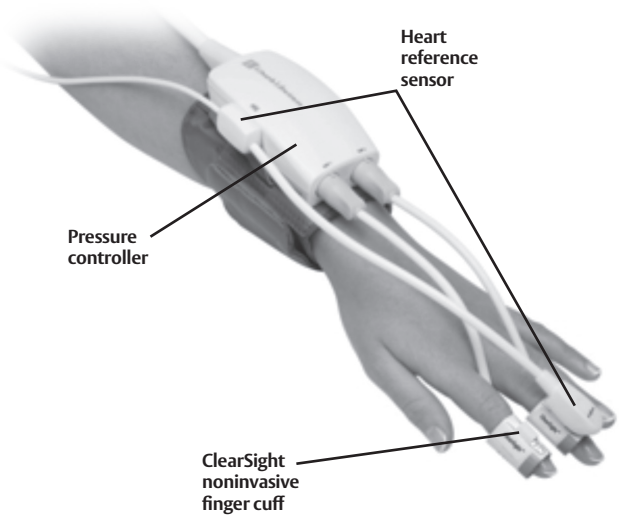
Similarly, the Nexfin technology cardiac output has been validated against several reference methods including pulmonary thermodilution, transpulmonary thermodilution and transesophageal/thoracic echo-Doppler.

Beyond the ability to measure absolute cardiac output values, several studies have shown that the Nexfin technology is able to reliably track changes in cardiac output. As a result, studies have concluded that the Nexfin technology is a suitable monitor for the perioperative continuous measurement of cardiac output.

The ClearSight system noninvasively continuously provides:

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

System components

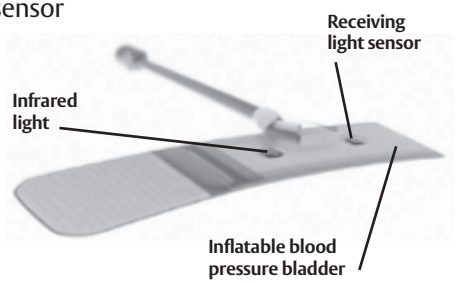


How it Works

Finger cuff

Each ClearSight finger cuff consists of:

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

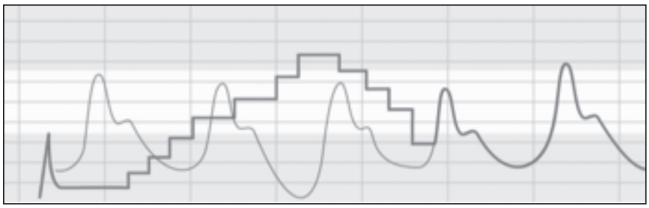


The infrared light and receiving light sensor work together to continually measure the changing arterial volume, which pulsates at the same rhythm as the heart. The pressure controller continually adjusts the pressure in the finger cuff's inflatable bladder with the result that the arteries and bladder 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**.

The Physiocal method

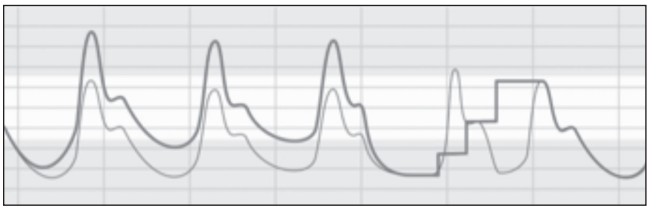
Using a process called the Physiocal method, the ClearSight system determines and periodically updates the target unloaded volume, known as the **setpoint**, in order to calibrate the blood pressure measurement.

When measurements are initiated, the ClearSight system runs the Physiocal method, 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.



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

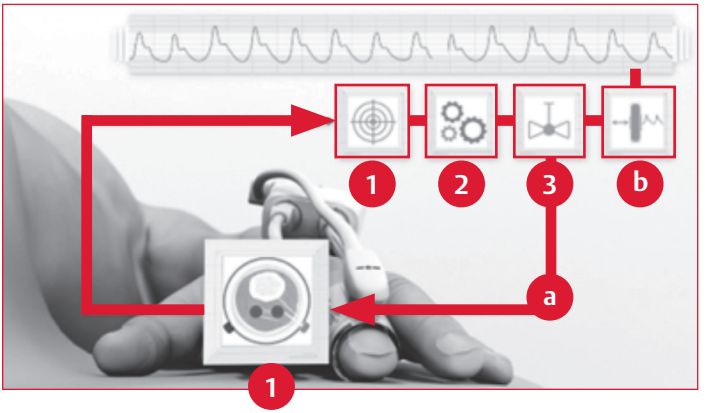
The Physiocal method periodically recalibrates the system which is essential for tracking a changing setpoint. Changes may result from smooth muscle tone changes during events such as vasoconstriction, vasodilation, and temperature change. This calibration increases to 70 beat intervals depending on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



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

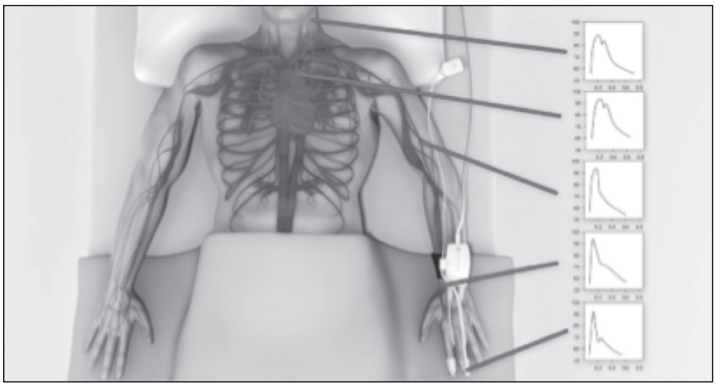
The volume clamp control loop, located within the pressure controller, consists of the following steps (see figure below):



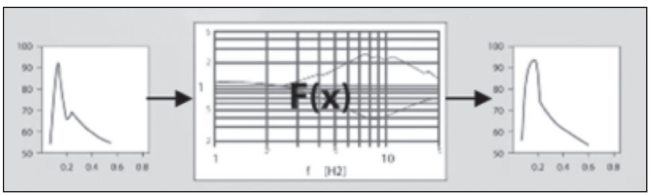
1. The arterial volume, which is measured by the infrared light and receiving light sensor, is compared to the Physiocal setpoint.
2. The pressure needed to counteract any arterial diameter change is determined by a controller.
3. a) The controller then sends a signal to the control valve which dynamically 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.

The pressure is applied by an inflatable bladder inside the cuff and is adjusted 1000 times per second to keep the arterial volume constant.

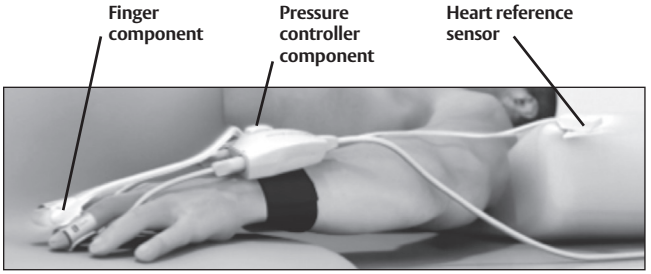
Brachial reconstruction
Since the arteries narrow as the distance from the heart increases, increased resistance and backwards reflection of the pressure waves occurs. This results in varying pressure levels and waveform shapes.



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.



Heart reference sensor
The heart reference sensor (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 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.

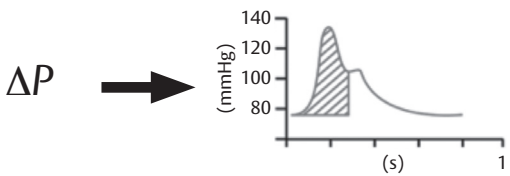
When pumping blood through the body, the left side of the heart experiences an impedance referred to as afterload, shown here as Z_{in}. This impedance is experienced due to the relationship between blood pressure and blood flow, which in this case is equivalent to SV.

$$Z_{in} = \frac{\Delta P}{SV}$$

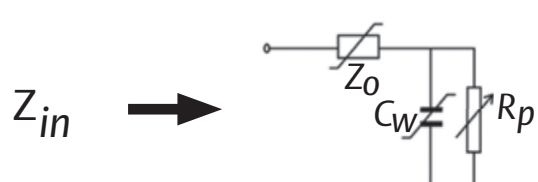
By re-arranging this relationship and individualizing it for each patient, we can use BP and afterload to calculate SV.

$$SV = \frac{\Delta P}{Z in}$$

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.



Zo – characteristic impedance
Cw – arterial compliance
Rp – 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.

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

$$CO = SV \times PR$$

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 to high-risk surgery at risk of developing complications, and critically ill patients in any hospital setting.

The ClearSight system contraindications

In some patients with extreme contraction of the smooth muscle in the arteries and arterioles in the lower arm and hand, such as many be present in patients with Raynaud's disease, blood pressure measurement can become impossible.

