



Quick Guide to Cardiopulmonary Care

5th Edition



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Quick Guide to Cardiopulmonary Care

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Introduction

The intent of this guide is to provide a quick reference for hemodynamic monitoring and oxygenation assessment of surgical and critically ill patients.

Critically ill patients are cared for in many parts of the hospital beyond the intensive care unit. Minimal and noninvasive monitoring techniques have become part of routine assessment and care for these patients, and can alert clinicians to changes in hemodynamic values and oxygenation status.

Because the practice of critical care medicine 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 to assist clinicians caring for critically ill patients.

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Anatomy and Physiology

Anatomy and Physiology

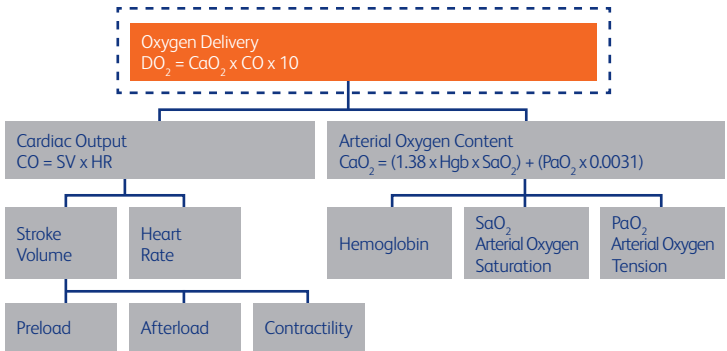
Cardiopulmonary monitoring can assist clinicians in the assessment of critically ill and surgical patients who are at risk for tissue hypoxia related to decreased oxygen delivery. Continuous cardiopulmonary monitoring allows for prompt recognition of physiologic imbalances, with the goal of optimizing tissue oxygenation. Continuous monitoring also allows for these imbalances to be observed (and intervened upon) at global, systemic, regional, and/or organ-specific levels of functioning.

Adequate tissue oxygenation depends upon oxygen delivery (DO_2) and the tissues' ability to utilize it at the cellular level, this is known as oxygen consumption (VO_2). Tissue hypoxia occurs when oxygen consumption is greater than delivery, which can be the result of poor pulmonary function, poor cardiac function, or anemia. The anatomy and physiology described in this chapter is intended to provide background that may be helpful to reference when using basic and advanced hemodynamic monitoring to maintain this balance or determine underlying causes of potential or impending imbalances.

Oxygen Delivery ($\text{DO}_2 = \text{CaO}_2 \times \text{CO} \times 10^*$)

Oxygen delivery, DO_2 , is the amount of oxygen delivered to the tissues per minute, $\text{mL O}_2/\text{min}$. It is the product of oxygen content in the blood, CaO_2 , measured in $\text{mL O}_2/\text{dL}$ of blood, and cardiac output, CO , measured in liters of blood pumped/min. Oxygen delivery is dependent upon pulmonary gas exchange and resulting oxyhemoglobin saturation, the amount of hemoglobin in the blood, and cardiac output. PaO_2 does not contribute significantly to oxygen content; therefore, its contribution to DO_2 is usually ignored.

* = conversion factor



Oxygen delivery (DO_2): milliliters of oxygen transported in the arterial blood per minute.

$$DO_2 = CaO_2 \times CO \times 10$$

DO_2 normal range: 950 - 1150 mL/min

Oxygen content (CaO_2): milliliters of oxygen in one deciliter of blood.

$$CaO_2 = (1.38^* \times Hgb \times SaO_2) + (0.0031 \times PaO_2)$$

1.38* = amount of O_2 that is carried per 1 gram of fully saturated Hgb

0.0031 = solubility coefficient of O_2 in the plasma

CaO_2 normal range: 17-20 mL/dL**

* The O_2 carrying capacity of Hgb is referenced between 1.31 - 1.39 in the literature.

** Assumes Hgb of 15 gm/dL

Oxygen Consumption $VO_2 = CO(CaO_2 - CvO_2) \times 10$

Oxygen consumption, VO_2 , refers to the amount of oxygen used by the tissues. This value cannot be measured directly, but can be assessed by measuring the difference between the arterial oxygen content proximal to the tissue (DO_2) and the venous oxygen content immediately distal to the tissue (CvO_2).

Oxygen Consumption

$$VO_2 = CO (CaO_2 - CvO_2) \times 10$$

Oxygen Delivery

$$DO_2 = CaO_2 \times CO \times 10$$

Venous Oxygen Content

$$CvO_2 = (1.38 \times Hgb \times SvO_2) + (0.0031 \times PvO_2)$$

Oxygen consumption (VO_2):

arterial oxygen content minus venous oxygen content

$$VO_2 = (CaO_2 \times CO \times 10) - (CvO_2 \times CO \times 10)$$

VO_2 normal range: 200 - 250 mL/min/m²

Venous oxygen content (CvO_2):

$$CvO_2 = (1.38 \times Hgb \times SvO_2) + (0.0031 \times PvO_2)$$

1.38 = amount of O_2 that is carried per 1 gram of fully saturated Hgb

0.0031 = solubility coefficient of O_2 in plasma

CvO_2 normal range: 12-15 mL/dL

Factors Affecting DO_2 and VO_2 *

Decreased DO_2	Increased VO_2 or O_2ER
↓ Cardiac Output (CO) ↓ Hemoglobin/Anemia ↓ SaO_2 / Hypoxemia	Seizures Shivering Pain Hyperthermia ↑ Work of breathing Bed bath
Increased DO_2	Decreased VO_2 or O_2ER
↑ Cardiac Output (CO) ↑ Hemoglobin/Red blood cell transfusion ↑ FiO_2	Hypothermia Anesthesia ↓ Pain / Pain control measures

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

Other Assessment Parameters for Oxygen Utilization

Arterial-venous oxygen difference

$C(a-v)O_2$: normal range 4-6 mL/dL

Oxygen extraction ratio

$O_2ER: [(CaO_2 - CvO_2)/CaO_2] \times 100$

O_2ER : normal range 22-30%

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 $C(a-v)O_2$. Mixed venous oxygen saturation, SvO_2 , reflects the balance between oxygen delivery and oxygen consumption based upon the following equations.

$$CO = VO_2 / CaO_2 - CvO_2$$

$$C(a-v)O_2 = VO_2 / (CO \times 10)$$

$$VO_2 = C(a-v)O_2 \times CO \times 10$$

$$S(a-v)O_2 = VO_2 / (CO \times 10)$$

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 .

VO₂/DO₂ Relationships

Oxygen consumption (VO₂) is determined by the metabolic demands at the cellular level. Normally, oxygen delivery (DO₂) is approximately four times the amount of oxygen consumed (VO₂ is about 25% of DO₂). This has been referred to as delivery independent VO₂, meaning consumption is independent of delivery. If oxygen delivery decreases or cellular oxygen demands increase, a larger percentage of oxygen can be extracted from hemoglobin to meet metabolic demands; however, when oxygen consumption exceeds oxygen delivery, it becomes delivery-dependent. In the setting of delivery dependence, oxygen supply is inadequate to meet metabolic demands and anaerobic metabolism occurs. Therefore, continuous monitoring of SvO₂ (measured by a catheter placed in the pulmonary artery) and/or StO₂ (measured by sensors placed directly on the skin; see chapter 3 for details on tissue oximetry) allows for continuous assessment of the balance between oxygen delivery and consumption.

Functional Anatomy

For hemodynamic monitoring purposes, the right and left heart are differentiated by function, structure, and pressure generation. The circulatory system is two circuits in series, pulmonic and systemic. The pulmonic circuit (low pressure) is comprised of the right heart and the highly compliant pulmonary capillary bed. The systemic circuit (high pressure) is comprised of the left heart, coronary arteries, and systemic vasculature.

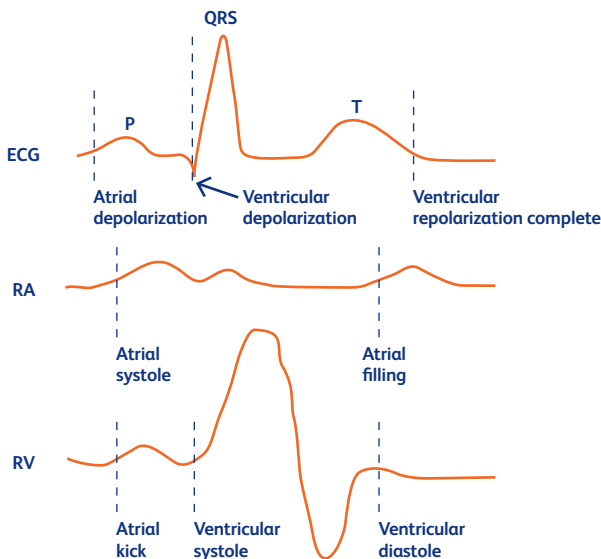
The autonomic activation of the cardiac electrical conduction system activates the mechanical structures of the heart (pump) to move volume through the circulatory system.

Cardiac Cycle: Correlating Electrical and Mechanical Activation

The activation of the electrical cardiac cycle occurs just 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, resulting in systole.

Next, repolarization occurs, promptly followed by relaxation of cardiac muscle fibers, resulting in 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 tracings shows that the electrical waveform activation precedes the mechanical waveform. Electrical activity is referred to as heart rate (HR) and mechanical activity is referred to as pulse rate (PR).

Electrical – mechanical cardiac cycle



Mechanical Cardiac Cycle Phases

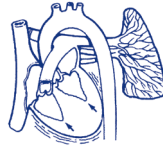
Ventricular Systole

1 Isovolumetric phase

Follows QRS of ECG

All valves closed

Majority of oxygen consumed

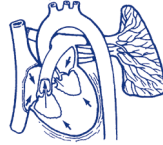


2 Rapid ventricular ejection

Aortic and pulmonic valves open

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



Ventricular Diastole

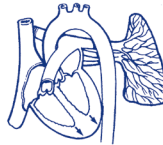
1 Isovolumetric relaxation

Follows "T" wave

All valves closed

Ventricular pressure declines further

LV pressure dips below LA pressure

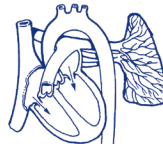


2 Rapid ventricular filling

A-V valves open

Approximately 70% of blood volume

goes into ventricle



3 Slow filling phase: end-diastole

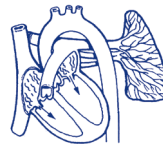
Atrial "kick"

Follows "P" wave during sinus rhythms

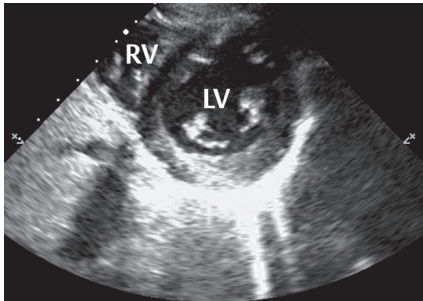
Atrial systole occurs

Produces "a" wave on atrial tracings

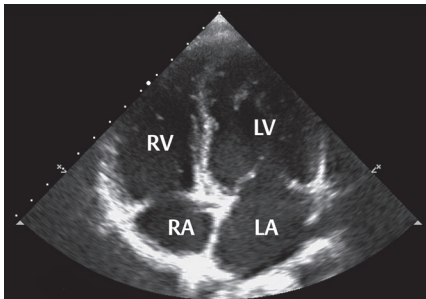
Remaining volume goes into ventricle



The figures below show echocardiographic views of the heart that illustrate key cardiac structures:

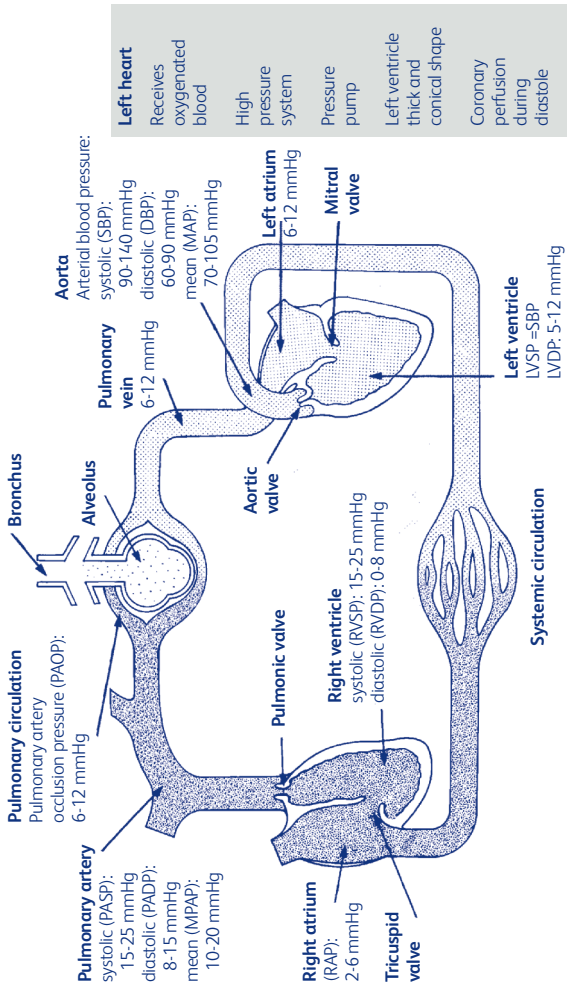


Short axis view at papillary muscle level: note the difference in wall thickness of the left ventricle (thick muscle = strong pump to generate ejection force) vs. right ventricle (thinner muscle = compliant, can accept varying amounts of volume from the venous circulation).

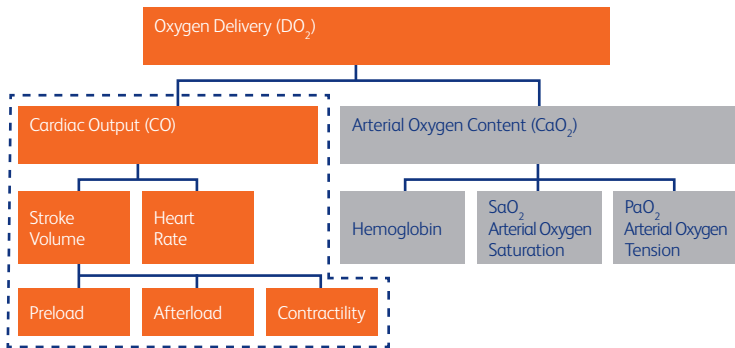


Apical four chamber view: notice the size difference between left and right heart. Muscular left sided chambers facilitate large volume of blood to be ejected forcefully to adequately distribute oxygenated blood through the systemic circuit. The less muscular right-sided chambers are more compliant to accommodate volume and pump blood into the low pressure pulmonary circuit.

Anatomical structures and associated pressure normal ranges



Right heart	Left heart
Receives deoxygenated blood	Receives oxygenated blood
Low pressure system	High pressure system
Volume pump	Pressure pump
Right ventricle thin and crescent shape	Left ventricle thick and conical shape
Coronary perfusion during systole and diastole	Coronary perfusion during diastole



Cardiac Output Definition and Components

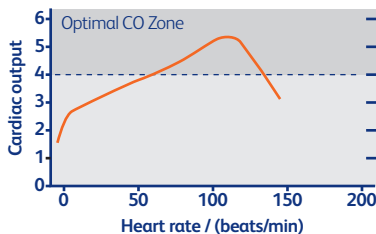
Cardiac output (CO) is the product of HR multiplied by SV, and is the amount of blood ejected from the ventricle per minute.

$$CO = HR \times SV / 1000^*$$

CO normal range: 4 - 8 L/min

Heart Rate (HR), the number of electrical depolarizations that stimulate the ventricles to contract per minute. HR and pulse rate (PR) may be used interchangeably depending on the signal source. ECG source = HR, arterial line waveform source = PR.

When the heart rate is slow, or bradycardic, CO is suboptimal. When the heart rate is high, or tachycardic, ventricular filling time is limited resulting in a suboptimal stroke volume and decreased CO (see image above).



Heart rate (HR) = beats/min

HR normal range: 60 - 100 bpm

Stroke volume (SV) is the difference between End-diastolic volume (EDV) – End-systolic volume (ESV); or volume of blood ejected from the ventricle per beat.

SV = EDV - ESV

SV normal range: 60-100 mL/beat

Indexed values

Stroke volume index (SVI) and cardiac index (CI) can be calculated when SV and CO are known. Indexed values are consistent among the population and are independent of a given patients size. By dividing SV and/or CO by a given patients body surface area (BSA, measured in meters²), the value becomes 'indexed'. BSA can be obtained from nomogram tables of height and weight.

Stroke Volume Index (SVI) = SV/BSA

Normal SVI: 33 - 47 mL/m²/beat

Cardiac Index (CI) = CO/BSA

Normal CI: 2.5 - 4 L/min/m²

Ejection Fraction (EF) is the percentage of the volume of blood that is ejected from the ventricle during systole, thus it is the ratio of EDV and SV.

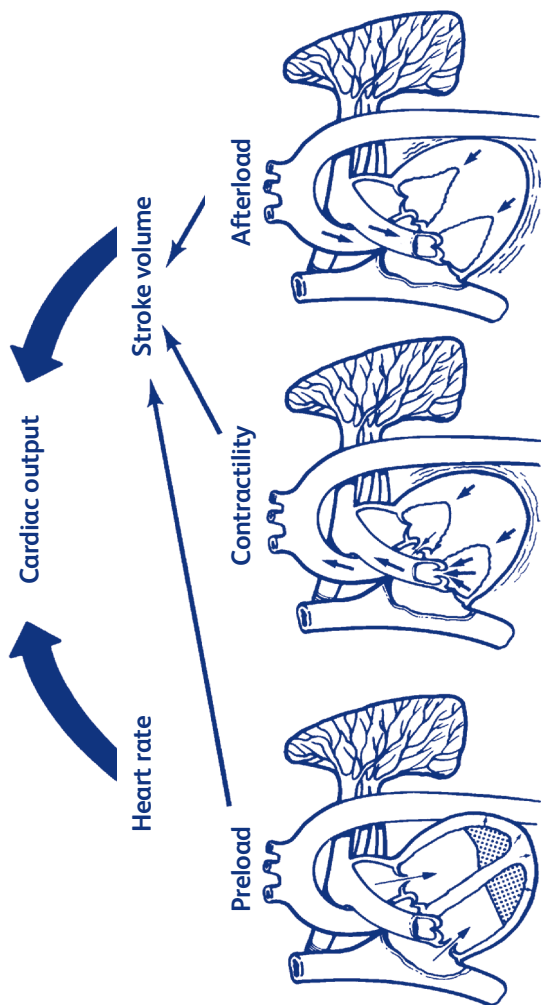
EF = SV/EDV x100

Normal ranges:

RVEF = 40 - 60%

LVEF = 50 - 70%

Determinants of stroke volume



The diagram above shows the three determinants of SV: preload, contractility, and afterload.

Preload Definition and Measurements

Preload refers to the amount of myocardial fiber stretch at the end of diastole, which is determined by the end diastolic volume (EDV) in the right (RVEDV) or left (LVEDV) ventricle. EDV is affected by the volume status and the patient's heart rhythm. Sinus rhythm maximizes EDV, while uncoordinated atrio-ventricular contractions, like those that occur during atrial fibrillation or ventricular nodal rhythms, lack preload optimization.

Right-sided pressure measurements have historically been used as an indirect assessment of ventricular preload. Pulmonary artery occlusion pressure (PAOP) and left atrial pressures (LAP) have historically been used to evaluate left ventricular preload. Right atrial pressure (RAP) and central venous pressure (CVP) have historically been used to assess right ventricular preload.

Any pressure measurement is dependent upon ventricular compliance, which is not only variable but dynamic, especially in the critically ill. For this reason, pressure measurements are unreliable assessments of preload. Volumetric parameters such as right ventricular end-diastolic volume (RVEDV) and left ventricular end-diastolic volume (LVEDV) are preferred preload measures as they eliminate the influence of ventricular compliance on pressure.

