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Edwards Critical Care Education

QUICK GUIDE TO

Cardiopulmonary Care

3RD EDITION

Edwards

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QUICK GUIDE TO

Cardiopulmonary Care

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Quick Guide to Cardiopulmonary Care

Pertinent Clinical Information Dedicated to the Critical Care Clinician

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

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

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

In this edition, the order of content reflects current concepts in assessment strategies and technology enhancements in which to monitor the patient. Additionally, pertinent sections of the Quick Guide to Central Venous Access have been incorporated to make this edition a more comprehensive reference guide.

The Quick Guide is organized into sections that build upon physiologic rationale. The first section begins with a review of oxygen delivery and consumption, including the determinants, implications of an imbalance, and the monitoring tools available.

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

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

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

Quick Guide to Cardiopulmonary Care

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ADVANCED MINIMALLY-INVASIVE MONITORING

SWAN-GANZ CATHETERS – ADVANCED AND STANDARD TECHNOLOGY

REFERENCES

Anatomy and Physiology

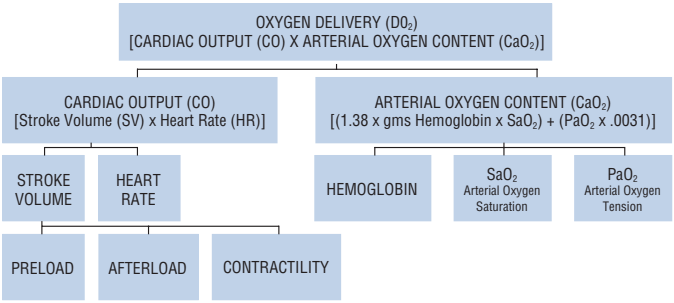
ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972

Anatomy and Physiology

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

Oxygen Delivery ($DO_2 = CO_2$ x CO x 10)

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



Oxygen Content (CO_2): amount of oxygen carried in the blood, both arterial and venous:

$$(1.38 \times Hgb \times SO_2) + (0.0031 \times PO_2)$$

1.38: amount of O_2 that can combine with 1 gram of hemoglobin
0.0031: solubility coefficient of O_2 in the plasma*

$$CaO_2 = (1.38 \times Hgb \times SaO_2) + (0.0031 \times PaO_2)$$
Normal 20.1 mL/dL

$$CvO_2 = (1.38 \times Hgb \times SvO_2) + (0.0031 \times PvO_2)$$
Normal 15.5 mL/dL

Oxygen Delivery (DO_2): amount of oxygen transported in blood to tissues. Both arterial and venous O_2 delivery can be measured:

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

Venous oxygen return (DvO_2): $CO \times CvO_2 \times 10$
5 L/min x 15.5 mL/dL x 10 = 775 mL/min

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

Oxygen Consumption

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

OXYGEN CONSUMPTION
Oxygen Consumption (VO_2) = Oxygen Delivery – Venous Oxygen Return

OXYGEN DELIVERY (DO_2)
[Cardiac output (CO) x
Arterial Oxygen Content (CaO_2)]
(CO) x (1.38 x 15 x SaO_2) + (PaO_2 x .0031)
5 x 20.1 =
NORMAL = 1005 mL O_2/min

VENOUS OXYGEN RETURN
[Cardiac output (CO) x
Venous Oxygen Content (CvO_2)]
(CO) x (1.38 x 15 x SvO_2) + (PvO_2 x .0031)
5 x 15.5 =
NORMAL = 775 mL O_2/min

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

Oxygen Consumption (VO_2)

Arterial Oxygen Transport – Venous Oxygen Transport

$VO_2 = (CO \times CaO_2) - (CO \times CvO_2)$
$= CO\ (CaO_2 - CvO_2)$
$= CO\ [(SaO_2 \times Hgb \times 13.8) - (SvO_2 \times Hgb \times 13.8)]$
$= CO \times Hgb \times 13.8 \times (SaO_2 - SvO_2)$