Edwards Clinical Education

Methods for Assessing Fluid Responsiveness

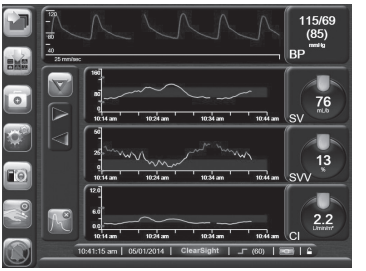
Methods for Assessing Fluid Responsiveness

Passive leg raise (PLR) maneuver

Patients who are preload responsive will:

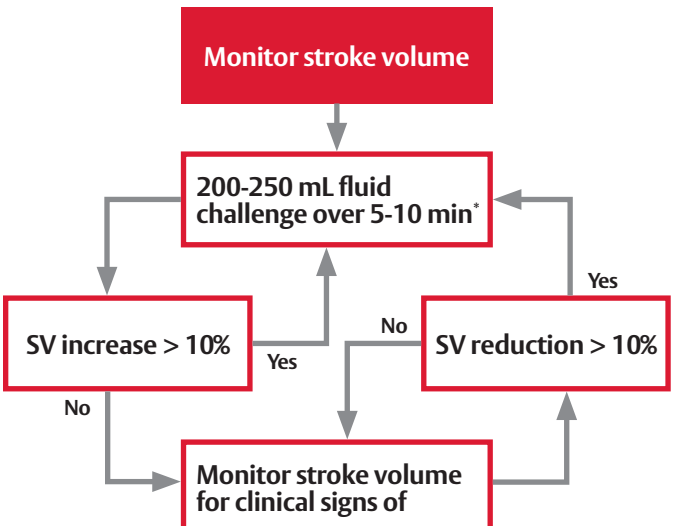
- Usually show a maximal effect within 120 seconds
- Reach a >10% increase in stroke volume

1. Start from semi-recumbent and not the supine position. Check that the trunk is at 45°
2. PLR effects must be assessed by a direct measurement of CO and not by measurement of BP
3. The technique must be able to detect short-term and transient changes since the PLR effects may vanish after one minute
4. CO must be measured before, during and after PLR in order to check that it returns to its baseline
5. Pain, cough discomfort, and awakening could provoke adrenergic stimulation, resulting in mistaken interpretation of cardiac output changes



Fluid challenge

% Change in Stroke Volume (ΔSV) is a sensitive method for assessing preload responsiveness of ***all patients***.



- A passive leg raising maneuver over 1-2 minutes in duration is a noninvasive means for assessing fluid responsiveness.

Trending dynamic parameters

Hemodynamic monitoring can be obtained continuously or intermittently and by 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 reliable indicator of preload responsiveness for patients on controlled ventilation.

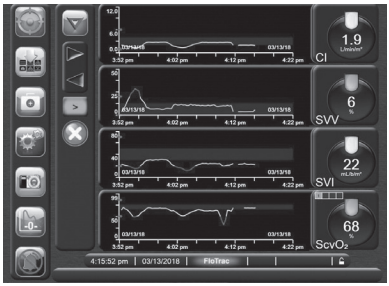
Hemodynamic parameters for assessing volume status and fluid responsiveness

Static parameters	Dynamic parameters
Central venous pressure (CVP)	Systolic pressure variation (SPV)
Pulmonary artery occlusion pressure (PAOP)	Arterial pulse pressure variation (PPV)
	Stroke volume variation (SVV)

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 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.

Advanced SVV trending screens



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 utility of SVV for predicting responsiveness to fluid challenge.

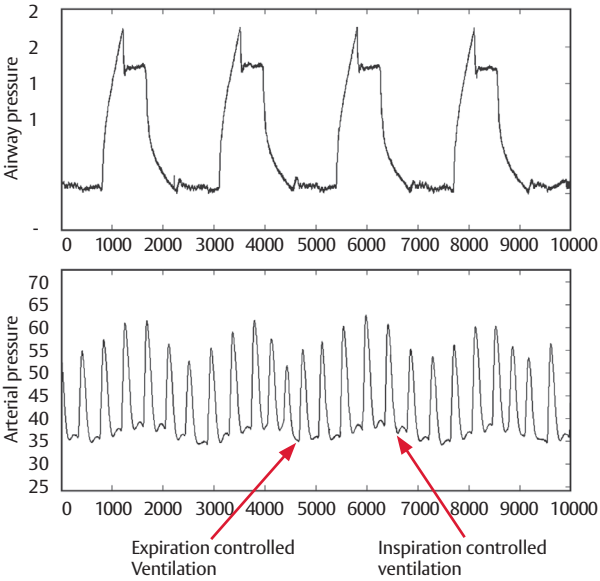
SVV is increasingly used to determine fluid responsiveness and to monitor the effects of volume therapy. Due to the provided parameters, tools such as the FloTrac and ClearSight systems are being adopted to provide insight into fluid optimization, blood flow and components of oxygen delivery.

The systems include advanced SVV trending screens that provide vital information to enhance the clinical workflow.

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 spontaneous 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 0-10 mmHg.

Reverse pulsus paradoxus is the same phenomenon with controlled mechanical ventilation. Arterial pressure rises during inspiration and falls during expiration due to changes in intra-thoracic pressure secondary to positive pressure ventilation. 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 determined period of time.

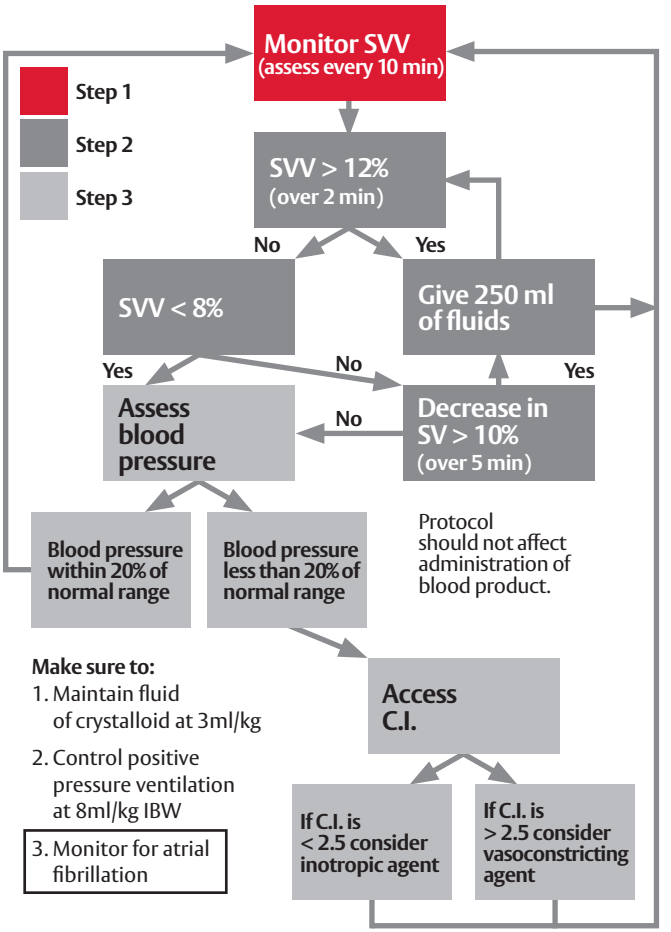


SVV and assessing fluid response

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 (CVP, PAOP, RVEDV), and their ability to determine fluid responsiveness.

Application of SVV

Normal SVV values are less than 10-15% on controlled mechanical ventilation.



Cannesson et al. (2015). Perioperative goal-directed therapy and postoperative outcomes in patients undergoing high-risk abdominal surgery: a historical-prospective, comparative effectiveness study. Critical Care 19:3.

Limitations to the use of SVV (LIMITS)

- **L**ow HR/RR ratio
- **I**rregular heart beats
- **M**echanical ventilation with low tidal volume
- **I**ncreased abdominal pressure
- **T**horax open
- **S**pontaneous breathing

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 assist with fluid optimization and provide information about cardiopulmonary interactions. SVV is an available parameter with the FloTrac sensor or a ClearSight fingercuff with a compatible Edwards monitoring system.

Note: The FloTrac sensor can be used to monitor cardiac output, stroke volume and systemic vascular resistance in the spontaneously breathing patient with dysrhythmias or the mechanical ventilated patient.

Edwards Clinical Education

Swan-Ganz Catheters – Advanced and Standard Technology

The Swan-Ganz Pulmonary Artery Catheter

Standard Swan-Ganz Catheter

The standard thermodilution Swan-Ganz pulmonary artery catheter was introduced in 1970 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 (PAP)
    - Pulmonary artery systolic (PAS)
    - Pulmonary artery diastolic (PAD)
    - Mean pulmonary arterial pressure (MPAP)
    - Pulmonary artery occlusion pressure (PAOP)
- Thermodilution cardiac output:
  - Edwards CO-Set iced, closed bolus injectate system
  - Edwards CO-Set room temperature, closed bolus injectate system
- Pulmonary artery blood sampling for laboratory analysis:
  - Mixed venous blood oxygen saturation (SvO_2)
  - 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

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 evaluating 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
- Temporary transvenous ventricular or atrioventricular 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. Through analysis of components of stroke volume (preload, afterload and contractility) patients with cardiac dysfunction can be identified early, treated appropriately thus potentially avoiding tissue hypoxia, and organ dysfunction.