Dynamic measures of preload responsiveness, such as stroke volume variation (SVV), or changes in stroke volume (Δ SV) in response to a fluid challenge, provide reliable clinical guidance regarding the utility of additional volume therapy to aid perfusion and oxygen delivery.

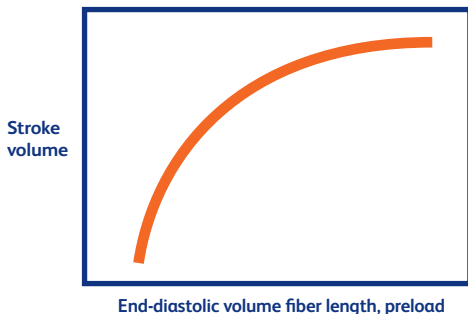
A patient is considered preload responsive if SVV, as assessed within its limitations, is $>12\%$ or there is a $>10\%$ increase in stroke volume (Δ SV) after a fluid bolus.

More details on assessing preload/fluid responsiveness can be found in chapter 5 of this guide.

Frank-Starling Law

Frank and Starling identified the relationship between myocardial fiber length and force of contraction. Increased end-diastolic volume causes increased myocardial fiber stretch, resulting in a stronger subsequent systolic contraction and a larger stroke volume, up to a certain point. This effect has limits, as overly stretched fibers can cause a weaker systolic contraction which results in a smaller stroke volume.

Frank-Starling curve



Ventricular compliance curves

The relationship between end-diastolic volume and end-diastolic pressure is dependent upon ventricular compliance. The relationship between the two is curvilinear. With normal compliance, relatively large increases in volume create relatively small increases in pressure (see graphic on next page). This will occur in a ventricle that is not completely volume loaded. When normal ventricles approach their maximum volume, they become less compliant, and thus, smaller increases in volume produce a greater rise in pressure.

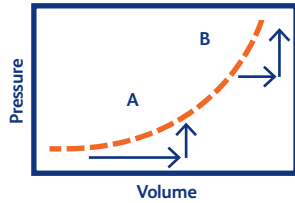
For example, increased compliance of the ventricle is oftenseen in patients with heart failure that have a low ejection fraction. In these patients there can be large changes in volume, or preload, with little resulting rise in pressure. These often subtle changes in ventricular compliance may not be overtly noticeable at the bedside, especially if single point in time measurements are being used to evaluate preload.

Effects of ventricular compliance

Normal compliance

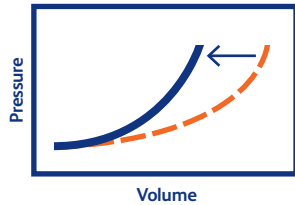
Pressure/volume relationship is curvilinear:

- A** Large increase in volume = small increase in pressure
- B** Small increase in volume = large increase in pressure



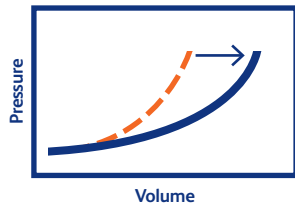
Decreased compliance states:

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



Increased compliance states:

Dilated cardiomyopathies
Systolic dysfunction
Decreased afterload due to vasodilators

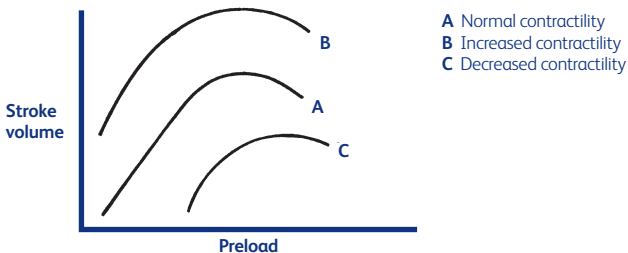


Contractility Definition and Measurements

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.

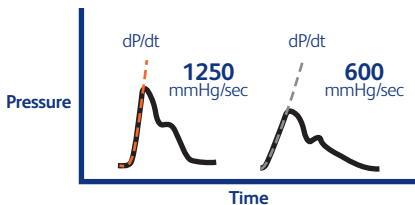
Contractility changes can be plotted on curves known as ventricular function curves, which represent the Frank-Starling mechanism of the heart.

Ventricular function can be represented by plots on the ventricular function curves. Cardiac contractility can be implied from these curves, comparing varying states of preload, ventricular compliance, or afterload.



Systolic slope (dP/dt)

Changes in pressure over time (dP/dt), also known as systolic slope, can be used at the bedside as a continuous indicator of contractility and is measured in mmHg/sec.



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 includes the gradient difference between the beginning or inflow of the circuit and the end or outflow of the circuit.

Afterload

Systemic vascular resistance (SVR): 800 -1200 dynes-sec/cm⁵

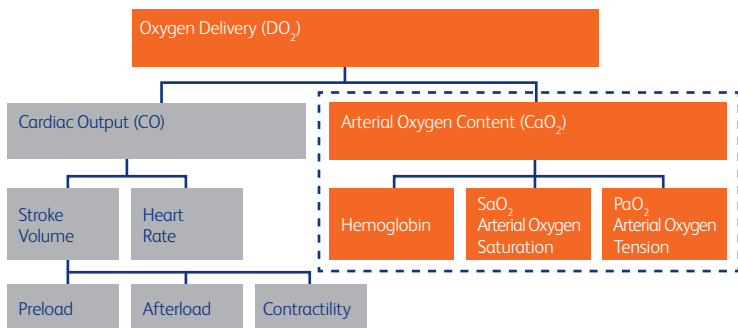
$$\text{SVR} = \frac{\text{MAP} - \text{RAP}}{\text{CO}} \times 80$$

Pulmonary vascular resistance (PVR): <250 dynes-sec/cm⁵

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

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

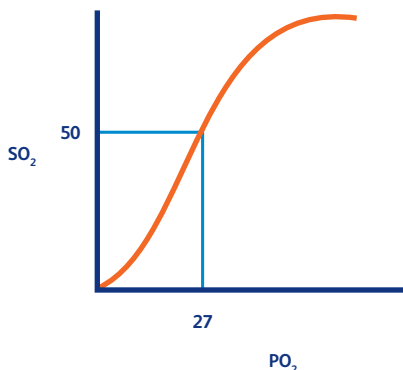
Additionally, $E_{a_{\text{dyn}}}$ (dynamic arterial elastance) is a measure of the systemic arterial elastance (i.e. afterload) on the left ventricle, and is calculated as a ratio of PPV / SVV. Refer to chapter 7 of this guide for more details on $E_{a_{\text{dyn}}}$.



Oxyhemoglobin Dissociation Curve

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

Normal oxyhemoglobin dissociation curve

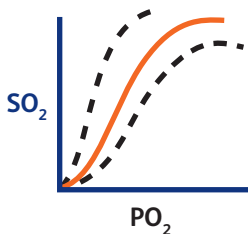


Factors shifting oxyhemoglobin dissociation curve

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

Leftward shift:

Increased affinity of Hgb for O_2 ,
 $\uparrow$ pH, Alkalosis,
 Hypothermia,
 $\downarrow$ 2, 3 DPG



Rightward shift:

Decreased affinity of Hgb for O_2 ,
 $\downarrow$ pH, Acidosis,
 Hyperthermia,
 $\uparrow$ 2, 3 DPG

Pulmonary gas exchange equations

Assessing pulmonary alveolar-capillary gas exchange 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 alveolar-capillary unit, and to determine the amount of intrapulmonary shunting. An alteration in any of these will impact oxygen delivery.

In the alveolar gas equation PAO_2 is the alveolar PO_2 and is calculated based on the following equation:

$$PAO_2 \text{ (partial pressure of oxygen in the alveoli)} = [(PB - PH_2O) \times FiO_2] - PaCO_2 / 0.8$$

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

PB: Atmospheric pressure at sea level is: 760 mmHg

PH_2O : Partial pressure of water vapor: 47 mmHg

FiO_2 : Fraction of inspired oxygen (% O_2 in the inhaled gas)

$PaCO_2$: Partial pressure of CO_2 in the blood, measured from arterial blood gas

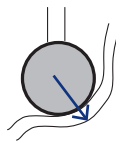
PaO_2 : Partial pressure of O_2 in the blood, measured from arterial blood gas

0.8: Respiratory quotient (VCO_2 / VO_2)

$P(A-a)O_2$: 'A-a' gradient is measured to assess the amount of O_2 diffusion across the alveolar-capillary unit

Normal 'A-a' gradient on room air: <15 mmHg

Abnormal 'A-a' gradient on room air: >15 mmHg is indicative of lung disease



Intrapulmonary Shunt

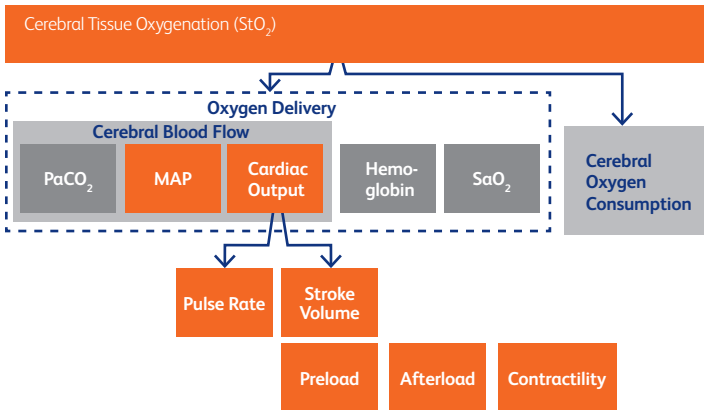
Intrapulmonary shunt (Q_s/Q_t) is defined as the amount of venous blood (approximately 1-2% in normal patients) that bypasses an alveolar-capillary unit and does not participate in oxygen exchange (Q_s represents shunt and Q_t represents flow).

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. See page 9.8 for shunt calculation.

Cerebral Oxygen Delivery and Consumption

Cerebral perfusion and oxygenation are dependent on global oxygen transport but influenced regionally by the diameter of the vasculature and intracranial pressure. Cerebral perfusion pressure is the difference between mean arterial pressure (MAP) and intracranial pressure (ICP), and can be manipulated via native physiology or through clinical interventions. MAP is proportional to cardiac output and determined by vascular tone (SVR). ICP is proportional to cerebral blood flow and depends on cerebral vascular compliance and resistance. Cerebral blood flow can be altered by changes in MAP, especially when cerebral autoregulation is impaired. When not impaired, it can be influenced by changes in PaCO_2 and PaO_2 (see graph on page 1.23).

Imbalances in cerebral tissue oxygenation (StO_2) can be assessed through the monitoring and analysis of PaCO_2 , MAP, CO, Hgb, and SaO_2 to help identify the root cause behind an imbalance.

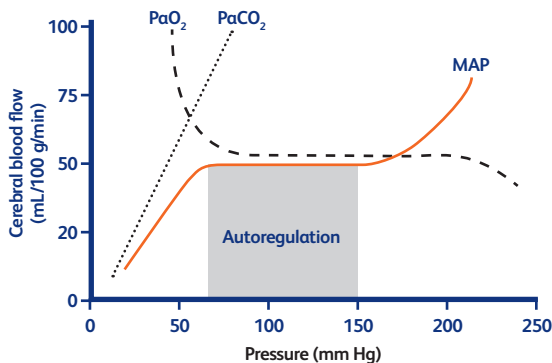


Maintaining the balance between oxygen delivery and consumption to the brain is critical for brain health. Cerebral oxygen delivery is unique in that it is affected by more than just the previously mentioned elements of oxygen delivery (CO , Hgb , O_2). Cerebral tissue oximetry (StO_2) allows for the continuous monitoring of the balance between cerebral oxygen delivery and consumption, because the blood that flows beneath the region where the sensor is placed on the forehead is composed of mostly venous ($\sim 70\%$) blood.

Autoregulation of blood flow

Many tissues in the body have autoregulation of blood flow, that is they maintain a near-constant flow over a wide range of perfusion pressures. The figure below illustrates that for the brain, blood flow is held virtually the same over a range of perfusion pressures from ~ 55 - 150 mmHg. When MAP is exceedingly high or low, it can supercede the brain's ability to auto-regulate blood flow. Rapid changes in cerebral StO_2 values can serve as an indication that MAP, and therefore, autoregulation may be compromised. The brain is unique in its autoregulation capacity in that global blood flow can be diverted from other tissues to maintain cerebral oxygenation.

Cerebral autoregulation factors



Summary

Continuous monitoring of the balance between oxygen delivery and consumption at the organ-specific, regional, systemic, and/or global level can provide clinicians valuable insights as to possible root causes when imbalances arise. The following chapters will discuss technologies available from BD, which may be deployed to provide clinicians with advanced hemodynamic measures for monitoring and maintaining these important balances.

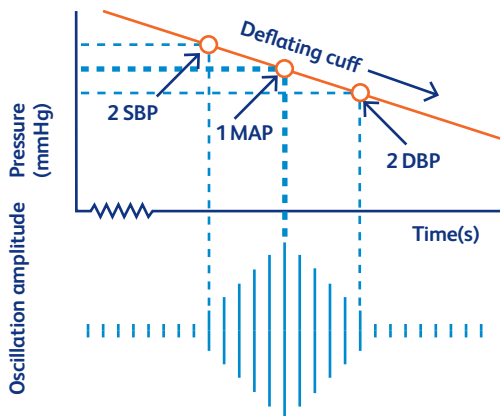


Basic Pressure Monitoring

Oscillometric Pressure Monitoring

Oscillometric blood pressure monitoring is standard of care in the clinical environment. Blood pressure measurement using oscillometry is commonly performed on the upper arm with a reusable cuff. The arm cuff is comprised of an inflatable bladder and pressure sensor. Intermittent arterial blood pressure measurements are generated as the cuff inflates to a pressure that is above the systolic pressure to occlude blood flow of the artery and slowly deflates to a pressure that is below the diastolic pressure, recording the returning pressure pulsations. The sensed oscillations create a pattern of pulsations known as an oscillometric pressure waveform. The maximal recorded pressure oscillation (pulse amplitude) identifies the mean arterial pressure (MAP). With the use of independent propriety estimation algorithms, systolic and diastolic pressures are derived from the MAP.

Oscillometric cuff pressure



- 1 MAP is calculated first
- 2 SBP and DBP are then extrapolated

While monitoring blood pressure detects deleterious conditions such as hypo- and hypertension, intermittent oscillometric blood pressure measurements may function poorly in dynamic states. Oscillometric measurements have shown to overestimate readings in the setting of hypotension and underestimates it in the setting of hypertension. Additionally, improper arm cuff sizing can result in erroneously high or low measurements.

Common oscillometric blood pressure cuff limitations include:

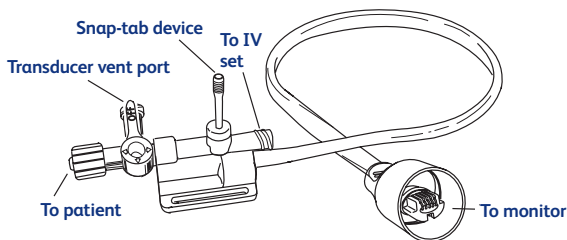
- Heart arrhythmias
- Dynamic changes in blood pressure
- Shock (low pressure states)
- Obese patients
- Arterial stiffness
- Movement from measured extremity

Continuous, beat-to-beat blood pressure monitoring helps minimize periods of hyper- and hypotension by providing clinicians with measurements at more frequent intervals. This in turn can lead to earlier recognition and earlier potential intervention. Continuous blood pressure measurements have been recognized as a means to reduce hypotension when compared to intermittent oscillometric measurements.

Physiologic Pressure Monitoring

Pressure monitoring is a basic tool for clinicians monitoring surgical and nonsurgical patients. Disposable pressure transducers (DPT) convert a mechanical physiologic signal (arterial pressure, central venous pressure, pulmonary artery pressure, etc.) to an electrical signal which is amplified, filtered, and displayed on a bedside physiologic monitor in both a waveform and numeric value in mmHg.

TruWave™ Disposable Pressure Transducer



Components of a physiologic pressure measurement system

- Invasive catheter
- Transducer kit
 - TruWave™ Disposable Pressure Transducer
 - Semi-rigid pressure tubing
 - Stopcocks
 - 3 mL/hr flush device
 - Compatible monitor cable
 - IV set
 - Disposable holder
 - Arm strap
- IV flush solution (500 or 1000 mL)
- Pressure infusion bag (appropriately sized for flush solution bag)
- Reusable pressure cable specific to TruWave™ Disposable Pressure Transducer
- Bedside physiologic monitor

Correct setup based on the manufacturer's instructions for use is crucial for obtaining accurate pressure readings from a physiologic pressure transducer system.



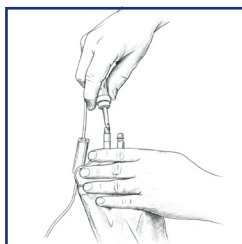
Scan the QR code to view the TruWave™ Disposable Pressure Transducer setup video-Priming the system



Scan the QR code to view the TruWave™ Disposable Pressure Transducer setup video-Calibrating the system

Setting up a physiologic pressure measurement system for intravascular monitoring

- 1 Ensure that the cable is compatible with the monitor being used. Connect the reusable cable to the monitor and turn monitor on.
- 2 Using aseptic technique, remove the transducer and kit from the sterile packaging, inspecting contents to ensure all connections are secure.
- 3 To de-air and prime IV flush bag and pressure transducer:
Invert the IV flush solution bag (anticoagulation per institutional policy). Spike IV bag with fluid administration set, keeping the drip chamber upright. While keeping IV bag inverted, gently squeeze air out of 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 at least half way or full.

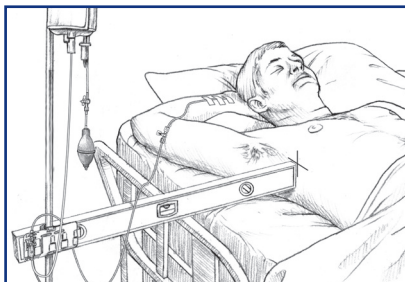


Ensure IV bag is inverted while spiking the IV fluid bag

- 4 Insert IV flush solution bag into pressure infuser bag (**do not inflate**) and hang on IV pole approximately 2 feet (60 cm) above the transducer.
- 5 With gravity only (no pressure in the pressure bag), flush the transducer, holding pressure tubing in an upright position. Flushing under pressure creates turbulence and increases occurrence of bubbles.
- 6 Pressurize the pressure bag until it reaches 300 mmHg, turn stopcock toward the pressure bag to lock off air pressure and maintain consistent pressure on fluid bag.
- 7 Fast-flush transducer tubing while tapping on the tubing and stopcocks to remove any residual bubbles.
- 8 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.
- 9 Connect non-disposable pressure cable that is compatible with the bedside monitor to the disposable pressure transducer and bedside monitor.
- 10 Connect tubing to the arterial or venous catheter. Aspirate and flush the system to ensure catheter is intra-vascular and remove residual bubbles.
- 11 Level the stopcock just above the TruWave™ Transducer to the phlebostatic axis (level of the heart).

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, see next page for additional details).
- 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 the non-vented cap, turn stopcock off to the patient.
- 6 Initiate “zero” function on the 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.