Normals: 200 – 250 mL/min
120 – 160 mL/min/m^2

Note: 13.8 = 1.38 x 10

CONDITIONS AND ACTIVITIES ALTERING DEMAND AND VO_2

Fever (one degree C)	10%	Work of breathing	40%
Shivering	50-100%	Post op procedure	7%
ET suctioning	7-70%	MSOF	20-80%
Sepsis	50-100%	Dressing change	10%
Visitor	22%	Bath	23%
Position change	31%	Chest X-Ray	25%
Sling scale weighing	36%		

Other Assessment Parameters for Oxygen Utilization

Arterial-Venous Oxygen Difference

$C(a\text{-}v)O_2$: normally 5 vol %

20 vol % – 15 vol % = 5 vol %

Note: Vol% or mL/dL

Oxygen Extraction Ratio

O_2ER: normally 22 – 30%

O_2ER: $CaO_2 – CvO_2 / CaO_2 \times 100$

$CaO_2 = 20.1$ $CvO_2 = 15.6$

$O_2ER = 20.1 – 15.6 / 20.1 \times 100 = 22.4\%$

Oxygen Extraction Index

Dual oximetry estimates oxygen extraction ratio. Evaluates the efficiency of oxygen extraction. Reflects cardiac reserve to increases in O_2 demand. Normal range is 20%–30%.

$O_2EI = SaO_2 – SvO_2 / SaO_2 \times 100$ ($SaO_2 = 99$, $SvO_2 = 75$)

$O_2EI = 99 – 75 / 99 \times 100 = 24.2\%$

CO vs SvO_2 Correlations

SvO_2 reflects balance between oxygen delivery and utilization relationship to Fick equation.

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

$CO = VO_2 / C(a – v)O_2$

$C(a – v)O_2 = VO_2 / (CO \times 10)$

$S(a – v)O_2 = VO_2 / (CO \times 10)$

When Fick equation is rearranged, the determinants of SvO_2 are the components of oxygen delivery and consumption:

If $SaO_2 = 1.0$, then $SvO_2 = CvO_2 / CaO_2$

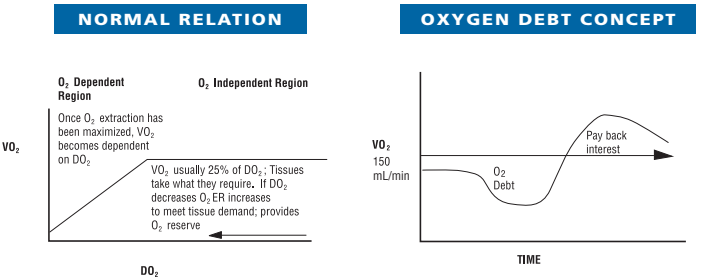
$SvO_2 = 1 – [VO_2 / (CO \times 10 \times CaO_2)]$

$SvO_2 = 1 – (VO_2 / DO_2) \times 10$

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

VO_2/DO_2 Relationships

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



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

Factors Influencing Accumulation of O_2 Debt

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

Functional Anatomy

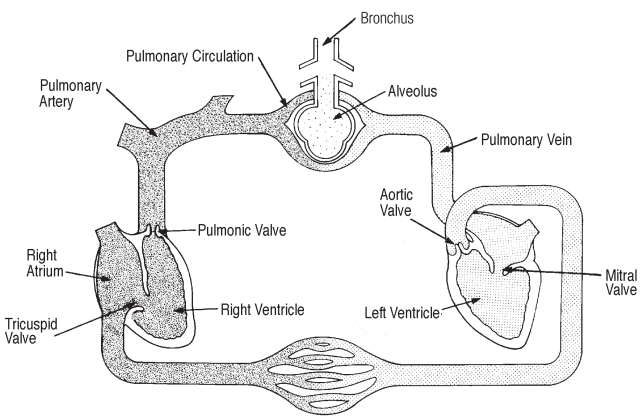
For hemodynamic monitoring purposes, the right and left heart are differentiated as to function, structure and pressure generation. The pulmonary capillary bed lies between the right and left heart. The capillary bed is a compliant system with a high capacity to sequester blood.