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)
  - Mean pulmonary arterial pressure (MPAP)
  - Pulmonary artery occlusion pressure (PAOP)
- 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 (SvO_2)
- SvO_2 – 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 continuously measures analysis indicates right ventricular function
- EDV - end diastolic volume of the right ventricle is continuously calculated by dividing stroke volume (mL/beat) by RVEF giving a key indicator of preload
- SVR – continuous systemic vascular resistance can be calculated when a compatible Edwards monitoring platform system (i.e. Vigilance II, Hemosphere advanced monitoring platform) obtains continuous MAP and CVP from the bedside physiologic monitor
- RVSWI - right ventricular stoke work index indicates how hard the right ventricle works to pump blood

Applications of advanced technology Swan-Ganz catheters:

- Continuous assessment of right heart pressures (RAP, PAD, PAS)
- Continuous assessment of oxygen delivery and consumption (SvO_2)
- Continuous assessment of cardiac output (CCO) a primary component of DO_2
- Continuous assessment of preload through EDV
- Continuous assessment of afterload through SVR, SVRI
- Continuous assessment of contractility through RVEF, and calculation of right ventricular stroke work index (RVSWI)
- Intermittent calculation of oxygen delivery (DO_2) and consumption (VO_2)

Advantages of the advanced technology Swan-Ganz catheter as compared to the standard Swan-Ganz catheter:

- Additional advanced diagnostic information with same procedure
- Continuous assessment of DO_2 /VO_2 balance with SvO_2 monitoring
- Continuous assessment of components of stroke volume (preload, afterload, and contractility) (EDV, SVR, RVEF)

Applications and contraindications*:
Clinical applications for Swan-Ganz pulmonary artery catheters:

- Acute respiratory distress syndrome (ARDS)
- Cardiac surgery
- Intra- and post-operative high-risk surgery management
- Patient on intra-aortic balloon counterpulsation
- Complex liver resections
- Liver transplantation
- Complex lung resection
- Pulmonary edema
- Pulmonary hypertension
- Shock of unknown etiology
- Shock unresponsive to attempts at resuscitation
- Severe trauma
- Post-resuscitation in critically ill patient

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
- Heparin coated catheters in patients with known sensitivity to heparin

* Not an exhaustive list

Catheter Insertion Distance Markings*

Location	Distance to Vena Cava (VC)/ Right Atrium (RA) Junction	Distance to Pulmonary Artery (PA)
Internal jugular	15 to 20	40 to 55
Subclavian vein	10 to 15	35 to 50
Femoral vein	30	60
Right antecubital fossa	40	75
Left antecubital fossa	50	80

*(in cm) and dependent upon patient height

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.

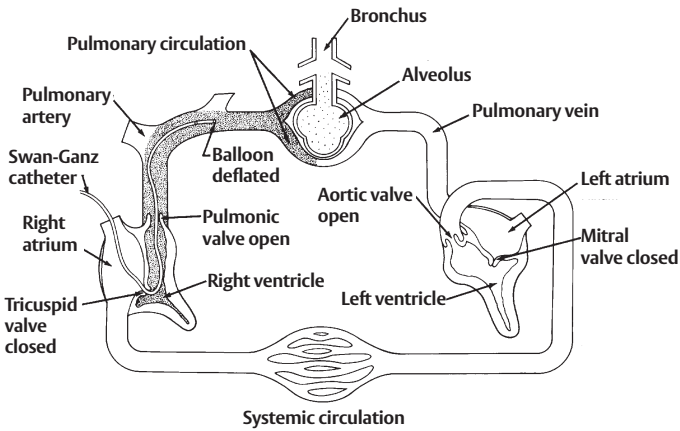
Physiological Basis for Pulmonary Artery Pressure Monitoring

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 essentially there is now a common chamber with a common volume and pressure.

Ventricular systole

RVSP = PASP



Ventricles in diastole

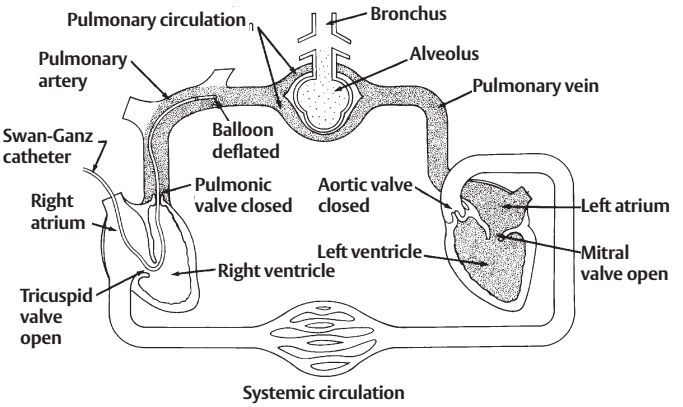
During diastole the tricuspid and mitral valves are open. The ventricles are filling with blood from their respective atria. At this time the tricuspid valve and mitral valve are open and the pulmonic valve and aortic valve are closed.

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 (LAP) 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 (RAP) reflects right ventricular end-diastolic pressure (RVEDP) when the tricuspid valve is open.

Ventricular diastole
RAP = RVEDP
RVEDP < PADP
PADP ≈ LAP ≈ LVEDP

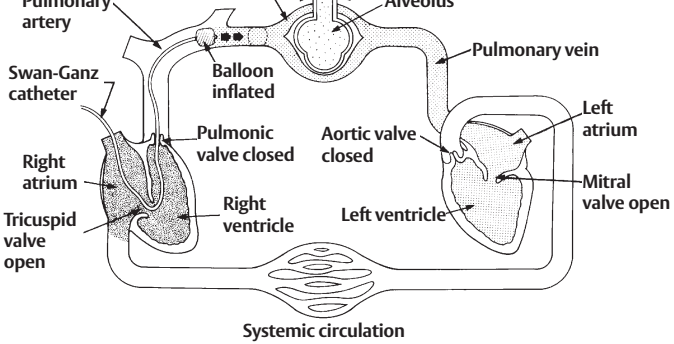


Ventricles in diastole: catheter wedged
By inflating the balloon, the catheter floats downstream into a smaller branch of the pulmonary artery. Once the balloon lodges, the catheter is considered "wedged". It is in this wedge position that right sided and pulmonary artery diastolic pressures are effectively occluded and PAOP can be obtained.

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

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.

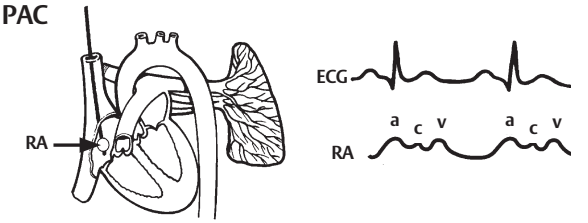
Ventricular diastole
PAOP ≈ LAP ≈ LVEDP



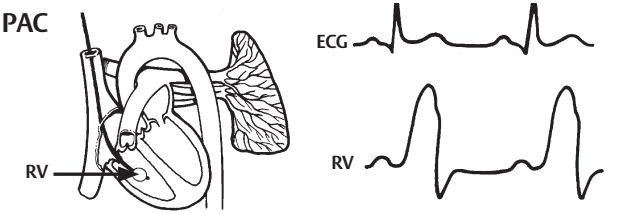
Normal Insertion Pressures and Waveform During Pulmonary Artery (PAC) Catheter Insertion

Catheter tip in right atria representing right atrial pressure (RAP) or central venous pressure (CVP)
2 to 6 mmHg
Mean 4 mmHg

a = atrial systole
c = backward bulging from tricuspid valve closure
v = atrial filling, ventricular systole

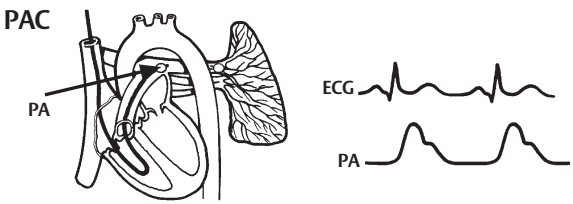


Catheter tip in right ventricle representing right ventricular pressure
Systolic Pressure (RVSP)
15–25 mmHg
Diastolic Pressure (RVDP)
0–8 mmHg

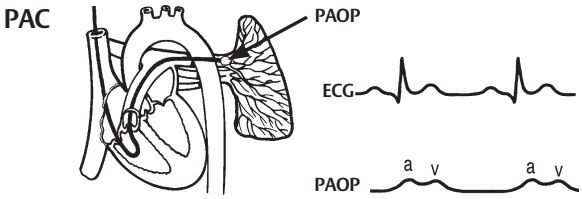


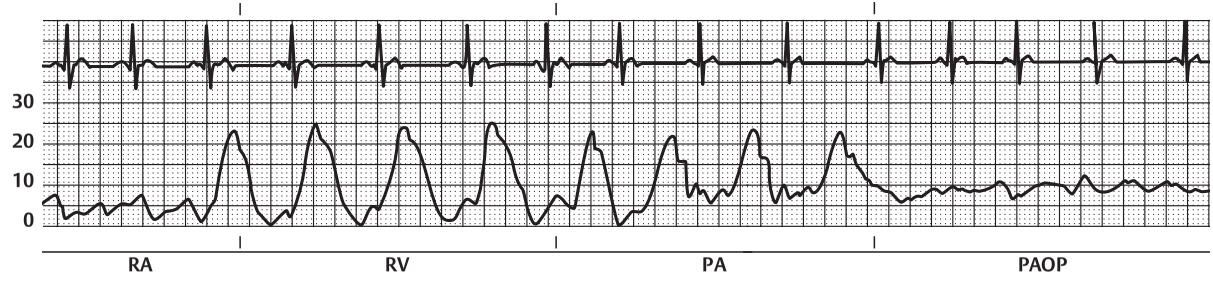
Swan-Ganz Catheter Insertion Waveforms

Catheter tip in pulmonary artery representing pulmonary artery pressure (PAP)
Systolic Pressure (PASP)
15–25 mmHg
Diastolic Pressure (PADP)
8–15 mmHg
Mean Pressure (MPA)
10–20 mmHg



Catheter tip in pulmonary artery representing pulmonary artery occlusion pressure (PAOP) or pulmonary artery wedge pressure (PAWP)
Mean 6–12 mmHg
a = atrial systole
v = atrial filling, ventricle systole




30
20
10
0
RA
RV
PA
PAOP

Abnormal Waveform Chart

Right atrial waveforms	Related causes
Decreased mean pressure	Hypovolemia Improperly leveled transducer
Elevated mean pressure	Fluid overload states Right ventricular failure Left ventricular failure causing right ventricular failure Tricuspid stenosis or regurgitation Pulmonic stenosis or regurgitation Pulmonary hypertension
Elevated "a" wave: atrial systole, increased resistance to ventricular filling	Tricuspid stenosis Decreased right ventricular compliance Right ventricular failure Pulmonic stenosis Pulmonary hypertension
Absent "a" wave	Atrial fibrillation Atrial flutter Junctional rhythms
Elevated "v" wave: atrial filling, regurgitant flow	Tricuspid regurgitation Right ventricular failure from tricuspid valve malfunction
Elevated "a" and "v" waves	Cardiac tamponade Constrictive pericardial disease Hypervolemia

Right ventricular waveforms	Related causes
Elevated systolic pressure	Pulmonary hypertension Pulmonic valve stenosis Increased pulmonary vascular resistance
Decreased systolic pressure	Hypovolemia Cardiogenic shock (RV failure) Cardiac tamponade
Increased diastolic pressure	Hypervolemia Congestive heart failure Cardiac tamponade Pericardial constriction
Decreased diastolic pressure	Hypovolemia

Note: These are not all inclusive related causes.