Measuring technique

Hydrostatic zero reference

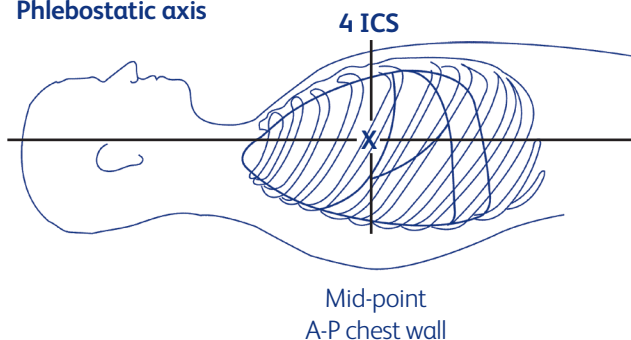
To obtain accurate pressure measurements, the level of the air-fluid interface must be aligned with the chamber or vessel being measured.

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 tilted up at 45-60 degrees as long as the zeroing stopcock has been aligned with the phlebostatic axis.

Phlebostatic axis

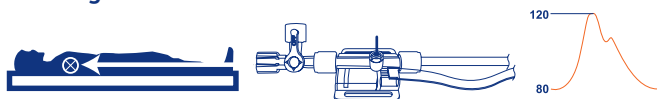


Impact of improper leveling on pressure readings

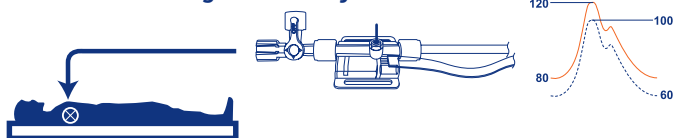
Intravascular pressure readings may have error introduced if alignment with the phlebostatic axis is not maintained. The amount of error introduced is dependent upon the degree of offset.

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.

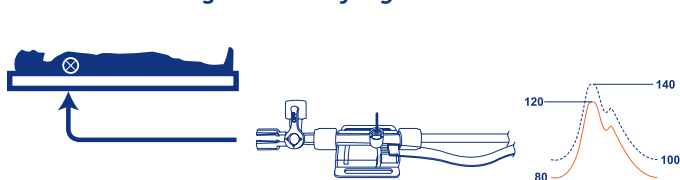
**Heart aligned with transducer =
0 mmHg error**



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



**Heart 10" (25 cm) *higher* than transducer =
Pressure 20 mmHg erroneously *high***



dotted line = erroneous measurement
solid line = correctly aligned measurement

Waveform fidelity and optimal frequency response

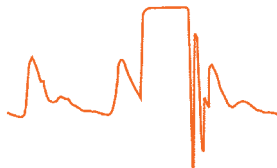
Optimal pressure monitoring requires a pressure system that accurately reproduces the physiologic signals applied to it. A simple evaluation of dynamic response can be obtained by performing a square wave test and by observing the resultant oscillations. In order to perform this assessment accurately, rapidly pull and release the Snap-Tab. All physiologic pressure transducers are damped. Optimal damping results in a waveform and displayed value that is physiologically correct.

Square wave testing

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

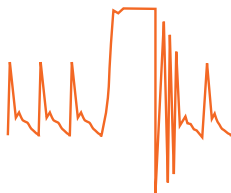
Optimally damped

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



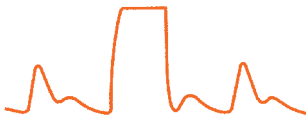
Underdamped

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



Overdamped

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



Maintaining physiologic pressure transducer system

- **Keep transducers level**

Re-level transducer whenever the patient's height or position changes in relation to the transducer

- **Re-zero transducer**

Periodic zeroing of the 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 $< \frac{1}{4}$ 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

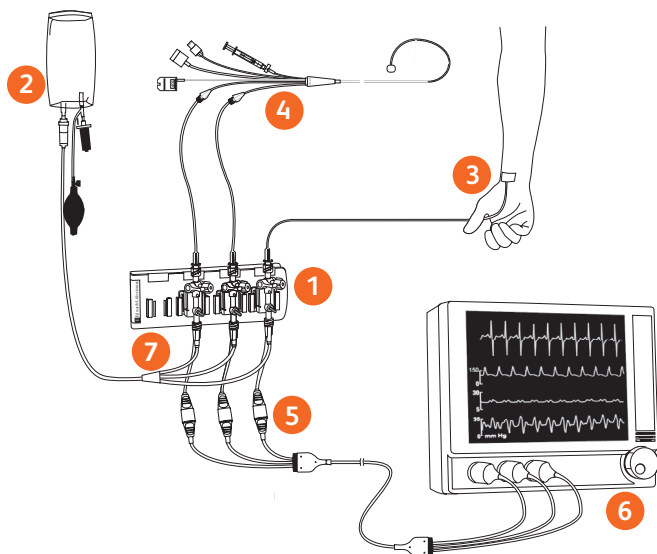
- **Changing system frequency**

Centers for Disease Control recommends changing disposable transducer flush systems, tubing and flush solutions every 96 hours

Pressure monitoring systems

The schematic below identifies the components of a standard pressure monitoring system. The Swan-Ganz™ Catheter and arterial catheter can be attached to a pressure monitoring line. The tubing must be semi-rigid 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™ Disposable Pressure Transducer
- 2 IV flush solution in pressurized infuser bag
- 3 Radial arterial line
- 4 Swan-Ganz™ Catheter PA and RA ports
- 5 Trifurcated TruWave™ Pressure Cable
- 6 Bedside monitor
- 7 Trifurcated fluid administration line

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

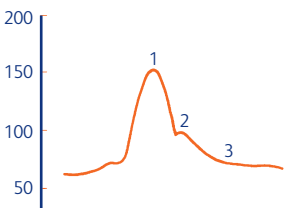
Dicrotic notch: reflects closure of the aortic valve, marking the end of systole and the onset of diastole

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

Pulse pressure: difference between systolic and diastolic pressure

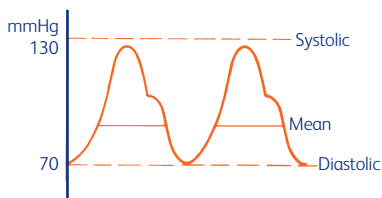
Mean arterial pressure (MAP): average pressure in the arterial system during a complete cardiac cycle. On the standard bedside monitor, invasive MAP is computed using the area under the curve within each cardiac cycle.

Components of an arterial pulse



- 1 Peak systolic pressure
- 2 Dicrotic notch
- 3 Diastolic pressure

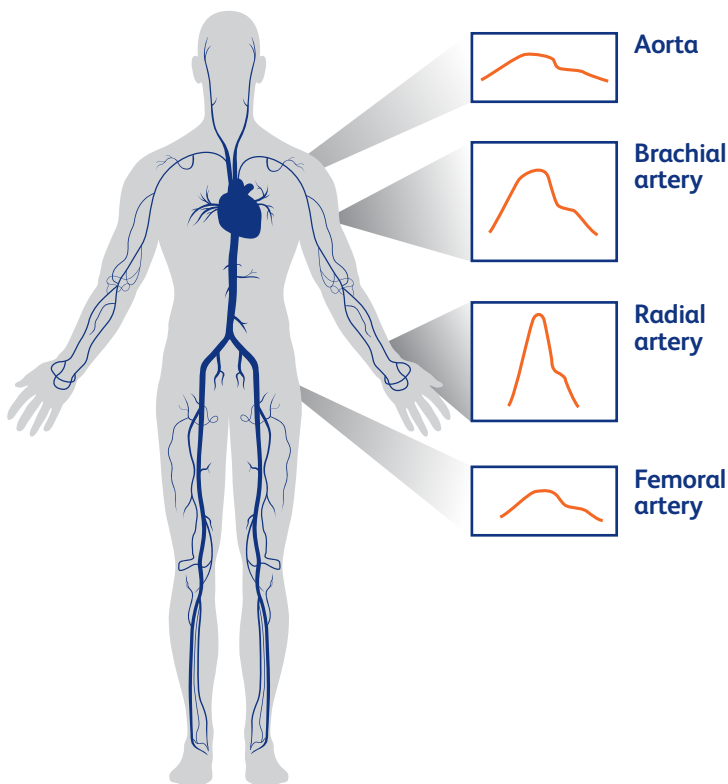
Mean arterial pressure



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

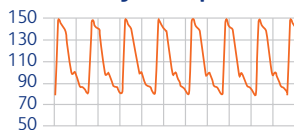
Arterial pressure waveform morphology differs by measurement site

With each contraction of the heart, an incidental pressure waveform is created as blood moves forward in the systemic circulation. As the blood moves through the narrowing peripheral arterial vasculature, a backward reflective waveform is generated, increasing the systolic and pulse pressure of the original arterial waveform. The occurrence can be noted when blood measurements are obtained at various points in the arterial vasculature (see image below: brachial vs. radial, etc.).



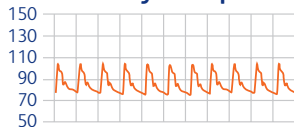
Abnormal arterial pressure waveforms

Elevated systolic pressure*



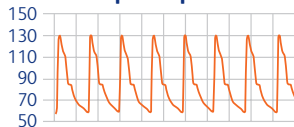
Systemic hypertension
Arteriosclerosis
Aortic insufficiency

Decreased systolic pressure*



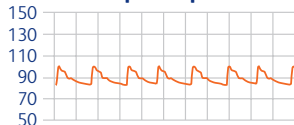
Aortic stenosis
Heart failure
Shock

Widened pulse pressure*



Vasodilation
Aortic insufficiency
Shock

Narrowed pulse pressure*



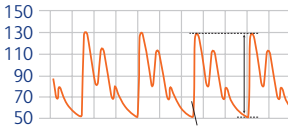
Cardiac tamponade
Cardiogenic shock

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

Note: this is not an exhaustive list of causes

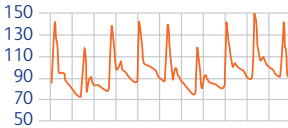
Abnormal arterial pressure waveforms *(continued)*

Pulsus bisferiens*



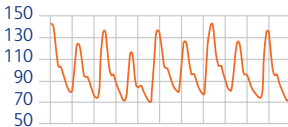
Aortic insufficiency
Obstructive hypertrophic
cardiomyopathy

Pulsus paradoxus*



Cardiac tamponade
Hypovolemia

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

A central venous catheter (CVC) is one whose tip resides in the central circulation. There are many types: tunneled, non-tunneled / percutaneously inserted, peripherally inserted, and implanted. CVCs can be single or multi-lumen.

Introducers are used to direct or 'introduce' 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.

Monitoring central venous pressure

Historically, CVCs have been used to measure the pressure under which the blood is returned to the right atrium and to give an assessment of intraventricular volume and right heart function. Normal range for CVP measurements are 2-6 mmHg. It is worth noting that continuous values trended over time are more useful than a single reading.

There are many factors that influence CVP values: cardiac performance, blood volume, vascular tone, intrinsic venous tone, increased intra-abdominal or intrathoracic pressures, catheter position, and vasopressor therapy. Therefore, using CVP to assess either preload or volume status of the patient may be unreliable (see page 1.14 for more details on preload measures).

Physiologic factors affecting CVP pressures

Increased CVP

- Increased venous return and hypervolemia
- Depressed cardiac function
- Cardiac tamponade
- Pulmonary hypertension
- PEEP
- Vasoconstriction

Decreased CVP

- Decreased venous return and hypovolemia
- Loss of vascular tone caused by vasodilation (as seen in sepsis) contributing to venous pooling

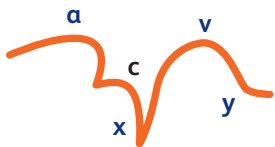
Normal CVP waveform

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 pressure. The v wave represents the atrial events during ventricular contraction – passive atrial filling – and follows the T wave on the ECG. When the atrial pressure is sufficient, the tricuspid valve opens, and the y descent occurs.

Right atrium



“a” = atrial contraction

“c” = closure of tricuspid valve

“v” = passive atrial filling

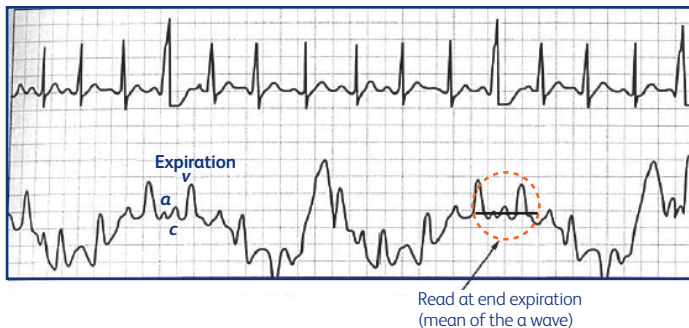
“x” = atrial diastole

“y” = atrial emptying

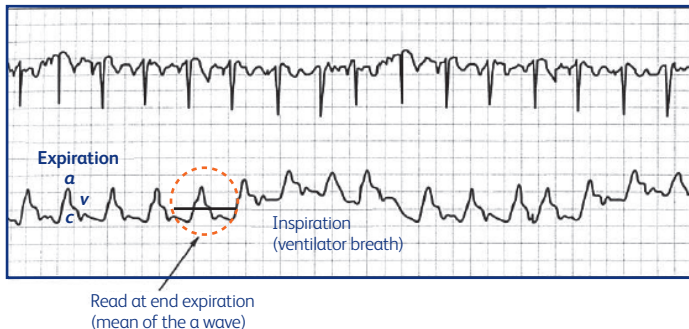
Accurate recognition of these waves requires that they be aligned with an ECG tracing, as mechanical events follow electrical events. The waveforms can be identified by lining them up with the ECG events.

Measuring CVP pressures with spontaneous vs. mechanical ventilation

Spontaneous ventilation (negative pressure)



Mechanical ventilation (positive pressure)





Noninvasive Monitoring



Noninvasive Monitoring

Advanced hemodynamic measurements can be obtained with noninvasive technologies, allowing application to a broader patient population. BD offers three noninvasive sensor technologies.

Acumen IQ™ and ClearSight™ Finger Cuff are both noninvasive arterial blood pressure and cardiac output sensor technologies that enable continuous beat-to-beat monitoring. Acumen IQ™ Finger Cuff builds on the legacy of ClearSight™ Finger Cuff with the addition of Acumen Hypotension Prediction Index™ (HPI™) Software, which unlocks additional advanced parameters and features.

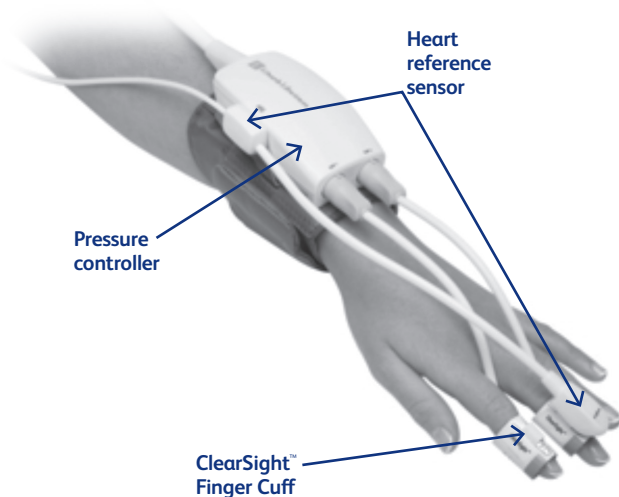
The third noninvasive sensor technology is ForeSight™ Tissue Oximetry Sensor, enabling cerebral and somatic tissue monitoring.

With these technologies, a clinician can noninvasively monitor a patient's perfusion (including pressure, flow, and tissue oximetry). This chapter will provide details regarding function and application for each of these sensor technologies.

ClearSight™ Finger Cuff

The combination of providing continuous blood pressure and arterial-based cardiac output (APCO) monitoring has historically depended upon utilization of an indwelling arterial catheter connected to a disposable pressure transducer or cardiac output sensor. The noninvasive ClearSight™ Finger Cuff provides clinicians with continuous blood pressure, cardiac output, and other advanced hemodynamic parameters without the need for an arterial catheter.

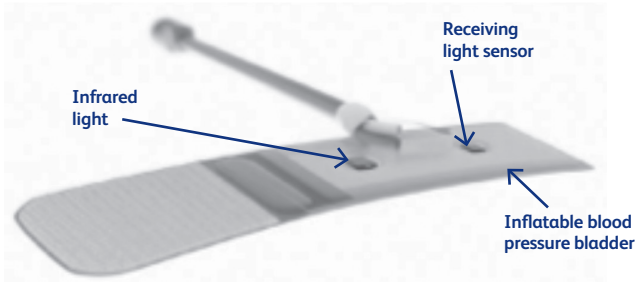
System components



Using information gathered from ClearSight™ Finger Cuff's components (inflatable bladder, infrared light, and receiving light sensor) the volume clamp and PhysioCal methods translate this information into an arterial pressure waveform from which beat-to-beat measurements can be continuously and accurately obtained.

ClearSight™ Finger Cuff leverages proven Nexfin technology which has historically been used in clinical settings and the aerospace industry for hemodynamic monitoring.

ClearSight™ Finger Cuff components

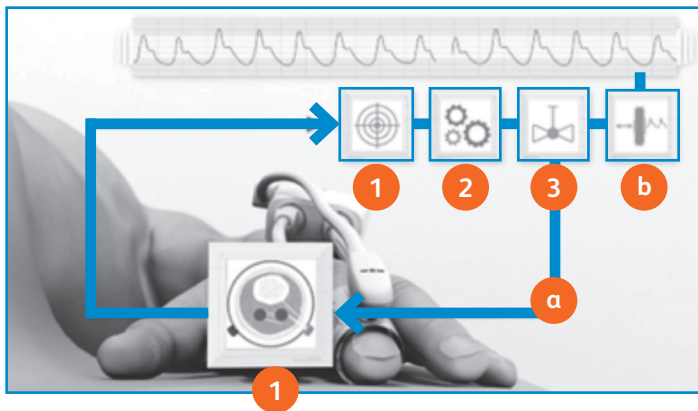


The emitting infrared light and receiving light sensor continuously measure the changing arterial volume that results from the pulsation of the heart. The pressure controller continuously adjusts the pressure in the inflatable bladder to match the pressure in the arteries of the digit(s) being measured, causing the arteries to temporarily stop pulsating. The volume of the arteries at this point is referred to as the **unloaded volume**.

Volume clamp method

The volume clamp method is the process employed to control the pressure in ClearSight™ Finger Cuff to maintain the unloaded volume pressure, which is equal to the blood pressure in the monitored digit(s). This measurement is then translated as a pressure waveform and is displayed on the advanced hemodynamic monitor.

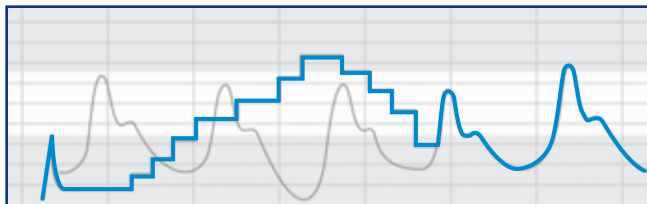
The volume clamp control loop, located within the pressure controller, consists of the following steps (see figure below):



- 1 The arterial volume, which is measured by the infrared light and receiving light sensor, is compared to the Physiocal method setpoint.
- 2 The pressure needed to counteract any arterial diameter change is determined by a controller.
- 3 a) The controller then sends a signal to the control valve which dynamically manages the amount of pressure applied to the finger cuff.
b) At the same time, the transducer directly senses the cuff pressure and translates it into a point on the blood pressure waveform. This continuous control loop, and adjustment of the cuff pressure, is performed 1000 times every second resulting in a real-time finger pressure waveform.

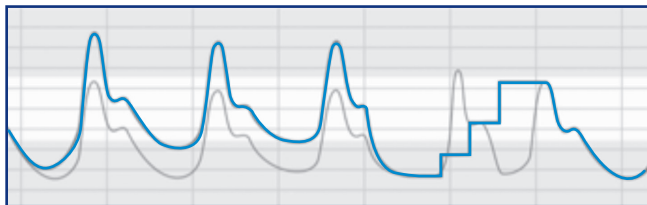
Physiocal method

To continuously calibrate blood pressure measurements to match changes in digital arterial volume, ClearSight™ Finger Cuff uses the Physiocal method to step up and down the pressure applied to the digit(s). This process is identified by the characteristic staircase-shaped waveform (see below). It allows ClearSight™ Finger Cuff to determine and periodically update the target unloaded volume, known as the **setpoint**.