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

RIGHT AND LEFT HEART DIFFERENCES

Right Heart	Left Heart
Receives deoxygenated blood	Receives oxygenated blood
Low pressure system	High pressure system
Volume pump	Pressure pump
RV thin and crescent shape	LV thick and conical shape
Coronary perfusion biphasic	Coronary perfusion during diastole

ANATOMICAL STRUCTURES



Coronary Arteries and Veins

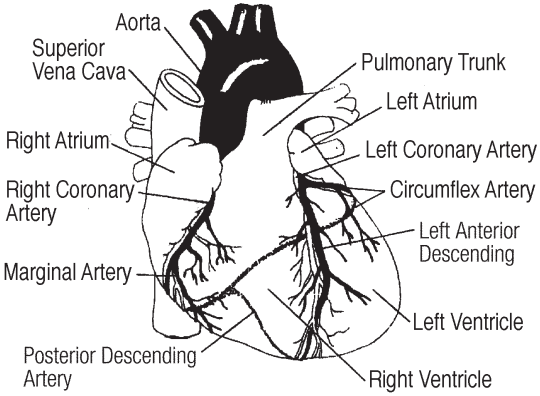
The two major branches of the coronary arteries arise from each side of the aortic root. Each coronary artery lies in the atrioventricular sulcus and is protected by a layer of adipose tissue.

Major Branches	Areas Supplied
Right Coronary Artery (RCA)	Sinus Node 55%, AV Node 90%, Bundle of His (90%) RA, RV free wall Portion of IVS
Posterior Descending Branch (Provided by RCA ≥ 80%)	Portion of IVS Diaphragmatic aspect of LV
Left Main Coronary Artery Bifurcates	
Left Anterior Descending (LAD)	Left anterior wall Anterior portion of IVS Portion of right ventricle
Left Circumflex (Provides posterior descending branch ≤ 20%)	Sinus node 45%, LA, 10% AV node Lateral and posterior wall of LV

Coronary Veins	Location Drains Into
Thebesian Veins	Directly into R and L ventricles
Great Cardiac Vein	Coronary sinus in the RA
Anterior Cardiac Veins	RA

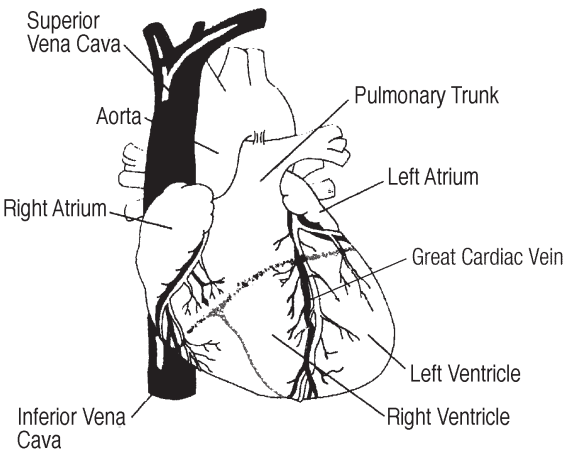
CORONARY ARTERIES

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



CORONARY VEINS

Blood is drained by branches of the cardiac veins.

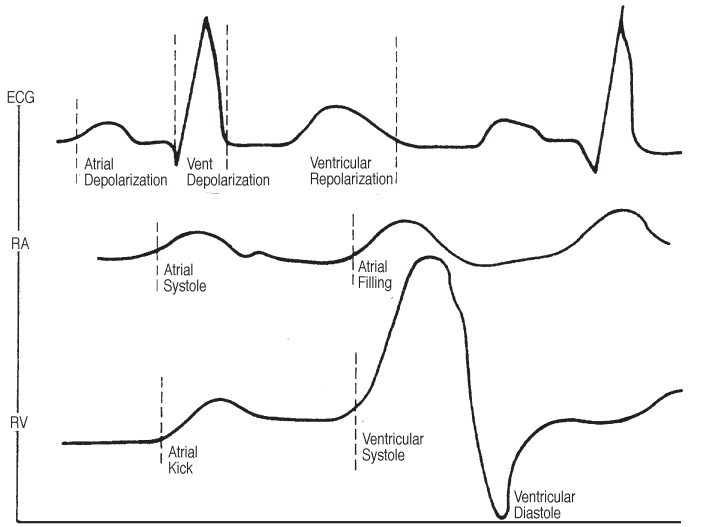


Cardiac Cycle: Electrical Correlation to Mechanical

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

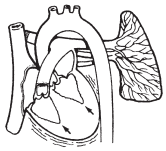
The next electrical activity is repolarization which results in the relaxation of the muscle fibers and produces diastole. The time difference between the electrical and mechanical activity is called electro-mechanical coupling, or the excitation-contraction phase. A simultaneous recording of the ECG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