Pulmonary artery waveforms	Related causes
Elevated systolic pressure	Pulmonary disease Increased blood flow, left to right shunt Increased pulmonary vascular resistance
Elevated diastolic pressure	Left heart failure Other left-sided heart disease Intravascular volume overload Mitral stenosis or regurgitation

Pulmonary artery wedge/ left atrial waveform	Related causes
Decreased (mean) pressure	Hypovolemia
Elevated (mean) pressure	Fluid overload states Left ventricular failure Other left-sided heart disease 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 Right ventricular failure Tricuspid regurgitation from valve malfunction Ventricular septal defect
Elevated "a" and "v" waves	Cardiac tamponade Constrictive pericardial disease Left ventricular failure

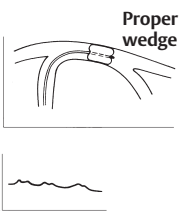
Note: These are not all inclusive related causes.

Insertion Techniques for the Swan-Ganz Catheters

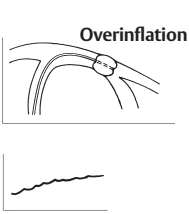
1. Before insertion of the Swan-Ganz catheter, prepare the pressure monitoring system for use according to institutional policies and procedures.
2. Insert the catheter following recommended guidelines and advance the catheter towards the thorax.
3. Once the catheter tip has exited the introducer sheath (approximately 15 cm) and reached the junction of the superior or inferior vena cava and right atrium, the balloon is inflated with air (per institutional policy) to the full volume indicated on the catheter shaft and gate valve is locked. 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 right ventricle 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 produces a wedge pressure tracing.

Continuous Pulmonary Artery Pressure Monitoring

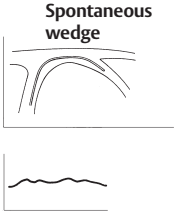
1. Optimize pressure monitoring systems according to manufacturers' recommendations.
2. Maintain patency of inner lumens with heparinized solution or continuous flush systems, per institutional protocol.
3. Observe waveforms for proper placement.
4. Catheter migration may occur. Note any damping of the PA tracing as catheter position may have changed.
5. Catheter may slip back to RV. Observe waveforms for spontaneous RV tracings and 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 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 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 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 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 full indicated volume, pull the catheter back to a position where the full volume 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 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. 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.

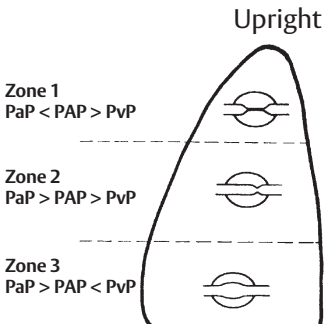
4. Obtain a pulmonary artery occlusion "wedge" pressure only when necessary
   - If the pulmonary artery diastolic (PAD) and the wedge (PAOP) pressures are nearly identical, wedging the balloon may not be necessary: measure PAD pressure instead of PAOP as long as the patient's heart rate, blood pressure, 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, abort attempting 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 patients with pulmonary hypertension

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.

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).

Lung zones



Zone 1: PaP < PAP > PvP. No blood flow occurs from the pulmonary 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.

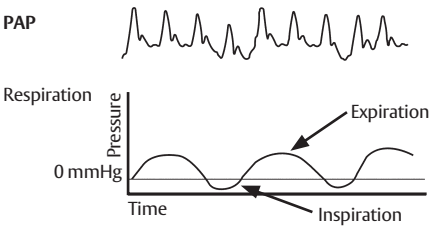
Zone 2: PaP > PAP > PvP. Some blood flow occurs since the pulmonary arterial pressure is greater than the alveolar pressure. Under some conditions catheter tip may reside in Zone 2 placement. PAOP readings may be inaccurate.

Zone 3: PaP > PAP < PvP. Capillaries are open resulting in blood flow. Generally the pulmonary artery catheter tip should reside in zone 3. PAOP readings will be accurate.

Ventilatory Effects on Pulmonary Artery Tracings

Spontaneous Breathing

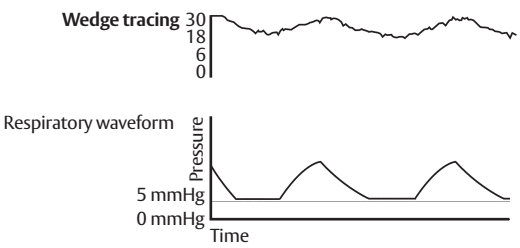
During normal respiration, inspiration results in decreased intrathoracic pressure and increased venous return resulting in increased cardiac filling. However, the waveforms on inspiration will be negative due to the greater inspiratory decrease in intrathoracic pressure than the inspiratory increase in the cardiac volumes. On expiration, the intrathoracic pressure is 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.



Note: positive deflection on respiratory waveform = expiration, negative deflection = inspiration

Controlled Mechanical Ventilation (CMV)

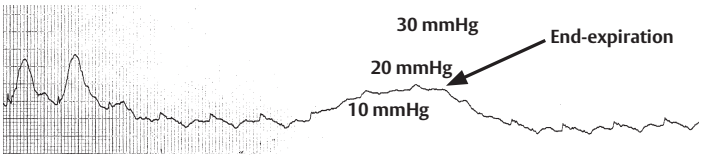
When a patient is ventilated and not spontaneously breathing, the intrathoracic pressure during inspiration is positive with ventilated breaths known as positive pressure ventilation. PA and PAOP, are read at end-expiration. Beginning of inspiration pressure begins to rise and expiration ends. This is where the wedge pressure should be obtained.



Note: Simultaneous reading of PAP and airway pressures are displayed in the CMV graph.

This is a tracing of a patient who is spontaneously breathing. Pressure values should be obtained at end-expiration. End of expiration is identified as the beginning of inspiration where the intra-thoracic pressure starts to decrease.

PAP to PAOP tracing during balloon inflation



Cardiac Output Determinations

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 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 through the proximal 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. The resultant change in temperature is then plotted on a time and temperature curve and is similar to the one produced by the indicator-dilution method.

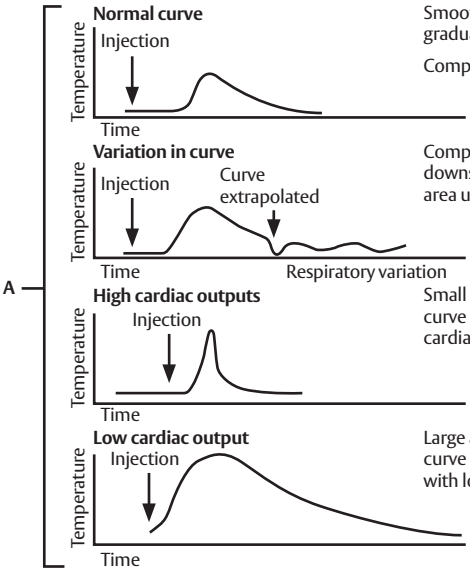
A modified Stewart-Hamilton equation is used to calculate the cardiac output taking into consideration the change in temperature as the indicator. Modifications include the measured temperature of the injectate and the patient's blood temperature, along with the specific gravity of the solution injected.

Thermodilution Curves

A normal curve characteristically shows a sharp upstroke from rapid injection of the injectate and change in temperature. This is followed by a smooth curve and slightly prolonged downslope back to the baseline. The area under the curve is inversely proportional to the cardiac output.

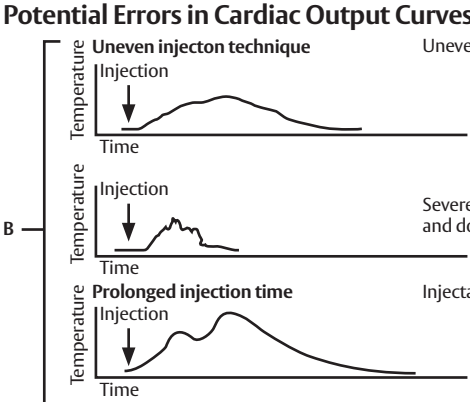
When cardiac output is low, more time is required for the temperature to return to baseline, producing a larger area under the curve. With high cardiac output, the cooler injectate is carried more quickly through the heart, and the temperature returns to baseline faster. This produces a smaller area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

Variations in Cardiac Output Curves



A, Variations in the normal cardiac output curve seen in certain clinical conditions.