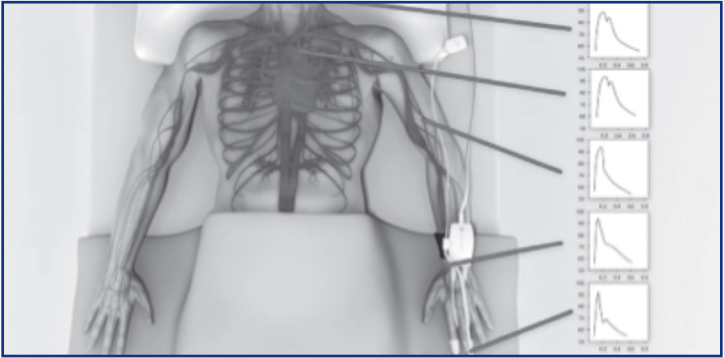
Typically, the first blood pressure waveform and its associated data will be displayed on the monitor in approximately 20 seconds.

The Physiocal method periodically recalibrates the system, which is essential for tracking the unloaded volume. Changes in the setpoint may result from variation in smooth muscle tone during events such as vasoconstriction, vasodilation, and temperature change. Calibration assessments begin after the 10th heartbeat and assessment intervals increase in length up to every 70th heartbeat, depending on the stability of the monitored digit(s) physiology.

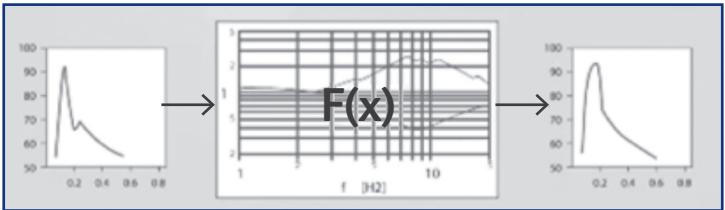


Arterial waveform reconstruction

Since the arteries narrow as the distance from the heart increases, increased resistance and backwards reflection of the pressure waves occur. This results in varying pressure readings and waveform morphologies when measurements are taken at different distances from the heart.



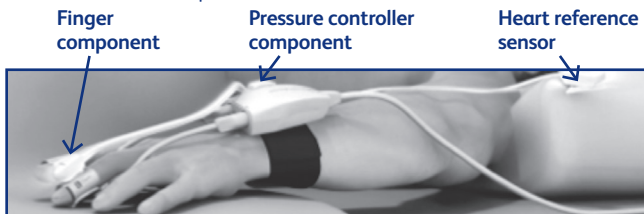
The radial artery is a common site for intravascular blood pressure monitoring. ClearSight™ Finger Cuff, when paired with a HemoSphere™ Monitor, has a waveform reconstruction algorithm that is able to mimic the pressures and waveform morphology of the radial artery to provide blood pressure-based parameter values (SBP, DBP, and MAP).



Additionally, the APCO algorithm that the ClearSight™ Finger Cuff uses to derive flow-based parameter values (SV, CO, etc.) relies on reconstruction of a waveform that mimics the brachial artery. The two algorithms (radial waveform reconstruction and APCO) work in parallel to noninvasively provide a complete set of advanced hemodynamic parameters.

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. The HRS functions like the transducer of an invasive arterial line. If it is placed below the patient's right atrium the measurements will be spuriously high; if placed above the right atrium, the measurements will be spuriously low. 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.

Acumen IQ™ Finger Cuff

The next generation noninvasive hemodynamic finger cuff technology offered by BD is the Acumen IQ™ Finger Cuff (pictured below). This technology leverages the fundamental principles of the volume clamp and Physiological methods for continuously monitoring blood pressure and flow parameters such as CO and SV. Additionally, Acumen IQ™ Finger Cuff unlocks Acumen Hypotension Prediction Index™ (HPI™) Software features and parameters (HPI™, Ea_{dyn} , and dP/dt). See page 7.1 for additional information on Acumen Hypotension Prediction Index™ (HPI™) Software.



Listed below are the advanced hemodynamic parameters available with each finger cuff sensor technology:

	CO/CI	SV/SVI	SVR/SVRI	SVV	PPV	MAP	HPI	dP/dt	Ea _{dyn}
Acumen IQ™ Finger Cuff	●	●	●	●	●	●	●	●	●
ClearSight™ Finger Cuff	●	●	●	●	●	●			



Scan the QR code to view the Acumen IQ™ Finger Cuff Application video.

Clinical Applications and Patients

Acumen IQ™ and ClearSight™ Finger Cuffs provide hemodynamic monitoring options for patients who could benefit from continuous monitoring and/or goal-directed therapy but may not receive an arterial line. These are usually patients undergoing moderate-to-high risk surgery who are at risk of developing complications, and critically ill patients in any hospital setting.

Contraindications

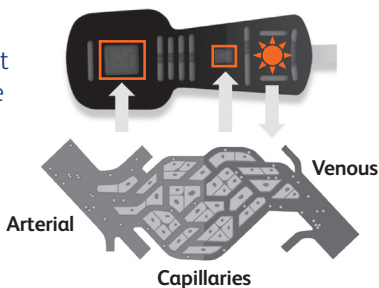
In some patients with extreme vasoconstriction of the arteries and arterioles in the lower arm and hand, as may occur in patients with Raynaud’s disease, digital blood pressure measurement may not be possible.

ForeSight™ Tissue Oximetry Sensor

ForeSight™ Tissue Oximetry Sensors utilize near infrared light spectroscopy (NIRS) to measure the amount of oxyhemoglobin and de-oxyhemoglobin concentrations at the level of the tissues (StO_2).

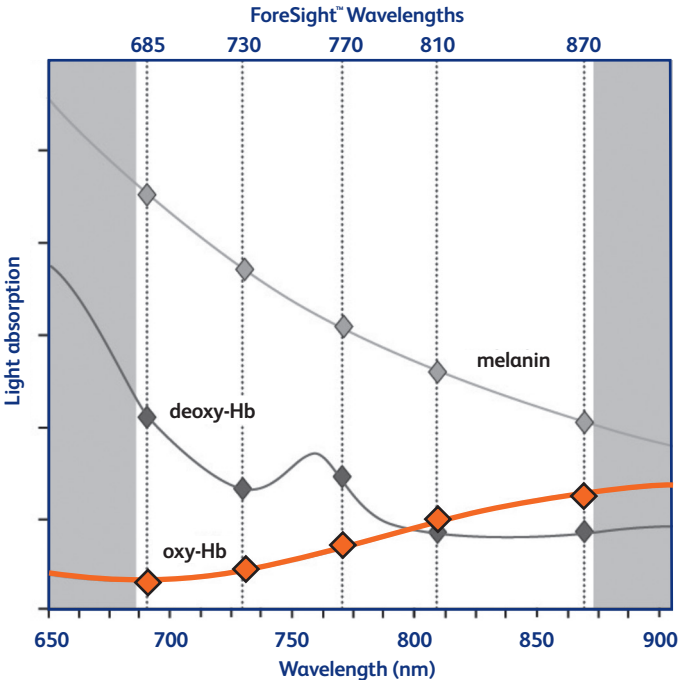
StO_2 is a representation of the regional (area under the sensor) microcirculation, with a mix of arterial, venous and capillary blood, and is displayed as a percent value on a BD monitor, which can be trended over time.

StO_2 can be considered a noninvasive surrogate for venous oxygen saturation and therefore represents the balance between oxygen delivery and consumption for the region of tissue over which a sensor is placed. Common sensor placements are on the forehead for cerebral tissue oximetry measurements before, during, and after surgery, and on the extremities during and after surgery. Additionally, tissue oximetry can be beneficial in monitoring critically ill patients in the ICU setting.



Five specific wavelengths of NIRS

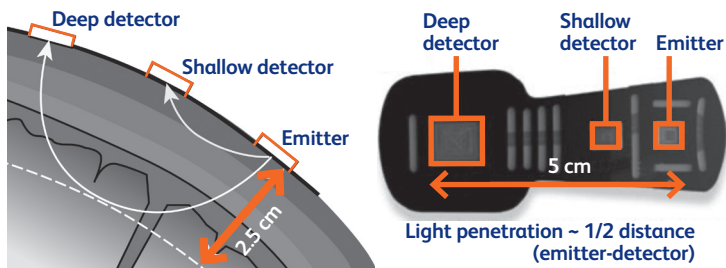
The five specific wavelengths of near infrared light analyze the tissue at the point where oxygenated and deoxygenated hemoglobin are more greatly distinguished. Utilizing the absorption curves of oxygenated and deoxygenated-hemoglobin and other chromophores (melanin and bilirubin) enables the ForeSight™ system to provide reliable and consistent readings regardless of the skin pigmentation.



Relationship of light absorption by three chromophores (melanin, deoxy- and oxy-hemoglobin) to light wavelengths between 650 and 900 nm. Note: the selection of five specific wavelengths and spacing between them follows the curvilinear characteristics of deoxygenated hemoglobin.

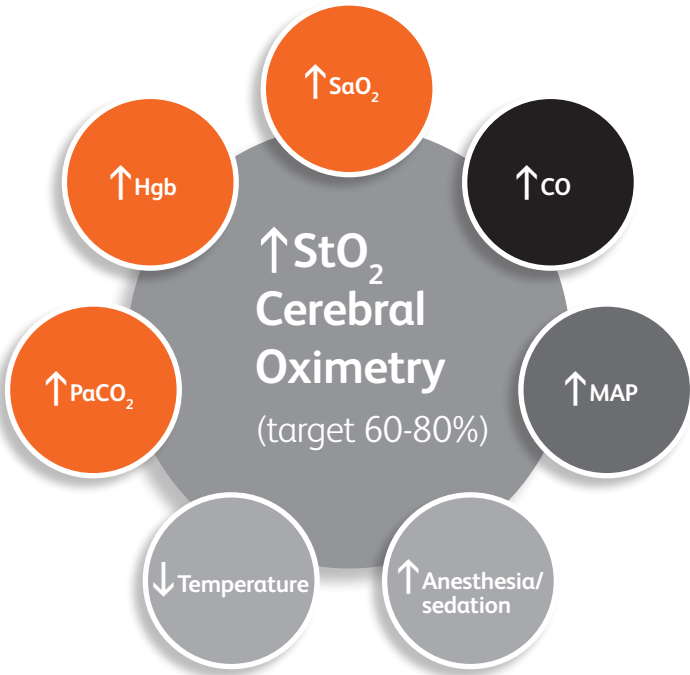
Sensor design

Near infrared light readily penetrates scalp and skull and thereby can assess cerebral oxygenation. The depth of light penetration is ~ 2.5 cm, which is half the distance between the emitter and deep detector (5 cm). This level of penetration mitigates contamination in patients with deeper scalp-to-cortex levels (>1.76 cm), which may be problematic in 2 cm systems. Additionally, ForeSight™ Sensors are intended to be gentle on the patient's skin by emitting a lower LED heat signature with CoolLight™ LED technology.



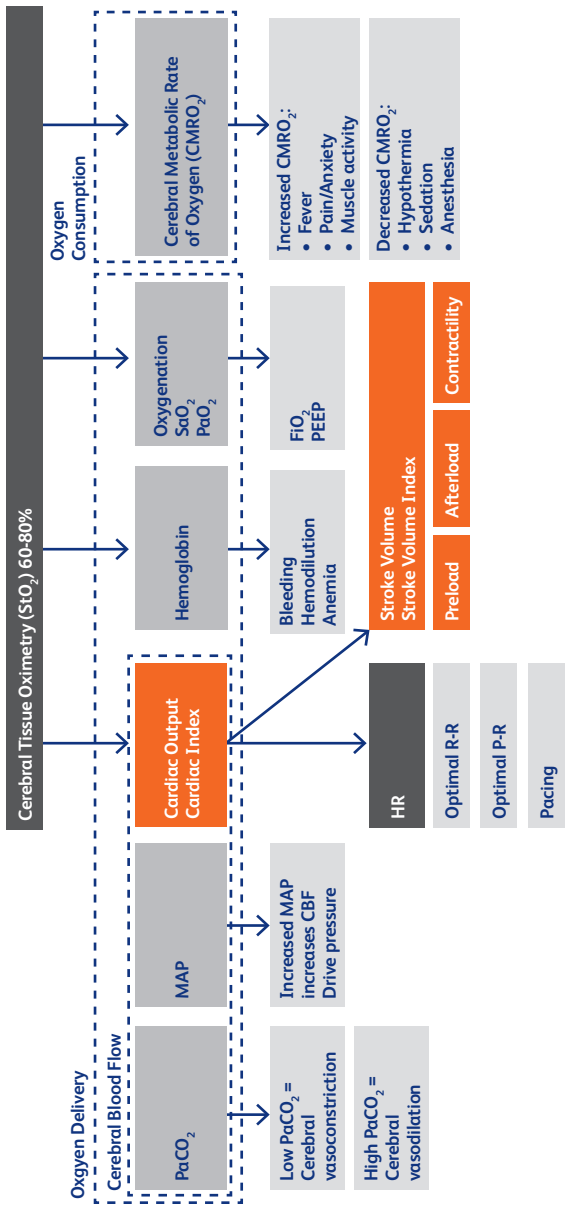
Above images illustrate the spacing between the light emitting source and the deep detector on the large ForeSight™ Sensor. The scalp-to-cortex distance is graphically illustrated in the flow of light from the emitter to the two detectors (i.e., shallow for detection of superficial tissues and deep for measuring brain oxygenation).

Considerations to increase
cerebral StO_2

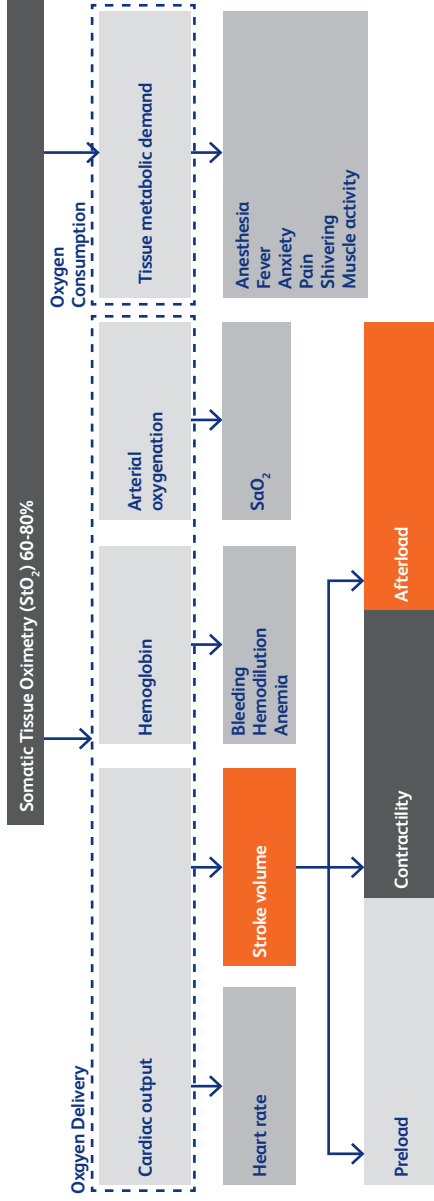


Scan the QR code to view the
Physiologic Factors Affecting Cerebral
Tissue Oximetry video.

Cerebral Tissue Oximetry (StO₂) Physio-relationship Graphic



Somatic Tissue Oximetry (StO_2) Physio-relationship Graphic



Potential Clinical Applications*

	Cerebral StO ₂	Somatic StO ₂
Surgical / procedural	• All cardiac surgeries • Thoracotomy / single lung ventilation • Carotid endarterectomy • Shoulder surgery – beach chair position • Complex surgical cases • V-tach ablations • Transcatheter cardiac procedures	• Detect distal limb ischemia – VA ECMO, Impella, IABP – Transcatheter cardiac procedures – Vascular procedures with loss of perfusion
Post-surgical / ICU	• VA and VV ECMO • Neuro SAH – vasospasm • Neuro TBI • Resuscitation quality • Heart failure • Bleeding • ROSC	• VA ECMO, Impella, IABP (<i>detect distal limb ischemia</i>) • Compartment syndrome • Post thoracoabdominal repair (<i>detect spinal ischemia</i>) • Resuscitation quality • Heart failure • Bleeding • Peripheral vascular (<i>post procedure loss of perfusion</i>)

* not an exhaustive list



Minimally-Invasive Monitoring



Minimally-Invasive Monitoring

BD minimally-invasive hemodynamic monitoring technologies utilize the arterial waveform, a robust source of physiologic data. When paired with a monitoring system, these high-fidelity arterial transducers can calculate stroke volume and cardiac output by using an arterial pressure-based cardiac output (APCO) algorithm.

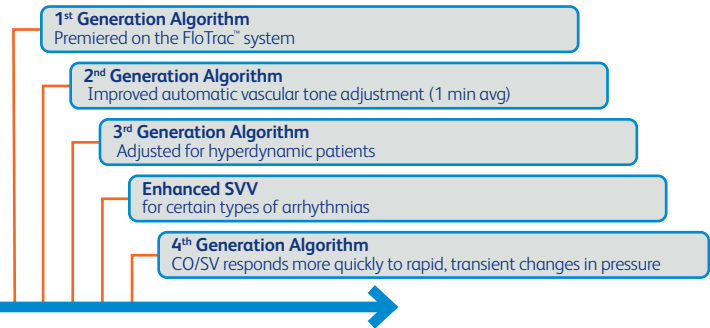
BD makes two minimally-invasive arterial sensors, Acumen IQ™ and FloTrac™ Sensors, both of which utilize the FloTrac™ (APCO-based) algorithm to derive stroke volume from the arterial waveform. In addition to all parameters available with the FloTrac™ Sensor, Acumen IQ™ Sensor unlocks additional parameters (see table below), these are described in greater detail in chapter 7.

	CO/CI	SV/SVI	SVR/SVRI	SVV	PPV	MAP	HPI	dP/dt	Ea _{dyn}
Acumen IQ™ sensor	●	●	●	●	●	●	●	●	●
FloTrac™ sensor	●	●	●	●	●	●			

FloTrac™ algorithm

The FloTrac™ algorithm has evolved since its introduction in 2005 with broader application across more clinical conditions, including the ability to adjust for rapid changes in pressure that occur with vasopressor administration.