ELECTRICAL – MECHANICAL CARDIAC CYCLE

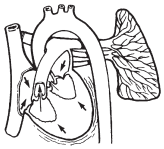


Mechanical Cardiac Cycle Phases

SYSTOLE



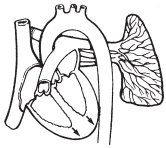
1. Isovolumetric Phase
Follows QRS of ECG
All valves closed
Majority of oxygen consumed



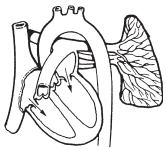
2. Rapid Ventricular Ejection
Aortic valve opens
Occurs during ST segment
2/3 or more of blood volume ejected

3. Reduced Ventricular Ejection
Occurs during "T" wave
Atria are in diastole
Produces "v" wave in atrial tracing

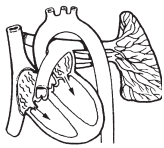
DIASTOLE



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



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

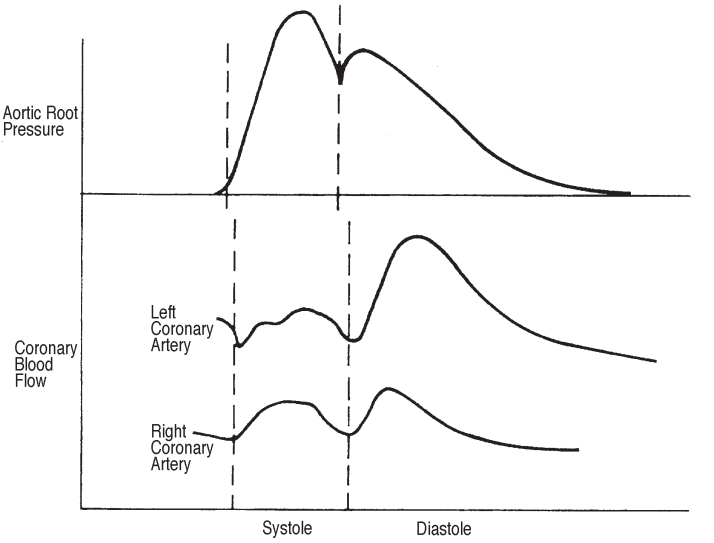


3. Slow Filling Phase: End-Diastole
Atrial "kick"
Follows "P" wave during sinus rhythms
Atrial systole occurs
Produces "a" wave on atrial tracings
Remaining volume goes into ventricle

Coronary Artery Perfusion

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

CORONARY ARTERY PERFUSION



Cardiac Output Definition

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

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

Normal Cardiac Output: 4 – 8 L/min

Normal Cardiac Index : 2.5 – 4 L/min/m^2

CI	= CO/BSA
BSA	= Body Surface Area

Normal Heart Rate Range: 60 – 100 BPM

Normal Stroke Volume: 60 – 100 mL/beat

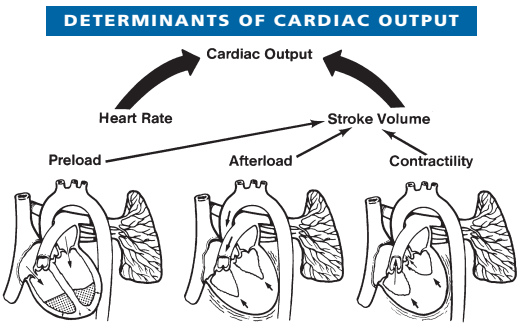
Stroke volume: difference between end-diastolic volume (EDV), [the amount of blood in the ventricle at the end of diastole]; and end-systolic volume (ESV), [blood volume in the ventricle at the end of systole]. Normal SV is 60 to 100 mL/beat.