Potential Errors in Cardiac Output Curves

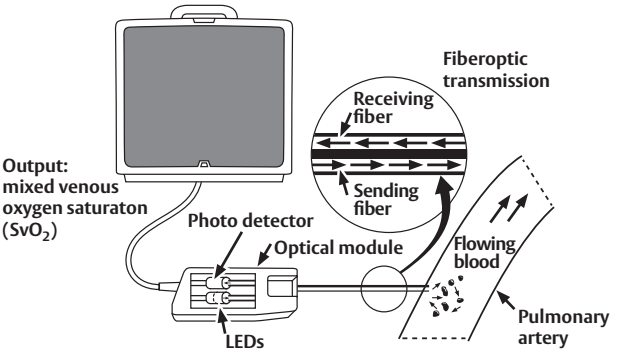


B, Abnormal cardiac output curves that produce an erroneous cardiac output value. *(From Urden LD, Stacy KM, Lough ME: Critical care nursing: Diagnosis and management, ed,7, St Louis, 2014, mosby.)*

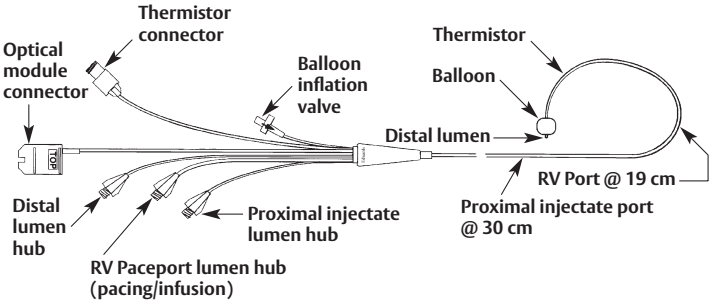
Note: y axis equals change in temperature

Continuous Mixed Venous Oxygen Saturation Monitoring

Reflection spectrophotometry



Swan-Ganz oximetry TD catheter



RVEDV Quick Reference

1. Parameters attained with a compatible Edwards monitoring platform system
   - Cardiac output (CO) = 4 – 8.0 L/min
   - Cardiac index (CI) = 2.5 – 4.0 L/min/m^2
   - Stroke volume (SV): The volume of blood ejected from the ventricle in each beat.
     SV = CO / HR x 1000
     Normal SV: 60 – 100 mL/beat
     Normal SVI: 33 – 47 mL/beat/m^2
   - End-diastolic volume (EDV): The volume of blood in the ventricle at the end of the diastole.
     EDV = SV/EF
     Normal RVEDV: 100 – 160 mL
     Normal RVEDVI: 60 – 100 mL/m^2
   - Ejection fraction (EF): The percentage of blood ejected from the ventricle with each beat.

$$EF = \frac{(EDV\text{-}ESV/EDV) \times 100}{EDV} \quad \text{or} \quad EF = \frac{SV/EDV \times 100}{EDV}$$

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.

Edwards Clinical Education

Monitoring Platforms

EV1000 Clinical Platform

The EV1000 clinical platform from Edwards Lifesciences presents the physiologic status of the patient in an intuitive and meaningful way. Designed in collaboration with and validated by clinicians, the EV1000 clinical platform offers scalability and adaptability for both the OR and ICU.

The EV1000 clinical platform provides the choice of the parameters to view and can be configured as desired. The platform may be used with the Edwards advanced hemodynamic monitoring portfolio including the ClearSight finger cuff, FloTrac sensor, Edwards oximetry central venous catheter and VolumeView set.

ClearSight finger cuff
(Noninvasive)
The ClearSight system extends the benefits of continuous hemodynamic monitoring including SV, SVV, SVR, CO and continuous blood pressure for a broad patient population including moderate to high-risk surgical patients.

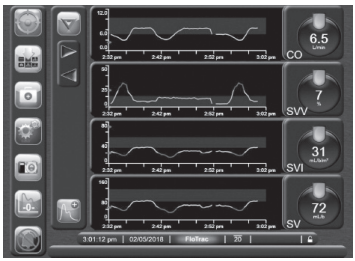
FloTrac sensor
(Minimally-Invasive)
The FloTrac sensor easily connects to any existing arterial catheter and automatically calculates key flow parameters (CCO/CCI, SV/SVI, SVV, SVR/SVRI) every 20 seconds, making it the practical and reliable solution for hemodynamic optimization in the critical care setting.

Edwards oximetry central venous catheter
The Edwards oximetry central venous catheter continuously monitors central venous oxygen saturation ($ScvO_2$), which may be used in goal-directed therapy (GDT) protocols for the treatment of sepsis.

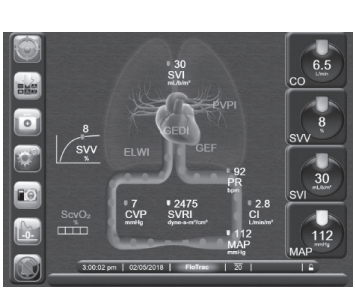
VolumeView set
(Transpulmonary thermodilution)
The VolumeView set provides volumetric parameters (EVLW, GEDV, GEF, PVPI, ITBV) and continuous, calibrated hemodynamic parameters (CCO/CCI, SV/SVI, SVV, SVR/SVRI).

EV1000 clinical platform screens

Graphical trend screen:
The graphical trend screen displays the current status and history of monitored parameters and the continuous, beat to beat arterial (ART) waveform when selected. The amount of history shown for monitored parameters can be configured by adjusting the time scale.

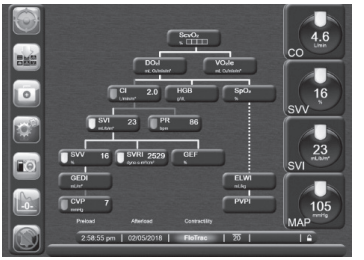


Physiology screen:
The physiology screen displays monitored parameters using a visual representation of the heart and circulatory system and their relevant measured volume.

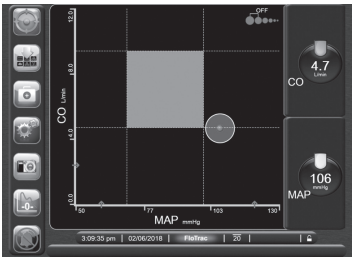


Cockpit screen:
The cockpit screen displays globes with the values of the parameter being monitored. They graphically indicate the target values, and whether they are out of range or within the desired target, with needle indicators to show where the patient's parameter falls. In addition, the value within the globe will flash when the parameter is alarming.

Physio relationship screen:
The physio relationship screen displays most of the parameters available on the system and their relationship to each other. The screen displays lines connecting the parameters highlighting the relationship of the parameters to each other.



Goal positioning screen:
The goal positioning screen allows the user to monitor and track the relationship of two key parameters by plotting them against each other on an XY plane.



HemoSphere Advanced Monitoring Platform

The HemoSphere advanced monitoring platform allows you to see, experience and interact with hemodynamic parameters.

The HemoSphere advanced monitoring platform introduces adaptive modularity:

- Scalable expansion modules and interchangeable cable ports
- Wired and wireless communication
- Multiple visual clinical support screens
- Seamless and encrypted integration with hospital information systems
- Hot-swappable battery

HemoSphere advanced monitoring platform is compatible with both Swan-Ganz pulmonary artery catheters and Edwards oximetry central venous catheter.

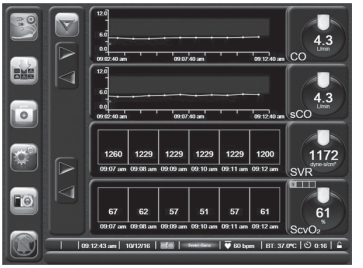
The HemoSphere advanced monitoring platform has the capability to provide:

- Central Venous Oxygen Saturation ($ScvO_2$)
- Continuous Cardiac Output and Continuous Cardiac Index (CCO, CCI)
- Continuous Right Ventricular Ejection Fraction (RVEF)
- Continuous Right Ventricular End Diastolic Volume and Right Ventricular End Diastolic Volume Index (RVEDV, RVEDVI)
- Mixed Venous Oxygen Saturation (SvO_2)
- Pulmonary Vascular Resistance (PVR)
- Right Ventricular Stroke Work Index (RVSWI)
- Stroke Volume and Stroke Volume Index (SV, SVI)
- Systemic Vascular Resistance and Systemic Vascular Resistance Index (SVR, SVRI)

The modular design allows the HemoSphere advanced monitoring platform to be adapted to future changes. Furthermore, it can also be placed on a tabletop, pole or rack to meet individual patient requirements.

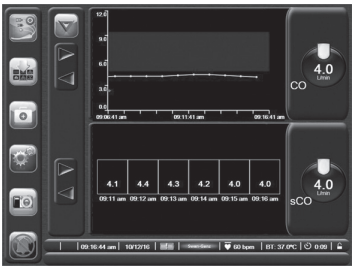
HemoSphere advanced monitor screens

Graphical trend:
Allows clinicians to select, place, and track interventions over time while providing key parameter trending data. The percent change indicator provides additional insight into the patient's condition.



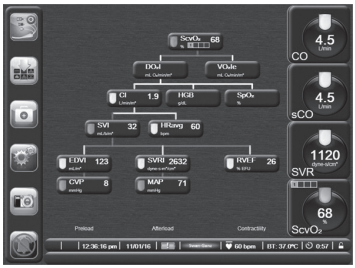
Tabular:
Displays selected physiologic properties and their history in tabular form.

Graphical tabular:
Useful for viewing both graphical and tabular format parameters on one screen.