FloTrac™ Algorithm Evolution



When adding a FloTrac™ or Acumen IQ™ Sensor to an existing arterial line and measuring the arterial pulse pressure, the FloTrac™ algorithm calculates stroke volume (SV) and multiplies it by the pulse rate (PR) to obtain cardiac output (CO).

$$\mathbf{CO} = \mathbf{PR} \times \mathbf{SV}$$

$$\mathbf{SV} = \sigma_{\text{AP}} * \mathbf{X}$$

$$\mathbf{X} = \mathbf{M} (\mathbf{HR}, \sigma_{\text{AP}}, \mathbf{C} (\mathbf{P}), \mathbf{BSA}, \mathbf{MAP}, \mu_{1\text{ap}}, \mu_{2\text{ap}}, \mu_{3\text{ap}}, \mu_{4\text{ap}} \dots)$$

PR = Pulse rate, calculated by counting the number of arterial upstroke pulsations in a 20 second period and extrapolating to a per minute value

σ_{AP} = Standard deviation of arterial pulse pressure around the mean arterial pressure (MAP) value and is proportional to the stroke volume. This is calculated by analyzing the arterial pressure waveform over 20 second periods at 100 times per second, creating 2,000 data points from which σ_{AP} is calculated and measured in millimeters of mercury

Khi (X) = Scaling multivariate parameter proportional to the effects of vascular tone on pulse pressure, see additional details on next page

M = Multivariate polynomial equation

C = Large vessel compliance, see additional details on page 4.4

BSA = Body surface area calculated by Dubois' equation

MAP = Mean arterial pressure calculated by taking the 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

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

- Measured beats per minute
- Averaged every 20 seconds

- Based upon the basic physiological principle of pulse pressure's (PP) proportionality to SV
- σ_{AP} utilized to compute a robust assessment of key PP characteristics

- Compensates for differences in vascular tone (compliance and resistance)
- Patient-to-patient differences estimated from biometric data
- Dynamic changes estimated by waveform analysis (skewness, kurtosis of the waveform)

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

The standard deviation of arterial pressures (mmHg) is converted into SV (mL/beat) by multiplying it by a conversion factor known as *Khi* (X). *Khi* is a multivariate polynomial equation that 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 the patient's demographics, and skewness and kurtosis of the arterial waveform. *Khi* is calculated on a rolling 60-second average and is continuously applied to the FloTrac™ algorithm.

Large vessel compliance (C)

Langewouters found a direct correlation between age, gender, and MAP with 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:

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

L = estimated aortic length

$A_{\max}$ = maximal aortic cross-sectional area

P = arterial pressure

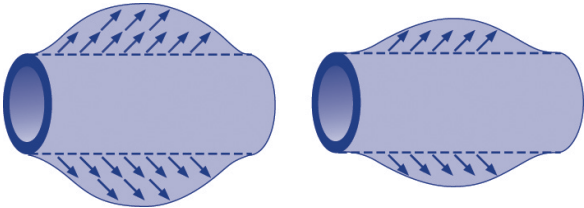
P_0 = pressure at which compliance reaches its maximum

P_1 = pressure where arterial compliance is half of maximum value

Vessel compliance is assessed in part by the Langewouters equation. 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.

- Younger
 - Male
 - Higher BSA
- vs.
- Older
 - Female
 - Lower BSA

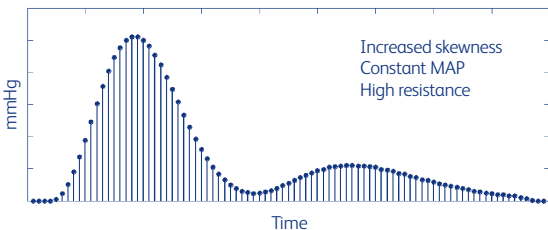
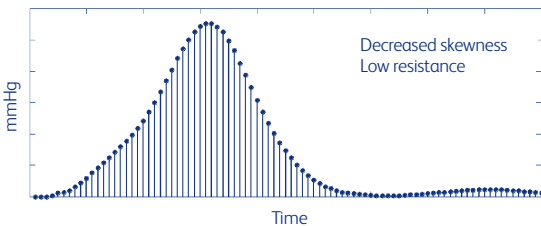
For the same volume →



- Compliance inversely affects pulse pressure (PP)
- The algorithm compensates for the effects of compliance on PP, based on age, gender, and BSA

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

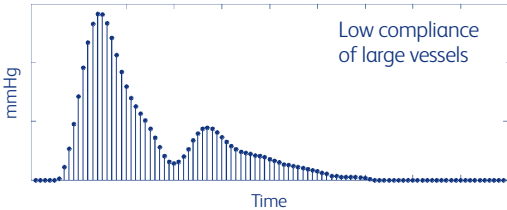
Symmetry characteristics on arterial pressure can indicate a change in vascular tone and/or resistance. Two different functions may have the same mean and standard deviation but will rarely have the same skewness. For example, an arterial pressure waveform in which the data points increase quickly in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



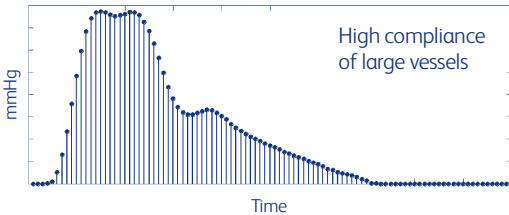
Kurtosis (a measure of how peaked or flat the pressure data points are distributed from normal distribution, $\mu_{4a}p$)

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



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* autocorrects for changes in vascular tone through a detailed waveform analysis. This feature also eliminates the need for indicator dilution methods used in technologies that require manual calibration.

Technical considerations

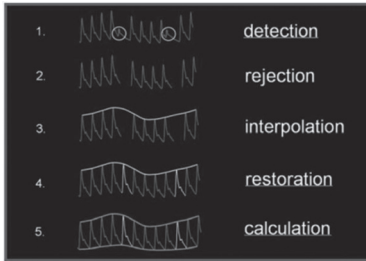
The FloTrac™ system algorithm is dependent upon a high-fidelity pressure tracing; therefore, attention to technique in pressure monitoring setup and maintenance is important. Critical components of good technique include priming with gravity, keeping the pressure bag at 300 mmHg, ensuring adequate I.V. bag flush volume, keeping sensor stopcock level with the phlebostatic axis, and periodic testing of optimal damping with a square wave test.

Modifying the FloTrac™ system (the sensor and tubing) may reduce the dynamic response, resulting in compromised hemodynamic monitoring. If the waveform is not optimal, SV and CO are not reliably measured.

The SVVxtra Algorithm

In previous versions of the algorithm, occasional dysrhythmias have been considered a contraindication for using SVV as a guide for fluid resuscitation. The SVVxtra algorithm allows for continued use of SVV despite the presence of premature atrial or ventricular contractions. The SVVxtra algorithm allows analysis of the respiratory component of the arterial pressure curve so that the physiological effects of mechanical ventilation on the heart and stroke volume can be interpreted and applied clinically.

The SVVxtra algorithm is based on five consecutive steps:



If the frequency of dysrhythmias has exceeded the algorithm's ability to filter these dysrhythmias, a broken **yellow heart** icon will appear in the SVV parameter tile as shown below.



Although interpretation of SVV to predict volume responsiveness has limitations (see 'LIMITS' discussed on page 5.6) the SVVxtra algorithm mitigates one of the most common limitations related to irregular heartbeats.

It is important to emphasize that none of the limitations of SVV interpretation (see 'LIMITS' on page 5.6) affect the accuracy of the SV and CO measurements calculated from the FloTrac™ Algorithm.

In those conditions where concerns about the limitations of SVV to predict fluid responsiveness exist, clinicians can use one of the other methods detailed in chapter 5 to assess fluid responsiveness.

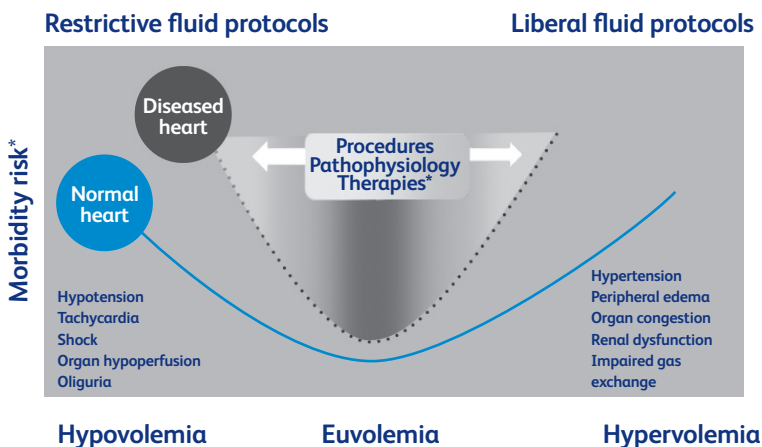


Methods for Assessing Fluid Responsiveness

Methods for Assessing Fluid Responsiveness

Correcting a volume deficit is often the first step when seeking to improve perfusion and oxygen delivery. However, only about half of hemodynamically unstable patients are fluid responsive. Using hemodynamic parameters such as CO, SV, and SVV to assess preload or preload responsiveness has demonstrated higher sensitivity and specificity when compared to traditionally monitored parameters such as heart rate or blood pressure.

As shown in the diagram below, insufficient or excessive fluid administration may increase risk of complications.



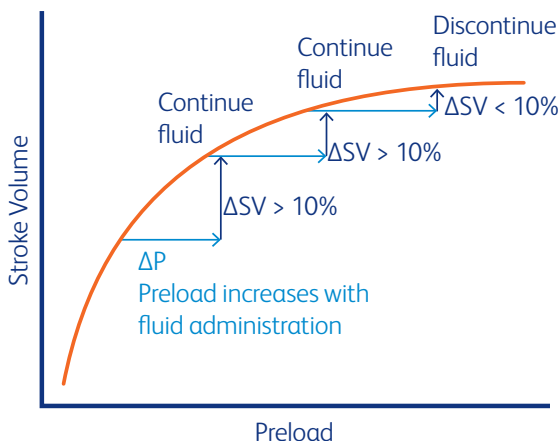
Appropriate and safe fluid administration can be achieved by utilizing one of these three common assessment techniques:

- Assessing stroke volume (SV) or cardiac output (CO) in response to a fluid challenge
- Assessing stroke volume (SV) or cardiac output (CO) with a passive leg raise (PLR)
- Monitoring a dynamic index such as stroke volume variation (SVV) or pulse pressure variation (PPV)

Fluid challenge

Fluid resuscitation only benefits patients who are preload responsive. Following the principles of the Frank-Starling curve, fluid responsiveness can be determined by monitoring % change in stroke volume (ΔSV) when a small amount of fluid (200-250 mL) is administered over a short amount of time (5-10 minutes). The same principles can be applied when monitoring CO instead of SV, as long as the patient's heart rate is stable.

Frank-Starling Curve



Passive leg raise (PLR) maneuver

A PLR maneuver can be performed as a reversible fluid challenge. Approximately 150-300 mL of blood moves from the lower extremities to the central circulation, and thus the effect of the increased preload can be evaluated.

The impact of this added blood volume in the central circulation can indicate if a patient will increase their blood flow (SV or CO) without having to administer fluid.

Patients who are preload responsive will show maximal effect in ~120 seconds, defined as a change in SV or CO > 10%.

PLR steps



- 1 Start from semi-recumbent and not the supine position. Check that the trunk is at 45°.
- 2 Patient positioned supine with legs straight and elevated to 45° for two minutes.
- 3 Return patient to starting position.

PLR considerations

- PLR effects must be assessed by a direct measurement of SV or CO and not by measurement of BP
- The assessment technique must be able to detect short-term and transient changes since the PLR effects may vanish after one minute
- SV or CO must be measured before, during, and after PLR in order to check that the measured parameter (SV or CO) returns to its baseline
- **A PLR may be inappropriate depending upon a patient's location, procedure, or injuries**
- **Pain, cough, discomfort, and awakening could provoke adrenergic stimulation, resulting in mistaken interpretation of SV or CO changes**

Stroke volume variation

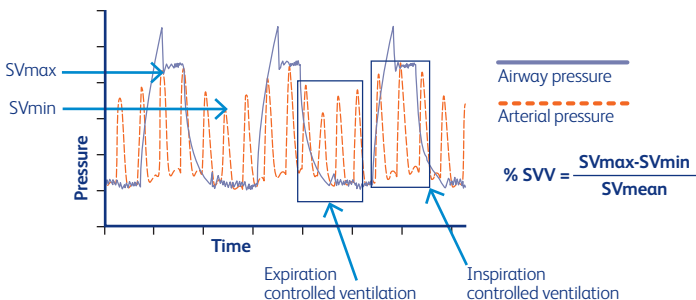
Hemodynamic monitoring can be obtained continuously or intermittently by using either static (HR, MAP, CVP) or dynamic (SVV, PPV) parameters. Static parameters are single snapshots taken at specific points in the cardiac or respiratory cycle. Dynamic parameters should be trended to assess changes in the cardiovascular status over time. Although stroke volume variation (SVV) and pulse pressure variation (PPV) are reliable indicators of preload responsiveness for patients on controlled mechanical ventilation, here we will focus on the utility of SVV.

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

Calculating stroke volume variation

With positive pressure ventilation, stroke volume increases during inspiration and decreases during expiration. This is due to changes in intrathoracic pressure. SVV is calculated by evaluating a full respiratory cycle and identifying $SV_{\min}$, $SV_{\max}$, and SV_{mean} .

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



- **SVV < 9%** Patient unlikely to respond to fluid administration with an increase in SV/CO
- **SVV 9 -13%** Patient may or may not respond to fluid administration with an increase in SV/CO. This is often referred to as the 'grey zone'. Proceed with fluid administration cautiously
- **SVV > 13%** Patient likely to respond to fluid administration with an increase in SV/CO

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 and PPV have been shown to have high sensitivity and specificity in their ability to determine fluid responsiveness under specific conditions (see next page for LIMITS). Additionally, trending CO and SVV together can provide an indication of fluid responsiveness and is a means of verifying that the fluid administration was beneficial.

Limitations to the use of SVV (LIMITS)

- **L**ow HR/RR ratio
- **I**rrregular 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), tidal volume, and, therefore, mean airway pressure may decrease preload and 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 will always decrease preload and increase SVV. This 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 provides information about cardiopulmonary interactions.



Swan-Ganz™ Pulmonary Artery Catheters



Swan-Ganz™ Pulmonary Artery Catheter

The first thermodilution Swan-Ganz™ Pulmonary Artery Catheter was introduced in 1970 by Dr. Jeremy Swan and Dr. William Ganz. Today, when connected to a bedside physiologic monitor and pressure transducer(s), the Swan-Ganz™ Catheter enables clinicians to measure right heart pressures, pulmonary artery occlusion pressure, sample mixed venous blood from the pulmonary artery, and measure cardiac output using thermodilution.

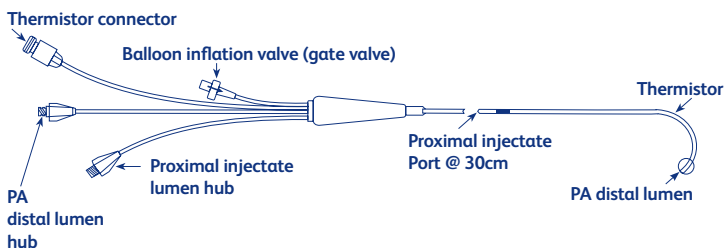
Swan-Ganz™ Catheter measures:

- Right heart pressures:
 - Right atrial pressure (RAP)
 - Pulmonary artery pressure (PAP)
 - Pulmonary artery systolic (PAS)
 - Pulmonary artery diastolic (PAD)
 - Mean pulmonary arterial pressure (MPAP)
 - Pulmonary artery occlusion pressure (PAOP)
- Thermodilution cardiac output:
 - BD CO-Set iced, closed bolus injectate system
 - BD CO-Set room temperature, closed bolus injectate system
- Pulmonary artery blood sampling for laboratory analysis:
 - Mixed venous blood oxygen saturation (SvO₂)
 - Serial measurements of right heart chamber oxygen saturations

Applications of Swan-Ganz™ Catheters

- Diagnostic right heart catheterization to obtain right heart pressures
- Point-in-time calculations of cardiac output using bolus thermodilution for evaluating cardiac function
- Mixed venous blood draws to obtain SvO₂ values that can aide in evaluation of 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

Swan-Ganz™ Pulmonary Artery Catheter



Advanced Technology Swan-Ganz™ Pulmonary Artery Catheter

In addition to providing the same functions as the Swan-Ganz™ Catheter, the advanced technology Swan-Ganz™ Catheter provides the ability to continuously monitor the balance between oxygen delivery and consumption through continuous monitoring of SvO₂ and CO. Additionally, stroke volume (SV) and measures of its contributing components (preload, contractility, and afterload) can be continuously calculated as described below.

The advanced technology Swan-Ganz™ Catheter additionally measures:

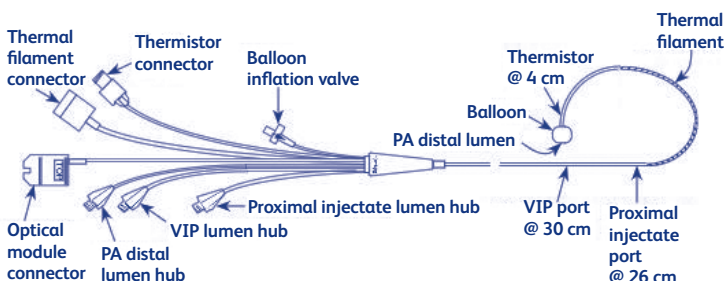
- SvO₂ – mixed venous oxygen saturation can be continuously measured through fiberoptic reflectance technology and is a global indicator of the balance between oxygen delivery and consumption
- CCO – continuous cardiac output can be measured using advanced thermodilution technology; CO is a key component of oxygen delivery
- SV* - stroke volume can be continuously calculated by dividing the measured CCO by heart rate (HR). SV is frequently used in determining preload responsiveness and is also a key component of oxygen delivery
- SVR – systemic vascular resistance can be continuously calculated when connected to a HemoSphere™ Monitor, if MAP and CVP are also input from the bedside physiologic monitor
- RVEF* – right ventricular ejection fraction can be continuously calculated and is an indicator of right ventricular function
- RVEDV* – right ventricular end diastolic volume can be continuously calculated by dividing SV by RVEF and is a key indicator of preload

* These values can be obtained when an ECG cable is used per the IFU

Applications of advanced technology Swan-Ganz™ Catheter:

- Continuous assessment of right heart pressures (RAP, PAS, PAD, MPAP, PAOP)
- 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 SV^* and RVEDV^*
- Continuous assessment of afterload through SVR, SVRI
- Continuous assessment of contractility through RVEF^*
- Intermittent calculation of oxygen delivery (DO_2) and consumption (VO_2)

Advanced Technology Swan-Ganz™ Catheter Pulmonary Artery Catheter



* These values can be obtained when an ECG cable is used per the IFU

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 resection
- Liver transplantation
- Complex lung resection
- Pulmonary edema
- Pulmonary hypertension
- Shock of unknown etiology
- Shock that is unresponsive to resuscitation attempts
- Severe trauma
- Post-resuscitation in critically ill patients

Relative contraindications for Swan-Ganz™ Pulmonary Artery Catheterization*

- Left bundle branch block
- Patients with tricuspid or pulmonic heart valve replacements
- Presence of endocardial pacing leads

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

* Not an exhaustive list

Swan-Ganz™ Pulmonary Artery 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

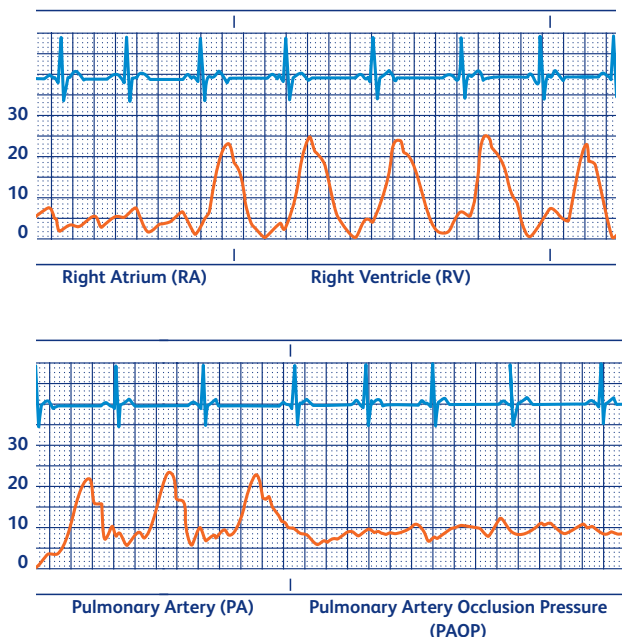
Note: Catheter markings occur every 10 cm and are denoted by a thin black ring. 50 cm markings are denoted by a thick black ring. Catheter must exit introducer sheath before inflating balloon, approximately 15 cm of catheter length.