SV = EDV – ESV SV also calculated by: **SV = CO / HR x 1000**

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

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

EF = (SV / EDV) x 100



Preload Definition and Measurements

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

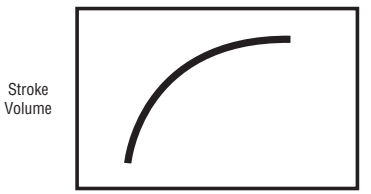
Preload

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

Frank–Starling Law

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

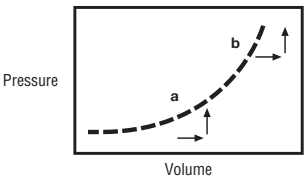
FRANK–STARLING CURVE



Ventricular Compliance Curves

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

EFFECTS OF VENTRICULAR COMPLIANCE

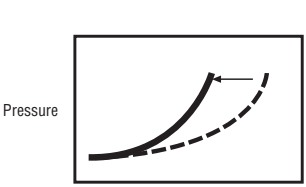


Normal Compliance

Pressure/volume relationship is curvilinear:

a: Large increase in volume = small increase in pressure

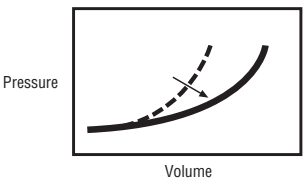
b: Small increase in volume = large increase in pressure



Decreased Compliance

Stiffer, less elastic ventricle

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



Increased Compliance

Less stiff, more elastic ventricle

Dilated cardiomyopathies
Decreased afterload
Vasodilators

Afterload Definition and Measurements

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

Afterload

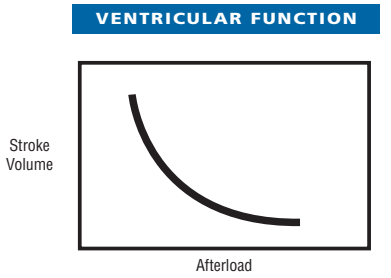
Pulmonary Vascular Resistance (PVR): <250 dynes - sec - cm^{-5}

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

Systemic Vascular Resistance (SVR): 800-1200 dynes - sec - cm^{-5}

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

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



Contractility Definition and Measurements

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

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

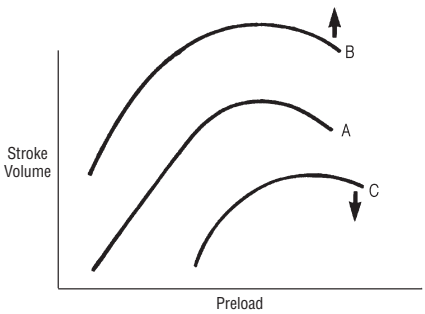
Measurements of contractility cannot be directly obtained. Clinical assessment parameters are surrogates and all include determinants of preload and afterload.

Contractility

Stroke Volume SV = (CO x 1000)/HR	60 – 100 mL/beat
SVI = SV/BSA	33 – 47 mL/beat/m^2
Left Ventricular Stroke Work Index LVSWI = SVI (MAP – PAOP) x 0.0136	50 – 62 g/m^2/beat
Right Ventricular Stroke Work Index RVSWI = SVI (PA mean – CVP) x 0.0136	5 – 10 g/m^2/beat

VENTRICULAR FUNCTION CURVES

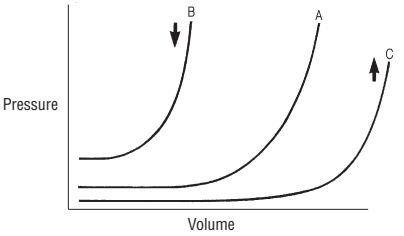
A: Normal Contractility
B: Increased Contractility
C: Decreased Contractility



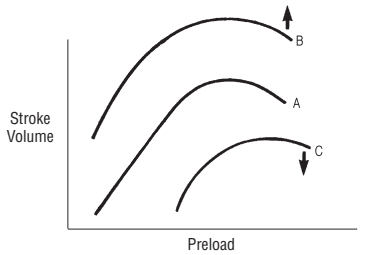
Family of Ventricular Function Curves

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

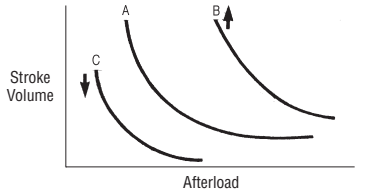
VENTRICULAR FUNCTION CURVES



A: Normal Compliance
B: Decreased Compliance
C: Increased Compliance



A: Normal Contractility
B: Increased Contractility
C: Decreased Contractility



A: Normal Contractility
B: Increased Contractility
C: Decreased Contractility

Pulmonary Function Tests

Definitions:

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

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

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

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

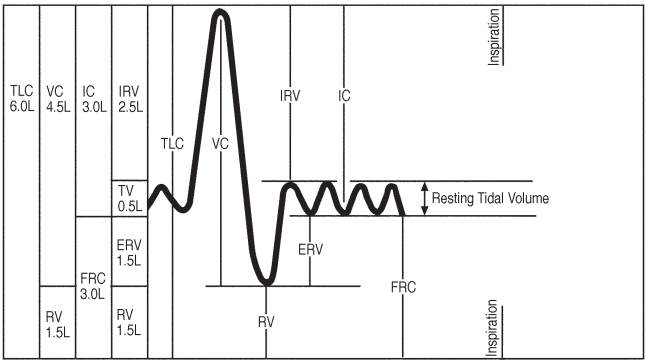
Expiratory Reserve Volume (ERV): maximal amount of air that can be exhaled from the resting level following a normal expiration. (~1.1L)

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

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

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

NORMAL SPIROGRAM



Acid Base Balance

Arterial Blood Gas Analysis

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

Definitions

Acid: A substance which can donate hydrogen ions

Base: A substance which can accept hydrogen ions

pH: The negative logarithm of H^+ ion concentration

Acidemia: An acid condition of the blood with pH < 7.35

Alkalemia: An alkaline (base) condition of the blood with pH > 7.45

PCO_2: Respiratory Component

$PaCO_2$: Normal ventilation 35 – 45 mmHg
Hypoventilation > 45 mmHg
Hyperventilation < 35 mmHg

HCO_3: Metabolic Component
Balanced 22 – 26 mEq/L
Base Balance -2 to +2
Metabolic Alkalosis > 26 mEq/L
Base excess > 2 mEq/L
Metabolic Acidosis < 22 mEq/L
Base deficit < 2 mEq/L

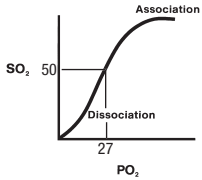
Normal Blood Gas Values

Component	Arterial	Venous
pH	7.40 (7.35 – 7.45)	7.36 (7.31 – 7.41)
PO_2 (mmHg)	80 – 100	35 – 45
SO_2 (%)	≥ 95	60 – 80
PCO_2 (mmHg)	35 – 45	42 – 55
HCO_3 (mEq/L)	22 – 26	24 – 28
Base excess/deficit	-2 – +2	-2 – +2

Oxyhemoglobin Dissociation Curve

The oxyhemoglobin dissociation curve (ODC) graphically illustrates the relationship that exists between the partial pressure (PO_2) of oxygen and oxygen saturation (SO_2). The sigmoid-shaped curve can be divided into two segments. The association segment or upper portion of the curve represents oxygen uptake in the lungs or the arterial side. The dissociation segment is the lower portion of the curve and represents the venous side, where oxygen is released from the hemoglobin.

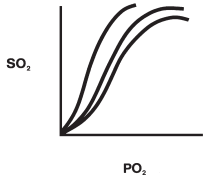
NORMAL OXYHEMOGLOBIN DISSOCIATION CURVE



The affinity of hemoglobin for oxygen is independent of the PO_2 – SO_2 relationship. Under normal conditions, the point at which the hemoglobin is 50% saturated with oxygen is called the P50 at a PO_2 of 27 mmHg. Alterations in the hemoglobin-oxygen affinity will produce shifts in the ODC.