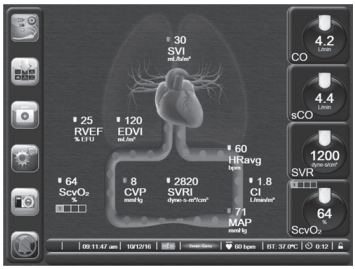


Cockpit:
Combines larger, easy-to-read numbers with specific color target ranges, parameter and alarms to clearly indicate patient status and monitoring needs.

Physio-relationship screen:
Depicts the balance between oxygen delivery and consumption, allowing clinicians to identify the root cause of the imbalance and the most appropriate intervention. Automatically updates with real-time data.



Real-time physiology screen:
Depicts real-time changes occurring in patients by delivering visual and numeric parameters.



Edwards Clinical Education

Clinical Decision Support Software

Acumen Hypotension Prediction Index Software

Overview

- Edwards Lifesciences offers a predictive decision support tool with the Acumen Hypotension Prediction Index software
- The Acumen Hypotension Prediction Index software is intended for use in operating room patients receiving advanced hemodynamic monitoring
- Acumen Hypotension Prediction Index software, when activated and when using the FloTrac IQ sensor, connected to a radial arterial catheter, provides the clinician with information regarding the likelihood of a patient trending towards a hypotensive event, defined as mean arterial pressure (MAP) < 65 mmHg for at least one minute
- The proprietary algorithm was developed using machine learning, trained from almost 59,000 past hypotensive events and 144,000 non-hypotensive events*
- The algorithm also provides clinicians with insights into advanced hemodynamic parameters through the secondary screen
- The HPI secondary screen provides hemodynamic information and may be a useful tool to review the patient hemodynamics related to hypotension

* Data on file

The Acumen Hypotension Prediction Index software is comprised of these elements:

- HPI parameter
- HPI high alert popup
- HPI secondary screen

HPI parameter

The proprietary algorithm coupled with machine learning techniques detects potential hypotensive trending of a patient's mean arterial pressure (MAP). The HPI parameter is updated every 20 seconds. Once the Acumen Hypotension Prediction Index software is activated, the user can choose to configure the HPI parameter as a key parameter, allow for display on the information bar, or choose to disable/not display.

HPI high alert popup

When the HPI parameter value exceeds 85 for two consecutive 20-second readings, or reaches 100 at any time, an HPI high alert popup will appear, warranting an acknowledgement or a deeper review of patient hemodynamics via the HPI secondary screen.

HPI secondary screen

The secondary screen is easily accessed through the HPI high alert popup, by pressing the button on the HPI parameter globe, or at any time through the menu on your monitor. It visually displays advanced parameters on the screen which are arranged by preload, contractility and afterload and which provide you potential insight into the cause of a hypotensive event.*

* A hypotensive event is defined as MAP < 65 mmHg for at least one minute in duration

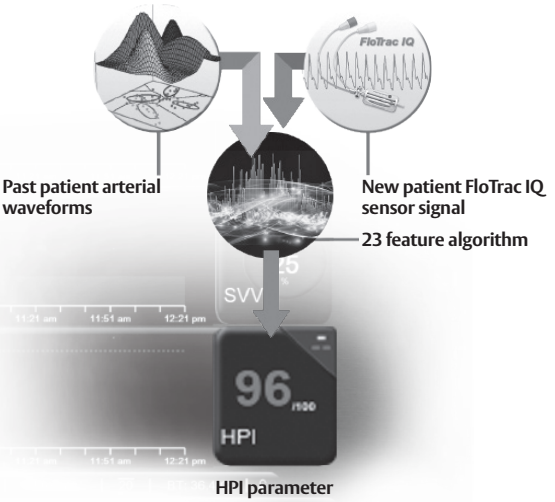
The Algorithm- How Does It Work?

The proprietary algorithm for the Acumen Hypotension Prediction Index software is a mathematical model developed by learning from almost 59,000 past hypotensive events and over 144,000 non-hypotensive events:

- Formulated using 23 key arterial waveform features from patients within the development dataset
- Features from current patient FloTrac IQ sensor signal are fed into the model to determine the HPI parameter value

The higher the value, the higher the likelihood that a hypotensive event will occur.

The lower the value, the lower the likelihood that a hypotensive event will occur.



Past patient arterial waveforms

New patient FloTrac IQ sensor signal

23 feature algorithm

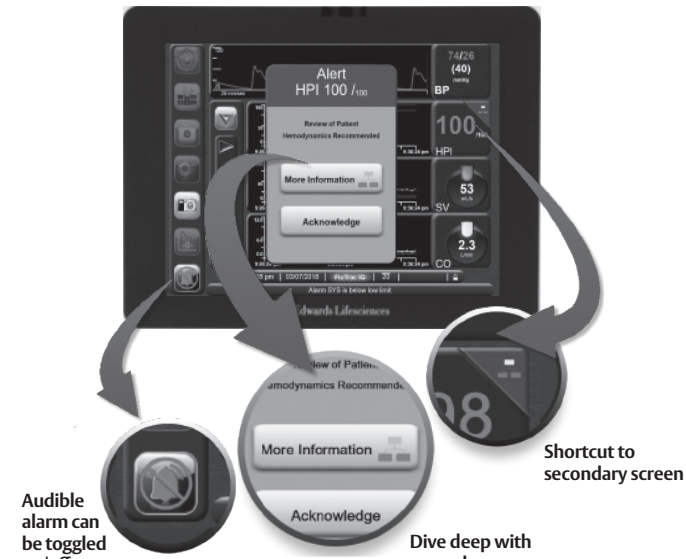
HPI parameter

Acumen Hypotension Prediction Index Software Functionality

Alarm and HPI high alert popup

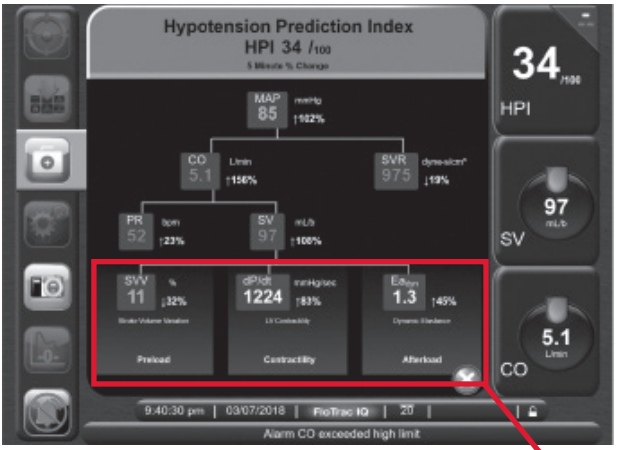
The HPI high alert popup alerts clinicians in the case of a potential hypotensive event.

- When the HPI parameter is above 85, an alarm will activate, indicating to the user that the patient may be trending toward a hypotensive event; this includes an alarm tone, red parameter status color, and flashing parameter value
- The alarm range for the HPI parameter is not configurable. It has a preset threshold value of 85
- When the HPI parameter value is above 85 for two consecutive readings or is at 100, an HPI high alert pop-up will appear on screen, warranting an acknowledgement or a deeper review of patient hemodynamics via the secondary screen
- Acknowledging the HPI high alert popup disables the alarm sound until a new alert occurs



HPI Secondary Screen

- The HPI secondary screen is part of the Acumen Hypotension Prediction Index software and provides the clinician with deeper insights into potential underlying causes of a hypotensive event
- The HPI secondary screen provides additional information to inform clinical decisions



Key details

- The HPI secondary screen can be accessed by pressing the shortcut icon in the upper right corner of the area outside the HPI parameter globe
- It can also be accessed by touching the display on the information bar if the HPI parameter is not a key parameter, and by touching the Clinical Actions icon and then touching the "More" icon
- Parameters on the HPI secondary screen update at the same rate as key parameters, every 20 seconds
- % change is also displayed next to each parameter, and the interval over which it is calculated is changed just as it is for other parameters. Default % change is 5 minutes
- The color coding for each parameter is set by the target limits for each parameter

Additional parameters

- dP/dt is a peripheral measure of left ventricular contractility[1,2] It is the maximal first derivative (positive slope) of arterial pressure
- Functional assessment of arterial load by dynamic arterial elastance (Ea_{dyn}), defined as the ratio between pulse pressure variation (PPV) and stroke volume variation (SVV), has recently been shown to predict the arterial pressure response to volume expansion (VE) in hypotensive, preload- dependent patients
- dP/dt and Ea_{dyn} always appear in white font as additional non-configurable parameters

Parameter	Description/formula	Units
dP/dt	**Maximal first derivative with respect to time of arterial pressure waveform** **dP/dt = max(P[n+1]-P[n]), ts for n=0 to N-1** where: P[n]: current sample of the arterial pressure signal, mmHg; ts - sampling time interval, second; N - total number of samples in a given cardiac cycle	mmHg/sec
SVV	**Stroke Volume Variation** SVV = 100 x (SVmax - SVmin) / mean(SV)	%
Ea_{dyn}	**Ea_{dyn} = PPV/SVV**	None

The correlation of these parameters to physiological status and their relationship to clinical outcome has been well studied with a large body of clinical literature.

Most interventions to treat SV (or SVI) and MAP, impact primarily SV and its determinants preload, contractility, afterload. Decision support for treatment decisions should integrally provide information on all three aspects, since they often inter-relate.

Preload Contractility Afterload

SVV is limited as preload measure to patients that are mechanically ventilated with stable ventilation frequency and tidal volumes and that do not have intra-abdominal insufflation. SVV is best used in conjunction with stroke volume or cardiac output assessment.

dP/dt is best used in conjunction with stroke volume variation and stroke volume or cardiac output assessment.