Cardiopulmonary Physiology as it Relates to Pulmonary Artery Catheter Pressure Monitoring

Understanding the physiologic implications of catheter tip positioning and the associated signs of appropriate positioning vs. malpositioning are of paramount importance during insertion and ongoing monitoring of patients with a pulmonary artery catheter in place.

As the catheter tip tracks through the right side of the heart and into the pulmonary artery, there are distinctive waveform morphology changes that provide clear evidence of where along this path the catheter tip is located.

Continuous waveform tracing from tip of a pulmonary artery catheter (PAC) as it tracks through the right heart



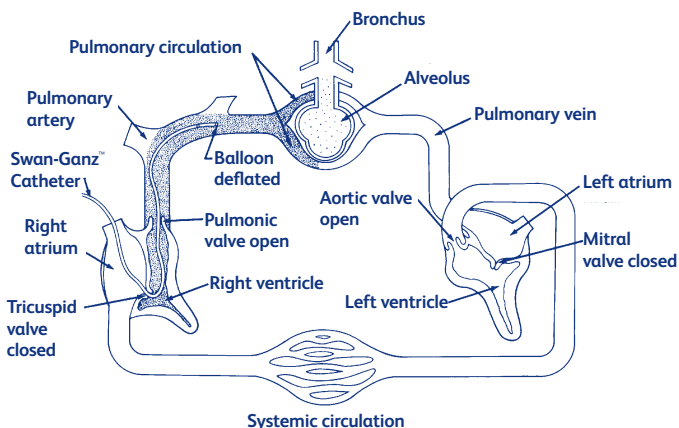
Based on which heart valves are opened or closed at a given time in the cardiac cycle, pressure readings from the tip of the PAC that is positioned in the pulmonary artery can be nearly identical to the pressures in the adjacent heart chambers.

Ventricles in systole

In the figure below, the balloon tip of the catheter 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 (PAS) pressure, which reflects right ventricular systolic pressure (RVSP); essentially there is now a common chamber with 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 (PAD) pressure 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 PAD.

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.

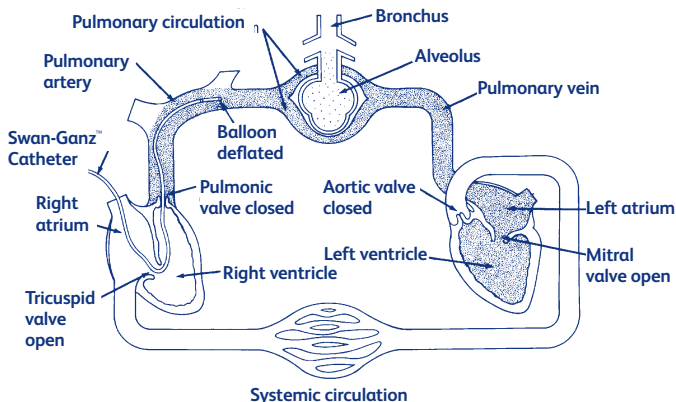
When transducing the proximal port, the right atrial pressure (RAP) reflects right ventricular end-diastolic pressure (RVEDP) when the tricuspid valve is open.

Ventricular diastole

RAP = RVEDP

RVEDP < PAD

PADP ≈ LAP ≈ LVEDP



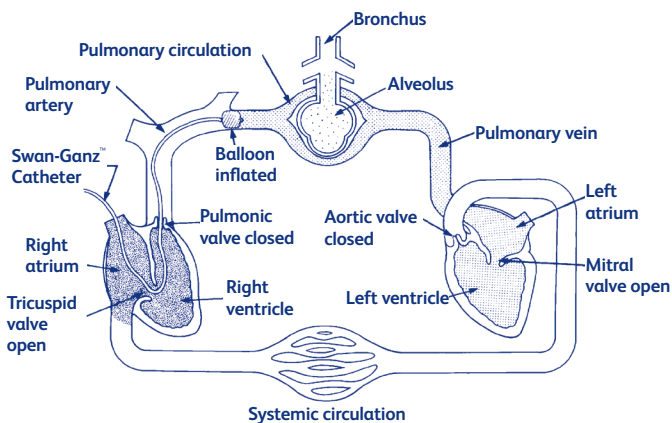
Ventricles in diastole: catheter wedged

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 $\approx$ LAP $\approx$ LVEDP



Normal Insertion Pressures and Waveform During Pulmonary Artery Catheter Insertion

Catheter tip in right atria representing right atrial pressure (RAP) or central venous pressure (CVP)

2 to 6 mmHg

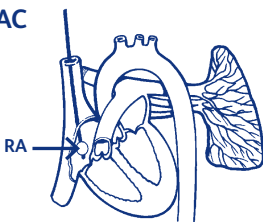
Mean 4 mmHg

a = atrial systole

c = backward bulging from tricuspid valve closure

v = atrial filling, ventricular systole

PAC



Catheter tip in right ventricle representing right ventricular pressure

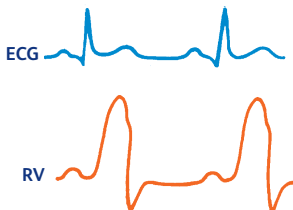
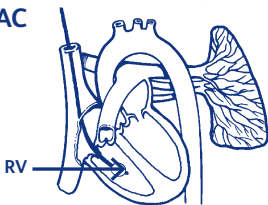
Systolic Pressure (RVSP)

15–25 mmHg

Diastolic Pressure (RVDP)

0–8 mmHg

PAC



Normal Insertion Pressures and Waveform During Pulmonary Artery Catheter Insertion

Catheter tip in pulmonary artery representing pulmonary artery pressure (PAP)

Systolic Pressure (PAS)

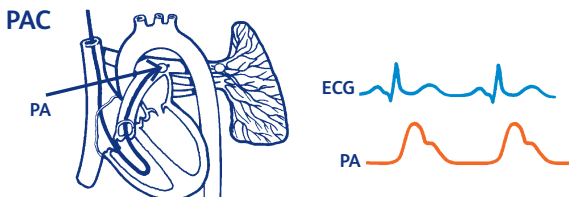
15–25 mmHg

Diastolic Pressure (PAD)

8–15 mmHg

Mean Pressure (MPAP)

10–20 mmHg

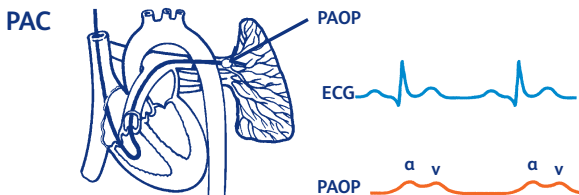


Catheter tip in pulmonary artery representing pulmonary artery occlusion pressure (PAOP) or wedge pressure

Mean 6–12 mmHg

a = atrial systole

v = atrial filling, ventricle systole



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: Not an exhaustive list of 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

Swan-Ganz™ Pulmonary Artery Catheter

Insertion Techniques

- 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. Of note, an introducer sheath that is 1F size larger than the catheter should be used to limit friction and/or damage to the catheters balloon tip.
- 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.
- 4 Catheter advancement to the PA should be rapid, since prolonged manipulation can result in loss of catheter stiffness. Some Swan-Ganz™ Catheters are 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 occlusion 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.

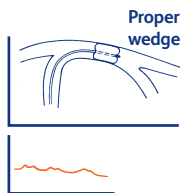
- 6 To reduce or remove any redundant length or loop in the right atrium or ventricle, slowly pull the catheter back 2 to 3 cm. Then reinflate the balloon to determine the minimum inflation volume necessary to obtain an occlusion pressure tracing. The catheter tip should be in a position where full inflation volume produces an occlusion pressure tracing. With the balloon deflated, reattach the syringe in an empty position to mitigate inadvertent compression of plunger and injection of air into the balloon.

Continuous Pulmonary Artery Pressure Monitoring

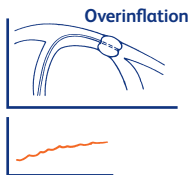
- 1 Optimize pressure monitoring systems according to manufacturers' recommendations.
- 2 Maintain patency of inner lumens with continuous flush solution (heparinization, if any, per institutional protocol).
- 3 Observe waveforms for proper placement.
- 4 Catheter migration may occur. Note any damping 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 the volume is less than 1.5 cc*, or more than the volume that is marked on the catheter shaft (this volume is also found in the catheter's IFU), the catheter position may have changed.
- 7 Never use more than the recommended balloon inflation volume marked on the catheter shaft.

* Most common Swan-Ganz™ Catheter balloon inflation volume. Refer to catheter IFU to confirm correct inflation volume

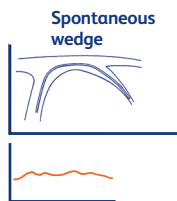
- 8 Never inflate the balloon more than the minimum required to obtain an occlusion (or wedge) tracing.



Full inflation with 1.5 cc* inflation volume. Appropriate a,c,v waves noted.



Overinflation of balloon. Note: waveform rise on screen and the loss of the a,c,v waveform.



Catheter spontaneous wedging. Wedge type tracing with balloon deflated.

* Most common Swan-Ganz™ Catheter balloon inflation volume; refer to catheter IFU to confirm correct inflation volume

Summary Guidelines for Safe Use of Balloon-tipped Swan-Ganz™ Pulmonary Artery Catheter

- 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 waveform 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 damped 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/occlusion pressure, inflate 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 occlusion pressure. Remove the syringe to allow rapid balloon deflation, and then reattach the syringe to the balloon lumen. Air should not be used in cases where air may enter the arterial circulation, consider using CO₂.
- 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 occlusion (PAOP) pressures are nearly identical, wedging the balloon may not be necessary; monitor 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 (e.g., sepsis, acute respiratory failure, and shock), the relationship between PAD and PAOP may change with the patient's clinical condition, and the occlusion/wedge position and measurement may need to be reassessed.
 - Keep occlusion time to a minimum (two respiratory cycles or 10 to 15 seconds), especially in patients with pulmonary hypertension.
 - Avoid prolonged maneuvers to obtain an occlusion pressure. If difficulties are encountered, abort attempting the "wedge."
 - Never flush the catheter when the balloon is wedged in the pulmonary artery.*
- 5 Bedside physiologic monitor settings initiated and maintained
 - Pulmonary artery pressure systolic/diastolic/mean alarm settings should 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.

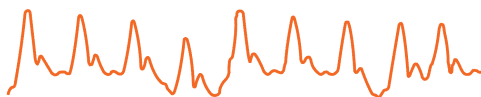
* See IFU for additional warning information

Ventilatory Effects on Pulmonary Artery Tracings

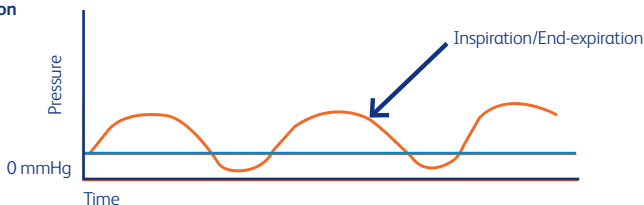
Spontaneous Breathing

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.

PAP waveform



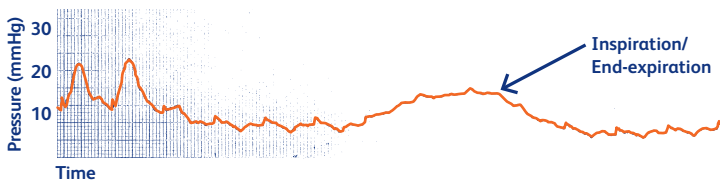
Respiration



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

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.

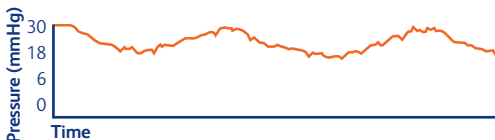
PAP to PAOP tracing during balloon inflation



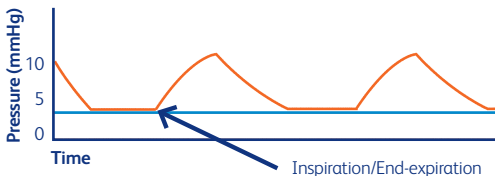
Controlled Mechanical Ventilation (CMV) and Pressure Supported Ventilation (PSV)

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. At the beginning of inspiration, pressure begins to rise and expiration ends. This is when the wedge pressure should be obtained.

PAOP waveform



Respiratory waveform



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

Cardiac Output Determinations

Fick Method

Adolph Fick established the principles of cardiac output determination in the 1870's. Fick's concept is based upon the premise that an organ's uptake or release of a substance, like oxygen, is the product of blood flow through the organ and the arterial and venous difference in values of the same substance.

The Fick method utilizes oxygen as the substance and the lungs as the organ. Arterial and venous oxygen content are measured to obtain the difference ($a - v O_2$). Oxygen consumption (VO_2) can be calculated from the inspired minus expired oxygen content and ventilation rate. The cardiac output can then be determined using this formula:

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

(volume % = 1 mL oxygen/100 cc)

- Normal (CaO_2) arterial oxygen content: 20 volume %
- Normal (CvO_2) mixed venous oxygen content: 15 volume %
- Normal (VO_2) oxygen consumption: 250 mL/min

Calculating cardiac output with the Fick equation requires accurate measurement of the oxygenation variables. Slight errors in the content values may produce large errors in the oxygen consumption result. Normal oxygen consumption ranges between 200–250 mL/min. Indexed normal VO_2 values are 120–160 mL/min/m². Critically ill patients may not have normal oxygen consumption values; therefore, insertion of normal values into the above Fick equation may produce erroneous cardiac output values.

Dye/Indicator Dilution Method

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

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

By taking continuous blood samples, a time-concentration plot, called an indicator-dilution curve can be obtained. Once this is plotted, the cardiac output can be calculated using the Stewart-Hamilton equation: $CO = \frac{I \times 60}{C_m \times t \times k}$

CO = cardiac output (l/min)

I = amount of dye injected (mg)

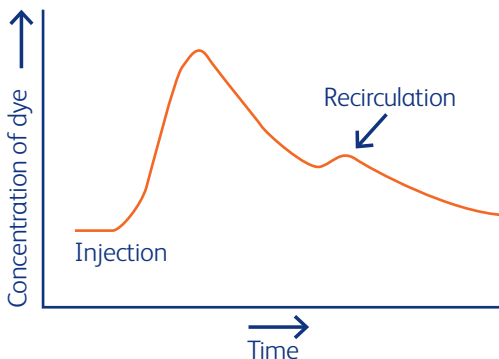
60 = 60 sec/min

C_m = mean indicator concentration (mg/l)

t = total curve duration (sec)

k = calibration factor (mg/mL/mm deflection)

Indicator dilution curve



Thermodilution Method

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

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

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

$$CO = \frac{V \times (TB-TI)}{A} \times \frac{(SI \times CI)}{(SB \times CB)} \times \frac{60 \times CT}{1} \times K$$

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

Thermodilution Curve

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.

Multiple factors can affect the accuracy of bolus CO measurements, refer to page 9.8 for troubleshooting chart.



Scan the QR code to view the
Swan-Ganz™ Continuous Cardiac
Output (CCO) Catheter video

Continuous Mixed Venous Oxygen Saturation Monitoring

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 (e.g., 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 (e.g., increased lactate and low SvO_2) even after they have been resuscitated to normalized vital signs.

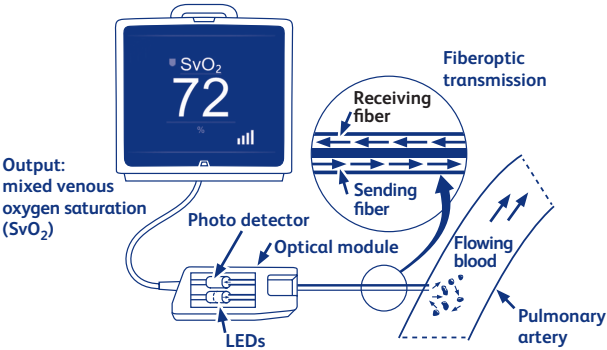
SvO_2 = early warning and prevention

Continuous venous oximetry is a sensitive real-time indicator of the balance between oxygen delivery and consumption. As such, SvO_2 can be considered a global indicator of this balance. 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. Additionally, blood draws from the pulmonary artery via the distal lumen can be analyzed to measure SvO_2 intermittently.

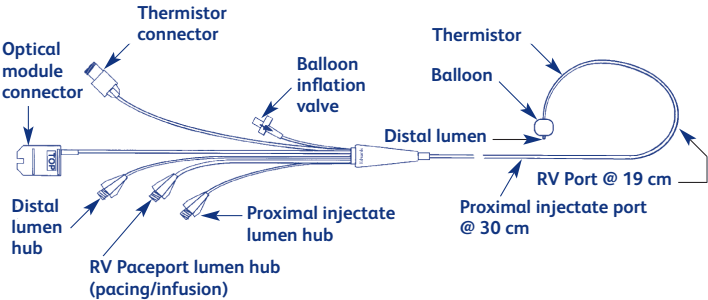
Continuous SvO_2 monitoring technology

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.

Reflection spectrophotometry



Swan-Ganz™ Oximetry Thermodilution Catheter oximetry thermodilution catheter



Interference with SvO₂ 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 (SvO₂) values

Normal range values for SvO₂ are 60-80%. Low oximetry readings usually indicate either low oxygen delivery (DO₂) or an increase in oxygen consumption (VO₂). High oximetry readings can indicate a low oxygen extraction state such as sepsis.

When change is significant

SvO₂ values are not static. These values may show significant changes with activities (e.g., fever, muscle activity, etc.) or interventions such as suctioning. When monitoring SvO₂, 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 SvO₂:

- Cardiac output (CO)
- Hemoglobin (Hgb)
- Arterial oxygen saturation (SaO₂)
- Oxygen consumption (VO₂)

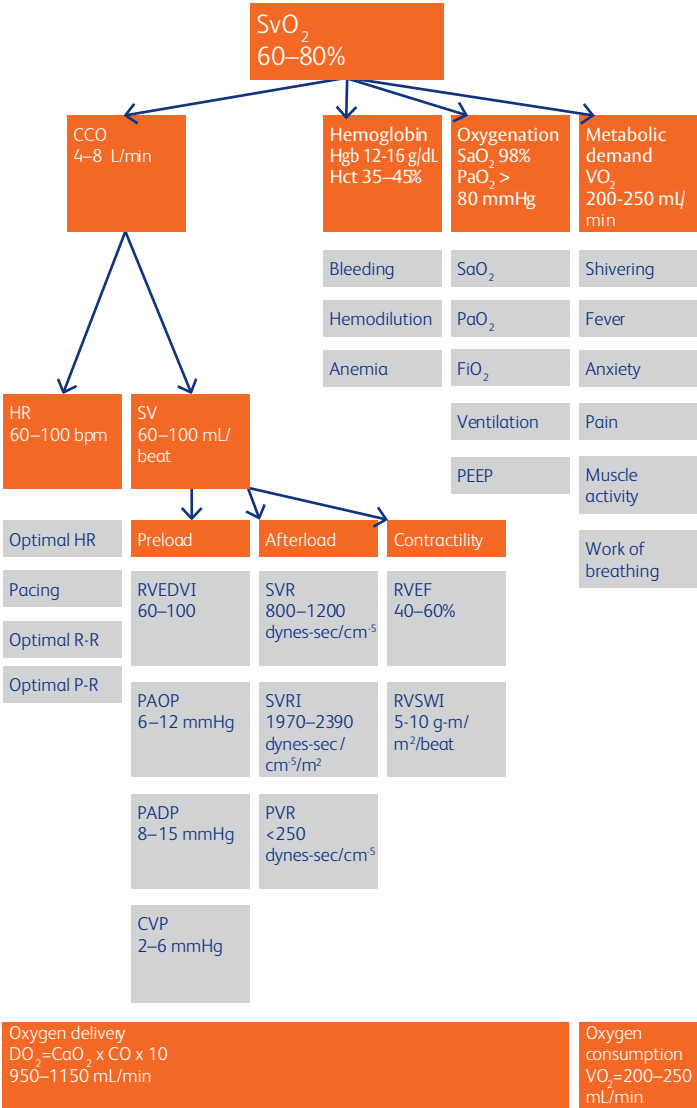
Note: CO, hemoglobin and SaO₂ are indicators of oxygen delivery (DO₂)

Clinical application of SvO₂

When used with cardiac output monitoring, either from an advanced technology Swan-Ganz™ Catheter or other minimally or noninvasive CO technologies, SvO₂ can assist clinicians in determining the adequacy of oxygen delivery as well as help to differentiate between issues of oxygen delivery vs. oxygen consumption.

Advanced Technology Swan-Ganz™ Catheter

Physio-relationship Graphic



Summary

As with all measurements in hemodynamic monitoring, single point in time SvO_2 values are not as informative as observed trends over time. Tracking multiple trended values over time can inform clinical decision making, individualize patient care and improve outcomes when used appropriately.



Clinical Decision Support Software and Sensors



Clinical Decision Support Software and Sensors

BD continues to make advances in clinical decision support software and sensors. These tools leverage machine learning to detect and predict occurrences of hemodynamic instability, as well as provide clinicians with additional information that can be used to assess and guide treatment of impending or ongoing hemodynamic instability.

Acumen Hypotension Prediction Index™ (HPI™) Software

Acumen HPI™ Software is intended for use in surgical and non-surgical* patients receiving advanced hemodynamic monitoring. It provides clinicians with information regarding the likelihood of a patient trending towards a hypotensive event. A hypotensive event is defined as mean arterial pressure (MAP) <65 mmHg for at least one minute.

Acumen HPI™ Software contains advanced parameters such as HPI™, Ea_{dyn} and dP/dt . The software's various visual display formats and screen layouts aid clinicians in identifying the root cause(s) of impending or ongoing hemodynamic instability.

* When monitoring with Acumen IQ™ Cuff, the Acumen HPI™ feature is cleared for use only in surgical patients

Acumen HPI™ Software and its associated parameters are unlocked by pairing a HemoSphere™ Monitor with either a minimally invasive Acumen IQ™ Sensor or a noninvasive Acumen IQ™ Finger Cuff. These technologies also provide access to the following pressure and flow based parameters:

	HPI™	dP/dt	Ea _{dyn}	SVV	PPV	MAP	CO/CI	SV/SVI	SVR/SVRI
Acumen IQ™ sensor	•	•	•	•	•	•	•	•	•
Acumen IQ™ cuff	•	•	•	•	•	•	•	•	•

Acumen HPI™ Software includes the following elements:

- HPI™ Parameter
- HPI™ Smart Alert Popup*
- HPI™ Secondary Screen (two viewing modes)
 - HPI™ Relationship View
 - HPI™ Smart Trend

HPI™ Parameter

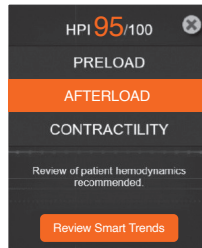
The HPI™ Parameter updates every 20 seconds and displays a unit-less value ranging from 0 to 100. Higher values indicate a greater probability of ensuing hemodynamic instability. Once the HPI™ Value exceeds 85 for two consecutive 20 second intervals, the HPI™ Smart Alert Popup* appears, recommending a review of the patient's hemodynamics. The HPI™ Parameter can be configured as a key parameter, allowing it to be displayed on all monitoring screens. The HPI™ Parameter, its related alerts, and additional screens can be configured according to users' preferences (see operator's manual for instructions).



* HPI™ Smart Alert Popup Displays when Smart Alerts are enabled (default). If Smart Alerts are disabled HPI™ High Alert Popup will appear prompting a review of patient hemodynamics via the HPI™ Secondary Screen

HPI™ Smart Alert Popup

When the HPI™ Value meets alarm criteria (HPI™ Value exceeds 85 for two consecutive 20-second intervals or when it reaches 100 at any time), the HPI™ Smart Alert Popup appears and highlights physiologic mechanisms (preload, afterload, and/or contractility) that may be causing the alarm. Selecting the 'Review Smart Trends' option will lead users to the HPI™ Secondary Screen for further investigation of the underlying hemodynamic parameters. Selecting the 'x' icon in the upper right corner of the popup silences the alarm.



The highlighted physiologic mechanism(s) associated with the HPI™ Alarm is based on configurable settings. The HPI™ Settings (gear wheel) icon can be accessed from the HPI™ Secondary Screen and intervention target triggers can be adjusted based on the patient's hemodynamic profile. For additional details on this and other elements of the Acumen HPI™ Software view the below linked video and/or consult the operator's manual.



Scan the QR code to view the Acumen IQ™ Finger Cuff and Sensor setup on the HemoSphere™ monitor videos

HPI™ Secondary Screen

The advanced hemodynamic pressure and flow parameters provided on the HPI™ Secondary Screen allow users to investigate and identify potential targets for intervention during impending or ongoing hypotensive events.

The HPI™ Secondary Screen can be accessed from the information bar, the HPI™ Key Parameter Tile, the settings icon, or through the HPI™ High Alert or Smart Alert Popup.

The HPI™ Secondary Screen has two viewing modes that users can toggle between:



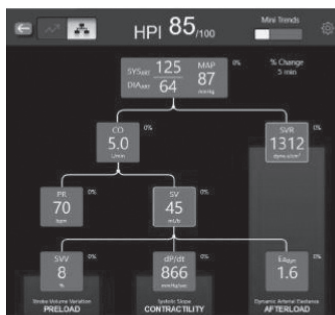
HPI™ Smart Trends Screen

The smart trends screen links a related parameter to each of the three physiologic mechanisms (preload, afterload, and contractility). It also contains graphical trends of these parameters, providing clinicians a view of hemodynamic changes over time, which may aid in identifying potential targets for intervention to prevent or treat hypotension. All intervention target settings or smart alert settings are configurable and can be customized for individualized hemodynamic assessment.



HPI™ Relationship View Screen

The HPI™ Relationship View Screen displays key parameters organized visually to align with the associated physiologic mechanism that they impact. This screen layout is intended to aid clinicians in determining which parameters may be impacting hemodynamic instability.



Either secondary screen viewing mode may be a useful tool to quickly review patient hemodynamics related to an impending or ongoing hypotensive event.

Additional parameters on HPI™ Secondary Screen

- **Stroke volume variation (SVV)** is a sensitive and dynamic measure of fluid responsiveness and should be trended to assess rapid changes over time. When used within its limits (see page 5.6) it can predict if fluid administration will increase stroke volume. SVV is a percentage, calculated

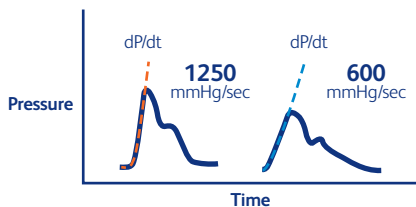
from: $SV_{\max} - SV_{\min} / SV_{\text{mean}}$

- **Pulse pressure variation (PPV)** is a sensitive and dynamic measure of fluid responsiveness, similar to SVV. PPV is also a percentage, calculated from: $PP_{\max} - PP_{\min} / PP_{\text{mean}}$

- **Systolic slope (dP/dt)**

is the maximum upslope of the arterial pressure waveform from a peripheral artery; its units are mmHg/sec. dP/dt is an indicator of left ventricular

contractility. dP/dt is not an absolute value, it is a dynamic measure that should be trended over time to assess LV contractility*



- **Dynamic arterial elastance, Ea_{dyn}** , is an estimate of arterial elastance, which contributes to the afterload experienced by the left ventricle and represents the complex interaction between the LV and the large arterial vessels; Ea_{dyn} is a unit-less value that is a ratio of PPV/SVV

* dP/dt has not been studied as a measure of LV contractility in all patient populations

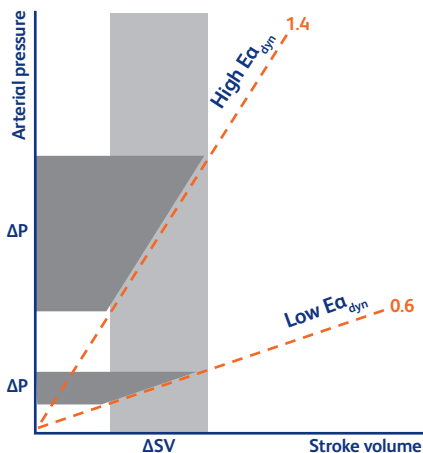
Ea_{dyn} has utility in both mechanically ventilated and spontaneously breathing patients who are fluid responsive.

Ea_{dyn} can predict if a fluid responsive patient's MAP will increase with the administration of fluids. Ea_{dyn} values >1.0 can predict that fluid administration will increase a patient's MAP. Ea_{dyn} values <0.8 , in patients who are fluid responsive, will likely increase their SV with fluid administration, but their MAP will not increase. Values between 0.8 - 1.2 represent a 'grey zone', where fluid administration may or may not increase MAP.

Ea_{dyn} is best used in conjunction with SVV, SV, and/or CO assessments in mechanically ventilated patients. Additionally, Ea_{dyn} has proven helpful in guiding vasopressor titration.

Ea_{dyn} applications:

- $Ea_{dyn} > 1.0$ has been shown to predict that blood pressure will increase in response to fluid administration in fluid responsive patients
- $Ea_{dyn} < 1.0$ has been shown to predict whether blood pressure will remain stable when weaning norepinephrine in vasoplegic states



SVV, PPV, dP/dt , and Ea_{dyn} are intended as integrative decision support parameters and provide additional insights to help clinicians mitigate and manage hemodynamic instability.

Acumen Assisted Fluid Management™ (AFM™) Software

Acumen AFM™ Software provides clinical decision support designed to guide optimal intravenous fluid administration. It can be used in surgical patients being monitored with an Acumen IQ™ Sensor that is connected to a radial arterial line and a compatible HemoSphere™ Monitor.

Acumen AFM™ Software includes a rule-based algorithm that makes fluid management suggestions based on the following inputs:

- Fluid strategy (desired % change in SV)
- Surgery mode (open vs. laparoscopic/prone)
- Hemodynamic data from the Acumen IQ™ Sensor* (PR, MAP, SV, SVV, SVR, and rate of SV change over the past two minutes)
- Fluid delivery data (start/stop time and amount delivered)
- Patient's past responses to previous fluid bolus administrations
- Patient population model[†]

Fluid administration suggestions are intended to assist clinicians in maintaining patients within their optimal fluid range. The algorithm's fluid administration suggestions are based on its recognition of patterns of fluid responsiveness in a given patient.

For additional details refer to the HemoSphere™ Monitor operator's manual.

* These inputs are obtained automatically from the HemoSphere™ Monitor



Shock States

Shock is defined as a state of cellular and tissue hypoxia due to either reduced oxygen delivery, increased oxygen consumption, inadequate oxygen utilization, or a combination of these processes.

Types of Shock

Hypovolemic Shock

Circulating volume loss that leads to a decrease in preload and cardiac output, resulting in decreased blood flow to the tissues. Common causes of hypovolemic shock include*: blood loss from trauma, GI bleed, burns, and vomiting.

Cardiogenic Shock

Loss of ventricular contractility leads to decreased cardiac output, which impairs blood flow to the tissues. Common causes of cardiogenic shock include*: myocardial ischemia or infarct, and drug toxicity.

Distributive Shock

Profound vasodilatation of the systemic vasculature affects the body's ability to auto-regulate perfusion to vital organs. Common causes include*: sepsis, neurologic/spinal cord injury, and allergic reaction.

Obstructive Shock

Impediment to blood returning to the heart or being ejected from the ventricles impairs cardiac output, which decreases blood flow to the tissues. Common causes of obstructive shock include*: pulmonary embolism, tension pneumothorax, and pericardial tamponade.

Mixed shock states

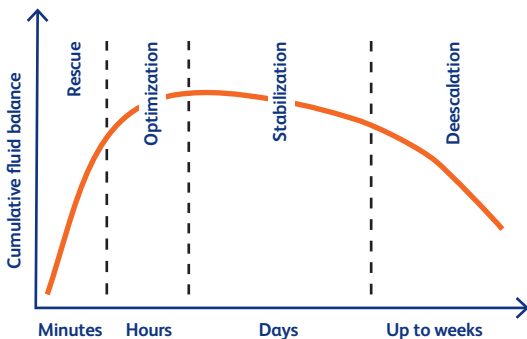
It is important to note that mixed presentations of shock are common. For example, an aortic dissection could induce all four types of shock simultaneously: hypovolemic shock from excessive blood loss, cardiogenic shock from lack of perfusion to the myocardium causing ischemia, the potential to produce obstructive shock if the dissection produces pericardial tamponade, and if left untreated, shock and tissue hypoperfusion ultimately causes vasodilation leading to distributive shock.

Presentation of patients in shock

Proper treatment of shock is dependent upon accurately identifying the underlying shock state. A clinical assessment that includes obtaining labs, pertinent imaging, hemodynamic assessment, risk stratification, and expert treatment are critical to patient outcomes.

Early hemodynamic support of patients in shock is crucial to preventing worsening organ dysfunction and failure.

Four phases of hemodynamic treatment in relation to cumulative fluid balance



Type of shock	MAP	CO/SV	CVP	MPAP	PCWP	SVR	SvO ₂	SVV	StO ₂	HPI™	dP/dt	Ea _{dyn}
Hypovolemic	↓↔↑	↓	↓	↓	↓	↑	↓	↑	↓	↑	↕↑	↑
Cardiogenic	↓↔↑	↓	↑	↑	↑	↑	↓	↓	↓	↑	↓	↕↑
Distributive	↓	↑	↓	↓	↓	↓	↑	↑	↑	↑	↑	↓
Obstructive	↓	↓	↑	↑	↕↑	↕↑	↓	↕↑	↓	↑	↑	↕↑

↑ Increased ↓ Decreased → No change

The table on page 8.3 shows the effects the various shock states commonly have on measured parameters, including those available from advanced hemodynamic monitoring technologies.

Treatments

Treating shock involves recognizing and addressing its underlying cause(s) and supporting oxygen delivery until recovery ensues. The contents of this guide are intended to assist clinicians in identifying, addressing, and optimizing patients' hemodynamics as they face physiologic challenges.



Quick Reference

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



Normal Hemodynamic Parameters

Parameter	Equation	Normal range
Arterial Blood Pressure (BP)	Systolic (SBP) Diastolic (DBP)	90-140 mmHg 60-90 mmHg
Mean Arterial Pressure (MAP)	$[SBP + (2 \times DBP)]/3$	70-105 mmHg
Right Atrial Pressure (RAP) Central Venous Pressure (CVP)		2-6 mmHg (CVP normal range is equivalent to RAP)
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)	$[PASP + (2 \times PADP)]/3$	10-20 mmHg
Pulmonary Artery Occlusion Pressure (PAOP)		6-12 mmHg
Left Atrial Pressure (LAP)		6-12 mmHg
Cardiac Output (CO)	$HR \times SV/1000$	4-8 L/min
Cardiac Index (CI)	CO/BSA	2.5-4 L/min/m ²
Stroke Volume (SV)	$CO/HR \times 1000$	60-100 mL/beat
Stroke Volume Index (SVI)	$CI/HR \times 1000$	33-47 mL/m ² /beat
Stroke Volume Variation (SVV)	$100 \times (SV_{max} - SV_{min}) / \text{mean } (SV)$	<10-15%
Systemic Vascular Resistance (SVR)	$MAP - RAP \times 80 / CO$	800-1200 dynes/sec/cm ⁵
Systemic Vascular Resistance Index (SVRI)	$MAP - RAP \times 80 / CI$	1970-2390 dynes/sec/cm ⁵ /m ²
Pulmonary Vascular Resistance (PVR)	$MPAP - PAOP \times 80 / CO$	100-250 dynes/sec/cm ⁵
Pulmonary Vascular Resistance Index (PVRI)	$MPAP - PAOP \times 80 / CI$	255-285 dynes/sec/cm ⁵ /m ²

Additional hemodynamic parameters

Parameter	Equation	Normal range
Cardiac Power Index (CPI)	$MAP \times CI \times 0.0022$	0.5-0.7 W/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)	$RVEDV/1.7$	59-94 mL/m ²
Right Ventricular End-Systolic Volume (RVESV)	EDV - SV	50-100 mL
Right Ventricular Ejection Fraction (RVEF)	SV/EDV	40-60%
Left Ventricular Stroke Work Index (LVSWI)	$SVI \times (MAP - PAOP) \times 0.0136$	50-62 g·m/m ² /beat
Right Ventricular Stroke Work Index (RVSWI)	$SVI \times (MPAP - RAP) \times 0.0136$	5-10 g·m/m ² /beat

Normal Hemodynamic Parameters

Normal oxygenation parameters

Parameter	Equation	Normal range
Arterial Oxygen Saturation (SaO ₂)		95-100%
Mixed Venous Saturation (SvO ₂)		60-80%
Cerebral Tissue Oxygen Saturation (StO ₂)		60-80%
Arterial Oxygen Content (CaO ₂)	$(0.0138 \times \text{Hb} \times \text{SaO}_2) + (0.0031 \times \text{PaO}_2)$	17-20 mL/dL
Venous Oxygen Content (CvO ₂)	$(0.0138 \times \text{Hb} \times \text{SvO}_2) + (0.0031 \times \text{PvO}_2)$	12-15 mL/dL
A- V Oxygen Content Difference [C(a-v)O ₂]	$\text{CaO}_2 - \text{CvO}_2$	4-6 mL/dL
Oxygen Delivery (DO ₂)	$\text{CaO}_2 \times \text{CO} \times 10$	950-1150 mL/min
Oxygen Delivery Index (DO ₂ I)	$\text{CaO}_2 \times \text{CI} \times 10$	500-600 mL/min/m ²
Oxygen Consumption (VO ₂)	$\text{C(a-v)O}_2 \times \text{CO} \times 10$	200-250 mL/min/m ²
Oxygen Consumption Index (VO ₂ I)	$\text{C(a-v)O}_2 \times \text{CI} \times 10$	120-160 mL/min/m ²
Oxygen Extraction Ratio (O ₂ ER)	$[(\text{CaO}_2 - \text{CvO}_2) / \text{CaO}_2] \times 100$	22-30%
Oxygen Extraction Index (O ₂ EI)	$[(\text{SaO}_2 - \text{SvO}_2) / \text{SaO}_2] \times 100$	20-25%

Normal blood laboratory values

Test	Convention units (reference values*)	SI units
Hematocrit (Hct)	Males: 41-50% Females: 35-45%	0.41-0.50 0.35-0.45
Hemoglobin (Hgb)	Males: 13.5-17.5 g/dL Females: 12.0-15.5 g/dL	135-175 g/L 120-155 g/L
Lactate	0.93-1.65 mEq/L	0.93-1.65 mmol/L

SI Units = International Units

*Reference Values vary by regional laboratory techniques and methods.

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 $\text{pH} < 7.35$.

Alkalemia: An alkalosis (base) condition of the blood with decreased hydrogen ions and $\text{pH} > 7.45$.