By normalizing the arterial elastance by the ventricular elastance, their ratio becomes an index of the matching between the LV and the arterial system. When matching there is an optimal transfer of blood from the LV to the arterial system without loss of energy and with optimal stroke work.

Ea_{dyn} has been shown to provide an indication of potential afterload responsiveness to increase MAP by giving volume in preload volume responsive mechanically ventilated patients and spontaneously breathing patients. Afterload responsiveness to increase MAP is greater potentially at values of $Ea_{dyn} > 0.8$.

Ea_{dyn} is not limited to patients that are mechanically ventilated because it is a computation presented as the ratio of PPV/SVV. Ea_{dyn} is best used in conjunction with stroke volume variation (in ventilated patients) and stroke volume or cardiac output assessment.

SVV, dP/dt, Ea_{dyn} share the property that one is seldom independent of one or the other. Giving volume to increase the preload and increase the stroke volume leads to an increase in cardiac output and arterial pressure; therefore, the afterload on the ventricle increases. Increasing afterload (increasing aortic pressure) by increasing systemic vascular resistance, will reduce the stroke volume. The resulting increased end-systolic volume, however, leads to a secondary increase in end-diastolic volume because more blood is left inside the ventricle following ejection and this extra blood is added to the venous return, thereby increasing ventricular filling, which increases contractility (Frank-Starling mechanism) and partially offsets the reduction in stroke volume caused by the initial increase in afterload.

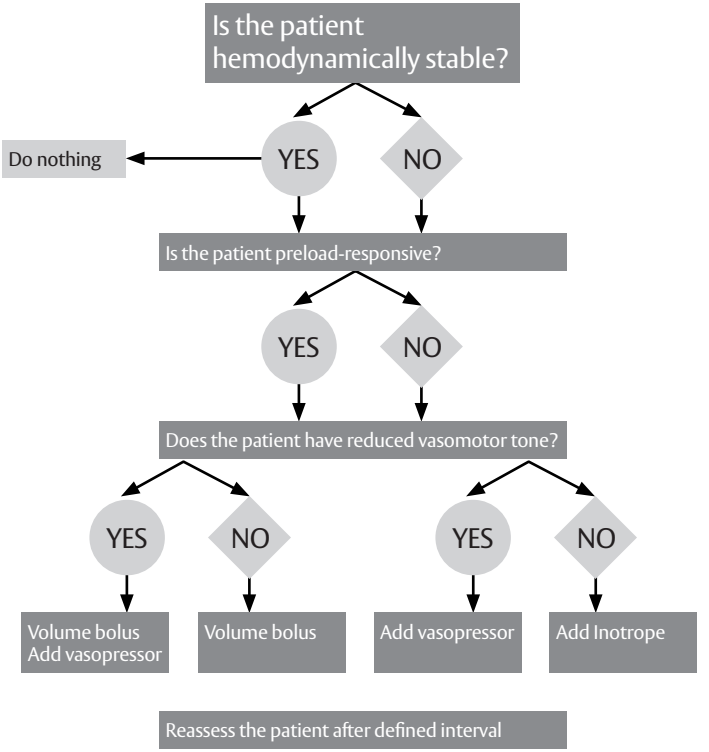
SVV, dP/dt and Ea_{dyn} are intended as integrative decision support parameters to guide an interventional treatment of SV or SV and MAP.

Edwards Clinical Education

Quick Reference

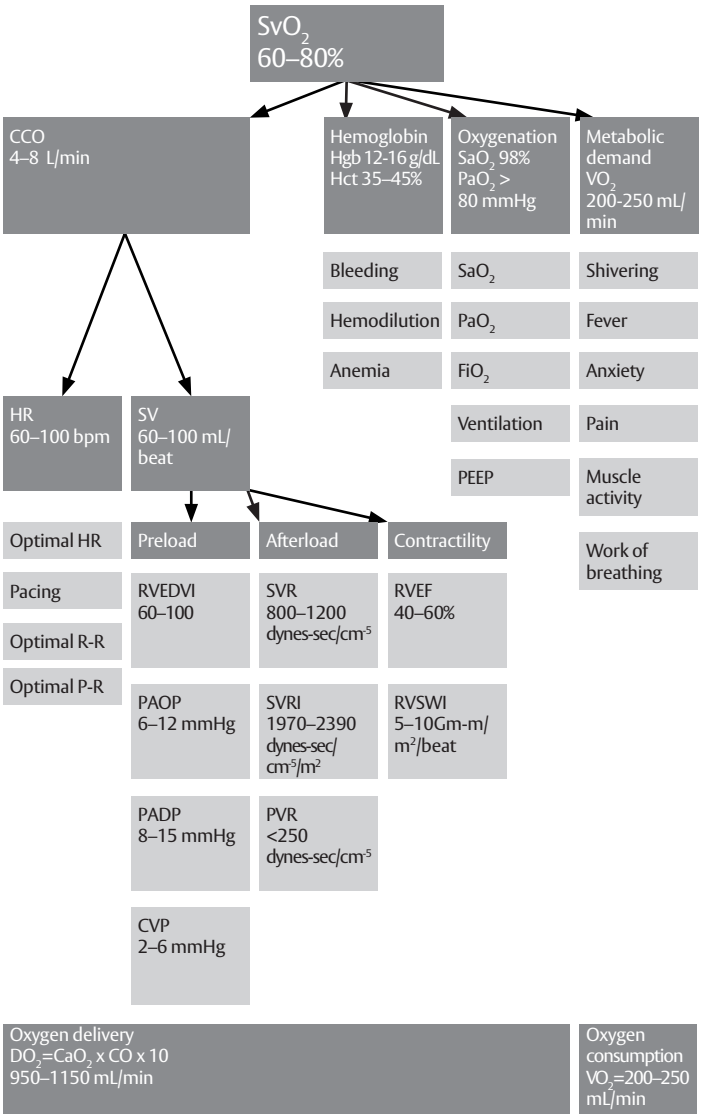
Note: The following algorithms and protocols are for educational reference only. Edwards does not endorse or support any one specific algorithm or protocol. It is up to each individual clinician and institution to select the treatment that is most appropriate.

Functional Hemodynamic Monitoring Protocol

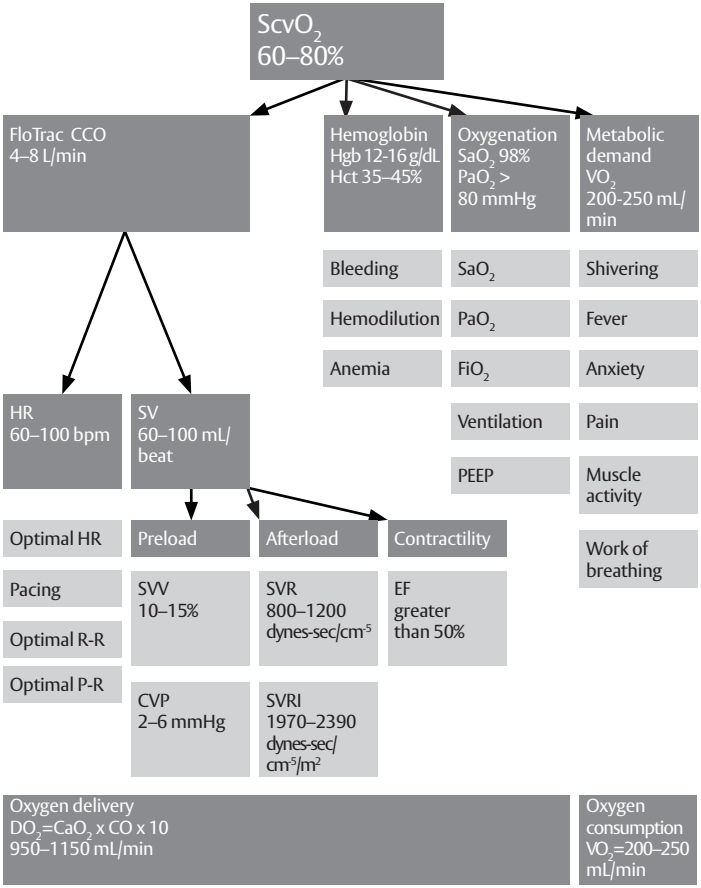


Schematic flow chart for the decision tree in the cardiovascular management of patients who are hemodynamically unstable using functional hemodynamic monitoring protocols. This protocol application is part of a patented treatment algorithm co-owned by the University of Pittsburgh and Michael R. Pinsky. MD.

Advanced Technology
Swan-Ganz Catheter Algorithm

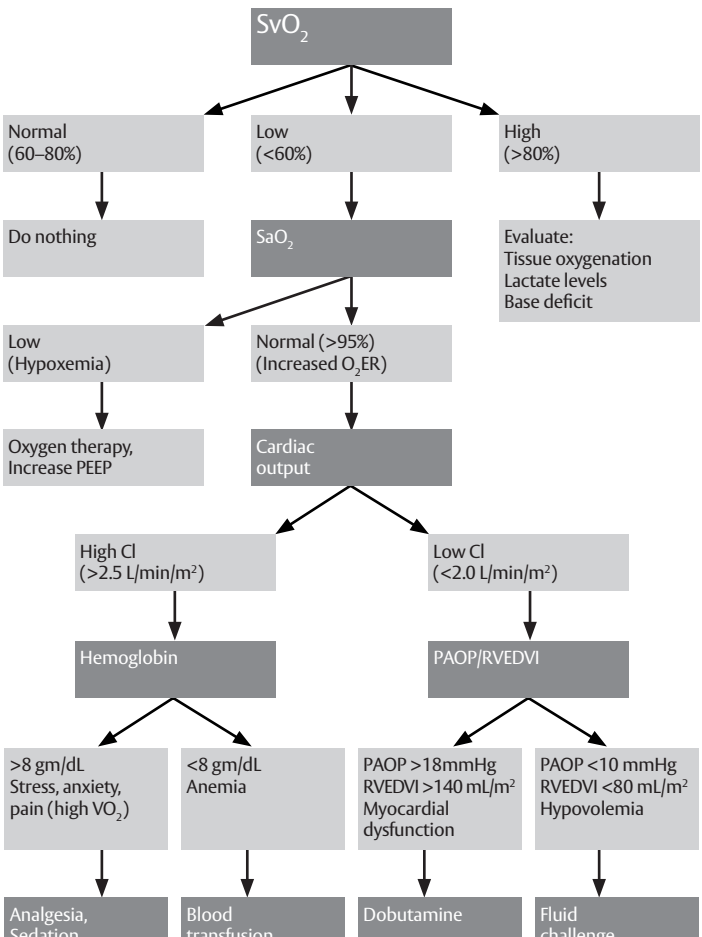


Advanced Minimally-Invasive Edwards Oximetry Catheter and FloTrac System Algorithm



Advanced Swan-Ganz Catheter Goal-Directed Protocol

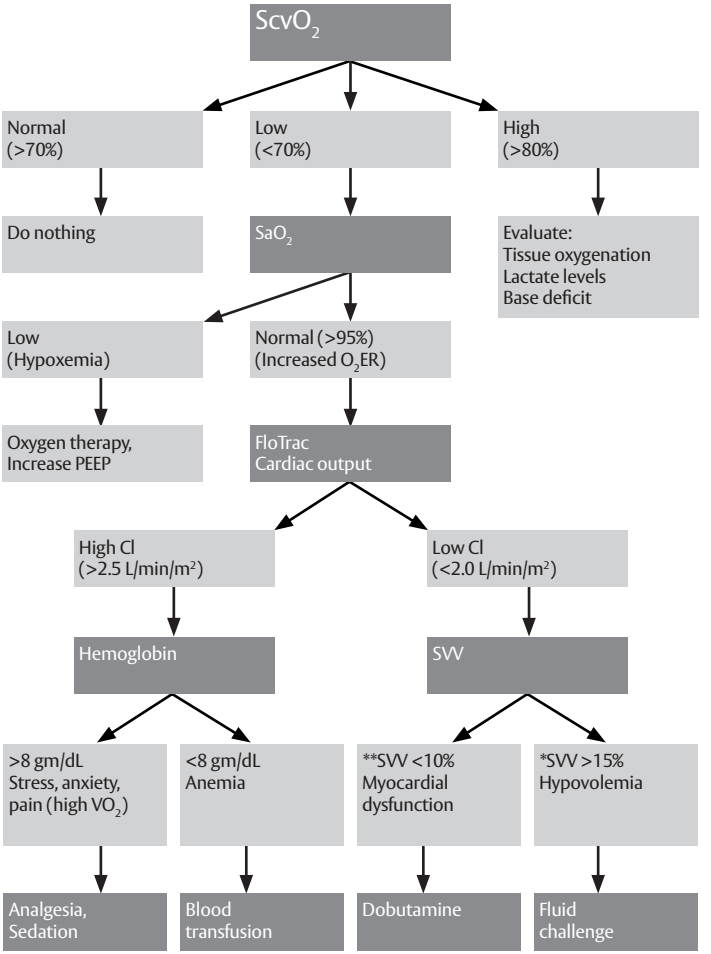
Resuscitate to a mean arterial pressure of >65 mmHg



Modified from Pinsky & Vincent. Critical Care Med. 2005;33:1119-22.

Advanced Minimally-Invasive Goal-Directed Protocol

Resuscitate to a mean arterial pressure of >65 mmHg



* 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.

Modified from Pinsky & Vincent. Critical Care Med. 2005;33:1119-22.

Normal Hemodynamic Parameters

Normal hemodynamic parameters – adult

Parameter	Equation	Normal range
Arterial Oxygen Saturation (SaO_2)		95-100%
Mixed Venous Saturation (SvO_2)		60-80%
Central Venous Oxygen Saturation ($ScvO_2$)		60-70%
Arterial Blood Pressure (BP)	Systolic (SBP) Diastolic (DBP)	90-140 mmHg 60-90 mmHg
Mean Arterial Pressure (MAP)	SBP + (2 x DBP)/3	70-105 mmHg
Right Atrial Pressure (RAP)		2-6 mmHg
Right Ventricular Pressure (RVP)	Systolic (RVSP) Diastolic (RVDP)	15-25 mmHg 0-8 mmHg
Pulmonary Artery Pressure (PAP)	Systolic (PASP) Diastolic (PADP)	15-25 mmHg 8-15 mmHg
Mean Pulmonary Artery Pressure (MPAP)	PASP + (2 x PADP)/3	10-20 mmHg
Pulmonary Artery Occlusion Pressure (PAOP)		6-12 mmHg
Left Atrial Pressure (LAP)		6-12 mmHg
Cardiac Output (CO)	HR x SV/1000	4.0-8.0 L/min
Cardiac Index (CI)	CO/BSA	2.5-4.0 L/min/m^2
Stroke Volume (SV)	CO/HR x 1000	60-100 mL/beat
Stroke Volume Index (SVI)	CI/HR x 1000	33-47 mL/m^2/beat
Stroke Volume Variation (SVV)	(SVmax - SVmin)/ SVmean x 100	10-15%
Systemic Vascular Resistance (SVR)	80 x (MAP - RAP)/CO	800-1200 dynes-sec/cm^{-5}
Systemic Vascular Resistance Index (SVRI)	80 x (MAP - RAP)/CI	1970-2390 dynes-sec/cm^{-5}/m^2
Pulmonary Vascular Resistance (PVR)	80 x (MPAP - PAOP)/CO	<250 dynes-sec/cm^{-5}

Normal hemodynamic parameters – adult (continued)

Parameter	Equation	Normal range
Pulmonary Vascular Resistance Index (PVRI)	80 x (MPAP - PAOP)/CI	255-285 dynes-sec/cm^{-5}/m^2
Left Ventricular Stroke Work Index (LVSWI)	SVI x (MAP - PAOP) x 0.0136	50-62 mmHg x mL/m^2
Right Ventricular Stroke Work Index (RVSWI)	SVI x (MPAP - CVP) x 0.0136	5-10 mmHg x mL/m^2
Coronary Artery Perfusion Pressure (CPP)	Diastolic BP-PAOP	60-80 mmHg
Right Ventricular End-Diastolic Volume (RVEDV)	SV/EF	100-160 mL
Right Ventricular End-Diastolic Volume Index (RVEDVI)	RVEDV/BSA	59-94 mL/m^2
Right Ventricular End-Systolic Volume (RVESV)	EDV-SV	50-100 mL
Right Ventricular Ejection Fraction (RVEF)	SV/EDV x 100	40-60%
Arterial Oxygen Content (CaO_2)	(0.0138 x Hgb x SaO2) + 0.003 x PaO_2	17-20 mL/dL
Venous Oxygen Content (CvO_2)	(0.0138 x Hgb x SvO_2) + 0.003 x PvO_2	12-15 mL/dL
A- V Oxygen Content Difference ($C(a\text{-}v)O_2$)	CaO_2 - CvO_2	4-6 mL/dL
Oxygen Delivery (DO_2)	CaO_2 x CO x 10	950-1150 mL/min
Oxygen Delivery Index (DO_2I)	CaO_2 x CI x 10	500-600 mL/min/m^2
Oxygen Consumption (VO_2)	$C(a\text{-}v)O_2$ x CO x 10	200-250 mL/min
Oxygen Consumption Index (VO_2I)	$C(a\text{-}v)O_2$ x CI x 10	120-160 mL/min/m^2
Oxygen Extraction Ratio (O_2ER)	(CaO_2 - Cv O_2)/CaO_2 x 100	22-30%
Oxygen Extraction Index (O_2EI)	(SaO_2 - SvO_2)/SaO_2 x 100	20-25%
Extra Vascular Lung Water (EVLW)	CO x DSt - 0.25 x GEDV	

Normal hemodynamic parameters – adult (continued)

Parameter	Equation	Normal range
Extra Vascular Lung Water Index (ELWI)	EVLW/PBW Predicted Body Weight (PBW): Female: 45.5 + 0.91 x (Height-152.4) Male : 50 + 0.91 x (Height-152.4)	0-7 mL/kg
Global End Diastolic Volume (GEDV)	CO x MTt x f(S1/S2)	
Global End Diastolic Volume Index (GEDI)	CI x MTt x f(S1/S2)	650-800 mL/kg
Global Ejection Fraction (GEF)	SV x 4 / GEDV	>20%
Cardiac Function Index (CFI)	1000 x CO / GEDV	4.5-6.5 1/min
Intra Thoracic Blood Volume (ITBV)	ITBV = 1.25 x GEDV	
Intra Thoracic Blood Volume Index (ITBI)	ITBI = 1.25 x GEDI	850-1000 mL/m^2
Pulmonary Vascular Permeability Index (PVPI)	EVLW/0.25 x GEDV	<3
Cardiac Power (CPO) Cardiac Power Index (CPI)	CO x MAP / 451 CI x MAP / 451	0.5-0.7 W/m^2

Edwards Clinical Education

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