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

Swan-Ganz™ Catheter Computation Constants

Thermodilution (Intermittent) Cardiac Output

Swan-Ganz™ Catheter model	Injectate Temperature								
	Cold (0-5°C)			Room Temperature (19-22°C)			Room Temperature (23-25°C)		
	10cc	5cc	3cc	10cc	5cc	3cc	10cc	5cc	3cc
139F75, 177F75N, 744F75, 774F75	0.564	0.257	0.143	0.582	0.277	0.156	0.594	0.283	–
746F8, 777F8	0.550	0.256	–	0.582	0.282	–	0.600	0.292	–
780F75M, 782F75M	0.564	0.257	0.143	0.582	0.277	0.156	0.607	0.294	0.170
631F55N	–	0.265	0.148	–	0.294	0.172	–	0.308	0.183
TS105F5	0.542	0.255	0.143	–	–	–	0.605	0.297	0.175
132F5	–	0.270	0.154	–	0.292	0.170	–	0.307	0.181
096F6	0.547	0.259	0.144	0.582	0.280	0.161	0.608	0.305	0.180
131F7	0.542	0.247	0.132	0.578	0.274	0.154	0.595	0.287	0.165
141F7	0.542	0.247	0.132	0.578	0.274	0.154	0.595	0.287	0.165
151F7	0.542	0.247	0.132	0.578	0.274	0.154	0.595	0.287	0.165
143TF7	0.544	0.259	–	0.587	0.286	–	0.599	0.291	–
831F75	0.564	0.262	0.139	0.588	0.283	0.158	0.612	0.301	0.177
834F75	0.564	0.257	0.143	0.582	0.277	0.156	0.607	0.294	0.170
C145F6N	0.547	0.252	0.134	0.579	0.277	0.156	0.597	0.295	0.169
C144F7, S144F7	0.547	0.252	0.134	0.579	0.277	0.156	0.597	0.295	0.169
T173F6	0.547	0.252	0.134	0.579	0.277	0.156	0.597	0.295	0.169
931F75	0.564	0.262	0.139	0.588	0.283	0.158	0.612	0.301	0.177
991F8	0.568	0.268	0.147	–	–	–	0.616	0.302	0.176
D200F7	0.542	0.247	0.132	0.578	0.274	0.154	–	–	–

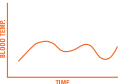
Swan-Ganz™ Catheter Computation Constants

CO-Set+ Closed Injectate Delivery System

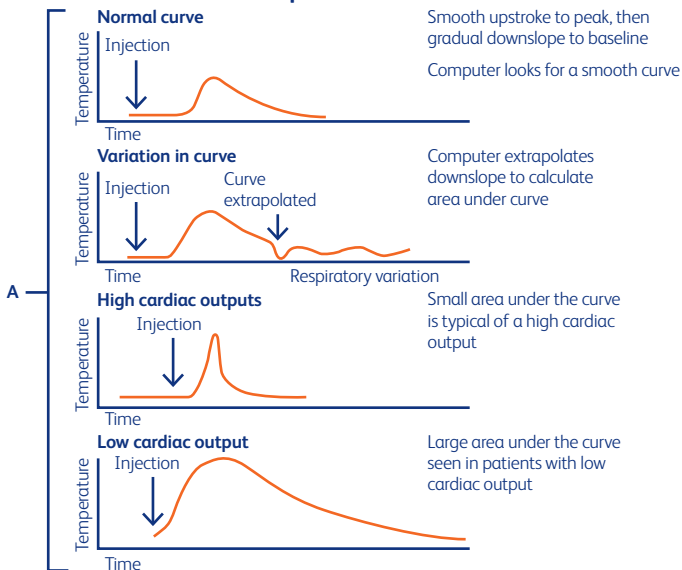
Swan-Ganz™ Catheter model	Injectate Temperature			
	Cold		Room Temperature	
	10cc (6-12°C)	5cc (8-16°C)	10cc (18-25°C)	5cc (18-25°C)
139F75, 177F75N, 744F75, 774F75	0.574	0.287	0.595	0.298
746F8, 777F8	0.559	0.263	0.602	0.295
780F75M, 782F75M	0.574	0.287	0.595	0.298
631F55N	–	0.284	–	0.306
TS105F5	0.552	0.265	0.589	0.289
132F5	–	0.285	–	0.307
096F6	0.558	0.277	0.607	0.301
131F7	0.561	0.259	0.608	0.301
141F7	0.561	0.259	0.608	0.301
151F7	0.561	0.259	0.608	0.301
143TF7	0.569	0.266	0.589	0.287
831F75	0.578	0.272	0.592	0.290
834F75	0.574	0.287	0.585	0.298
C145F6N	0.570	0.271	0.585	0.287
C144F7, S144F7	0.570	0.271	0.585	0.287
T173F6	0.570	0.271	0.585	0.287
931F75	0.578	0.272	0.592	0.290
991F8	0.553	0.277 (8-12°C)	0.607	0.295
D200F7	0.561	0.259	0.608	0.301

Troubleshooting Key Factors in Optimizing Bolus CO Determinations

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

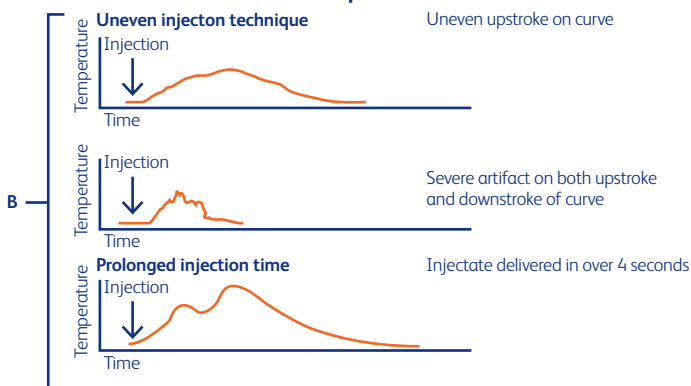
	Factor Affecting Accuracy of Bolus CO Measurement	Potential Error
	Inaccurate Injectate Temperature: • 1°C error in iced injectate • 1°C error in room temperature injectate If injectate is removed from the ice bath for: • 15 seconds • 30 seconds	± 2.7% ± 7.7% Mean increase of $0.34 \pm 0.16^{\circ}\text{C}$ Mean increase of $0.56 \pm 0.18^{\circ}\text{C}$
	Inaccurate Injectate Volume	0.5 mL of error in 5 mL injection: ± 10% 0.5 mL of error in 10 mL injection: ± 5%
	Rapid Volume Infusion During Bolus Injections: • Room temperature infusion • Warmed infusion inspiration (or expiration)	CO decreased 30–80% CO decreased 20–40%
	Respiratory Cycle Influences • Time of injection during	Normal variance of 20% Maximum variance up to 70%
	Inaccurate Computation Constant	1–100%
	Thermal Instability Post Cardiopulmonary Bypass (CPB): • 1–10 minutes post • 30 minutes post	10–20% Up to 9%

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.)

Pulmonary shunt equation

Q_s/Q_t is calculated as follows:

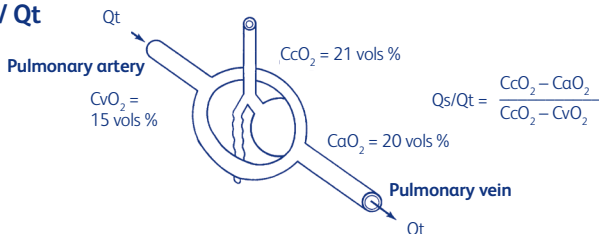
$$Q_s/Q_t = \frac{CcO_2 - CaO_2}{CcO_2 - CvO_2}$$

CcO_2 = Capillary oxygen content
 $(1.38 \times \text{Hgb} \times 1) + (\text{PAO}_2 \times 0.003)$

CaO_2 = Arterial oxygen content
 $(1.38 \times \text{Hgb} \times \text{SaO}_2) + (\text{PaO}_2 \times 0.003)$

CvO_2 = Venous oxygen content
 $(1.38 \times \text{Hgb} \times \text{SvO}_2) + (\text{PvO}_2 \times 0.003)$

Q_s / Q_t



Idealized version of the heart and lungs depicting 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 (Q_s/Q_t).

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 $\text{SaO}_2 < 100\%$
- 2 Poor agreement with Q_s/Q_t if $\text{PaO}_2 > 99$ mmHg
- 3 Good correlation when $Q_s/Q_t > 15\%$

Equation derivations

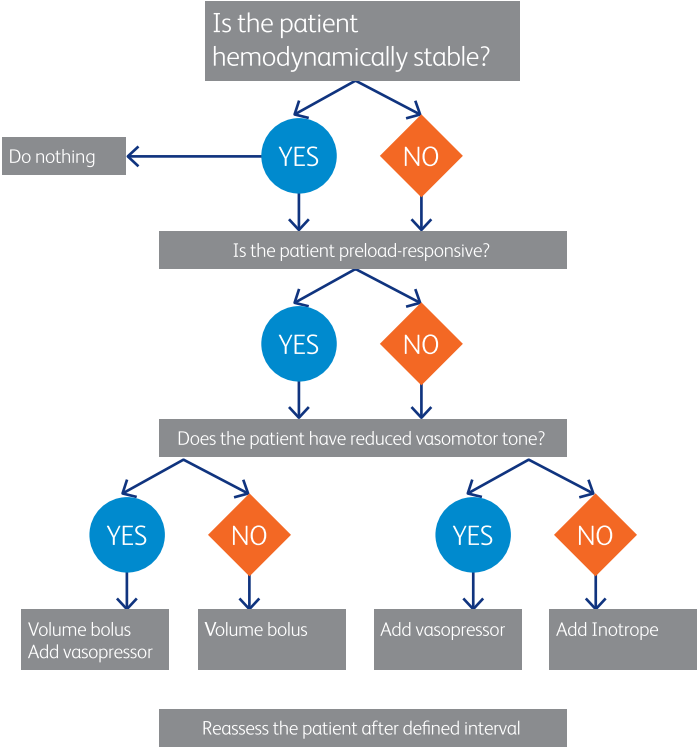
$$Q_s/Q_t = \frac{100 \times [(1.38 \times \text{Hgb}) + (0.003 \times \text{PAO}_2) - \text{CaO}_2]}{[(1.38 \times \text{Hgb}) + (0.003 \times \text{PAO}_2) - \text{CvO}_2]}$$

$$\text{VQI} = \frac{100 \times [1.38 \times \text{Hgb} \times (1 - \text{SaO}_2 / 100) + (0.003 \times \text{PAO}_2)]}{[(1.38 \times \text{Hgb}) + (0.003 \times \text{PAO}_2) - \text{CvO}_2]}$$

Dual oximetry, simplifies the shunt equation

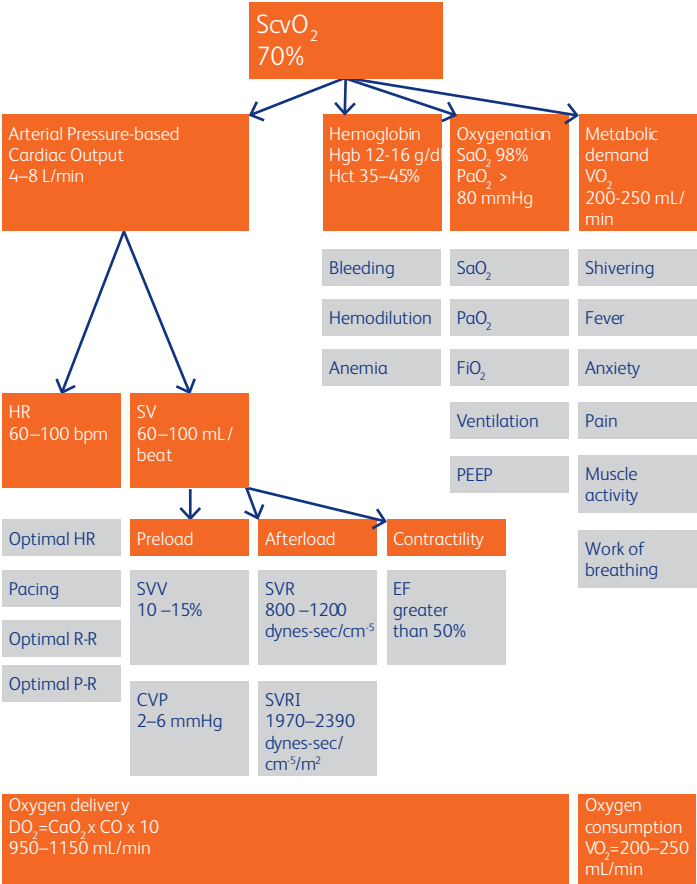
$$\text{VQI} = \frac{\text{SAO}_2 - \text{SaO}_2 = 1 - \text{SaO}_2}{\text{SAO}_2 - \text{SvO}_2 = 1 - \text{SvO}_2} \quad \text{or} \quad \frac{1 - \text{SpO}_2}{1 - \text{SvO}_2}$$

Functional Hemodynamic Monitoring Protocol



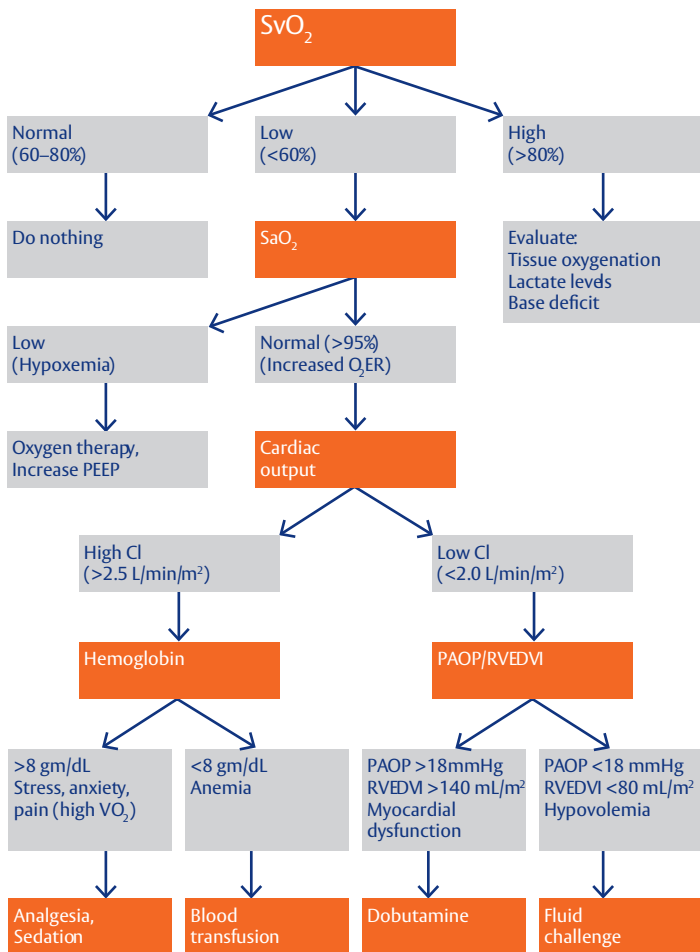
Schematic flow chart for the decision tree in the cardiovascular management of patients who are hemodynamically unstable using functional hemodynamic monitoring protocols. This protocol application is part of a patented treatment algorithm co-owned by the University of Pittsburgh and Michael R. Pinsky,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

Advanced Minimally-Invasive Physio-relationship Graphic



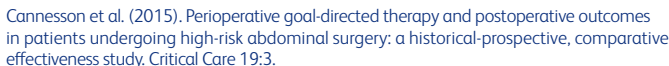
Advanced Swan-Ganz™ Catheter Goal-Directed Protocol

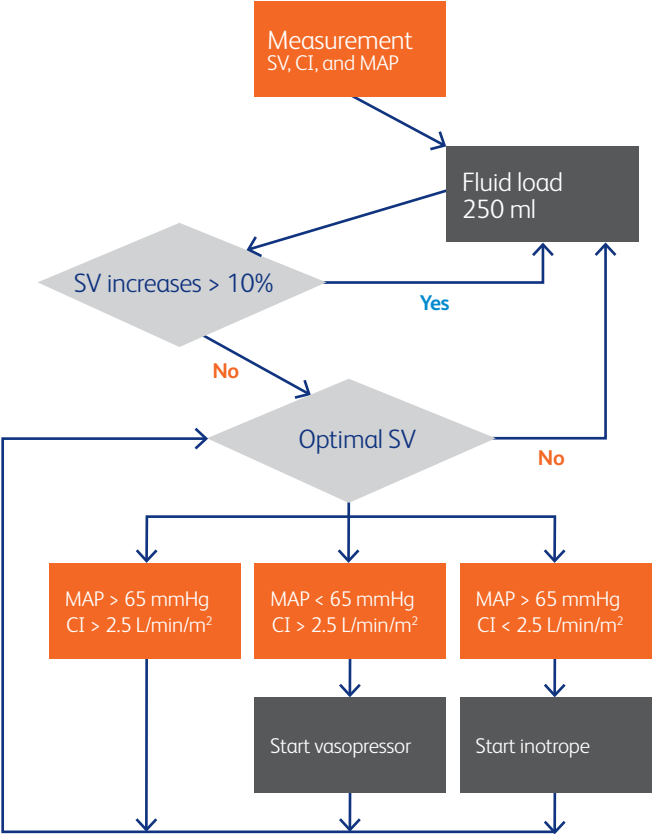
Resuscitate to a mean arterial pressure of >65 mmHg



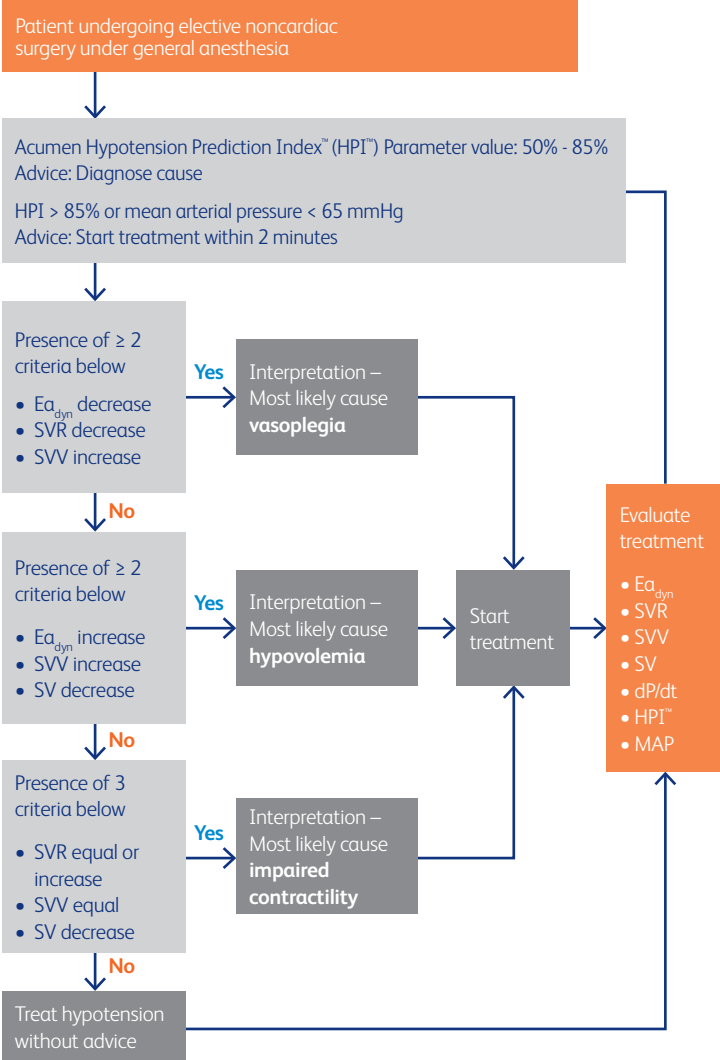
Quick Reference

9.13





HYPE Protocol



Wijnberge et al.(2020). Effect of a machine learning-derived early warning system for intraoperative hypotension vs standard care on depth and duration of intraoperative hypotension during elective noncardiac surgery. JAMA.com



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