FACTORS SHIFTING OXYHEMOGLOBIN DISSOCIATION CURVE

Leftward shift:
Increased affinity
Higher SO_2 for PO_2
↑ pH, Alkalosis
Hypothermia
↓ 2-3 DPG



Rightward shift:
Decreased affinity
Lower SO_2 for PO_2
↓ pH, Acidosis
Hyperthermia
↑ 2-3 DPG

The clinical significance of shifting the ODC is that SO_2 and PO_2 assessment parameters may not accurately reflect the patients' clinical status. A shift of the ODC to the left can lead to tissue hypoxia in spite of normal or high saturation values.

Pulmonary Gas Exchange Equations

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

Alveolar Gas Equation: PAO_2 is known as the ideal alveolar PO_2 and is calculated knowing the composition of inspired air.

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

Alveolar–arterial Oxygen Gradient (A–a Gradient or P(A–a)O_2)

P(A-a)O_2: Assesses the amount of oxygen diffusion across the alveolar capillary unit. Compares the alveolar gas equation to the arterial partial pressure of oxygen.

$[(PB - PH_2O) \times FiO_2] - PaCO_2 \times [FiO_2 + (1 - FiO_2) / 0.8] - (PaO_2)$

Normal: < 15 mmHg on room air

Normal : 60 – 70 mmHg on FiO_2 1.0

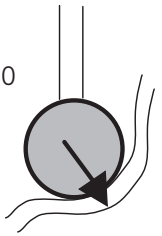
PB: Atmospheric pressure at sea level: 760

PH_2O: Pressure of water: 47 mmHg

FiO_2: Fraction of inspired oxygen

$PaCO_2$: Partial pressure of CO_2

0.8: Respiratory quotient (VCO_2 / VO_2)



A–a GRADIENT CALCULATION

(Barometric Pressure	–	Water Vapor Pressure)	x	Patient's FiO_2	–	$\frac{PaCO_2}{0.8}$	–	Patient's PaO_2		
(760	–	47)	x	0.21	–	$\frac{40}{0.8}$	–	90		
	713		x	0.21	–	50	–	90		
				99.73			–	90		= 9.73
				A–a Gradient			≅	10		

Assumes breathing at sea level, on room air, with a $PaCO_2$ of 40 mmHg and PaO_2 of 90 mmHg.

Intrapulmonary Shunt

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

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

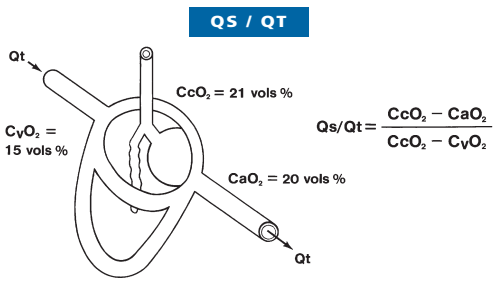
Some controversies exist in regards to measuring Qs/Qt. A true shunt is said to be accurately measured only when the patient is on an FiO_2 of 1.0. Venous admixture which produces a physiologic shunt can be determined when the patient is on an FiO_2 of < 1.0. Both determinations require pulmonary artery saturation values to complete the calculation.

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

CcO_2 = Capillary oxygen content
(1.38 x Hgb x 1) + (PAO_2 x 0.0031)

CaO_2 = Arterial oxygen content
(1.38 x Hgb x SaO_2) + (PaO_2 x 0.0031)

CvO_2 = Venous oxygen content
(1.38 x Hgb x SvO_2) + (PvO_2 x 0.0031)



Ventilation Perfusion Index (VQI) has been described as a dual oximetry estimate of intrapulmonary shunt (Qs/Qt).

Assumptions involved in the equation are:

1. Dissolved oxygen is discounted
2. 100% saturation of pulmonary end-capillary blood
3. Hgb changes are not abrupt

Limitations of VQI include:

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

Equation Derivations

$$Qs/Qt = \frac{100 \times [(1.38 \times Hgb) + (0.0031 \times PAO_2) - CaO_2]}{[(1.38 \times Hgb) + (0.0031 \times PAO_2) - CvO_2]}$$

$$VQI = \frac{100 \times [1.38 \times Hgb \times (1 - SaO_2 / 100) + (0.0031 \times PAO_2)]}{[1.38 \times Hgb \times (1 - SvO_2 / 100) + (0.0031 \times PAO_2)]}$$

Dual Oximetry, Simplifies the Shunt Equation

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

Basic Monitoring

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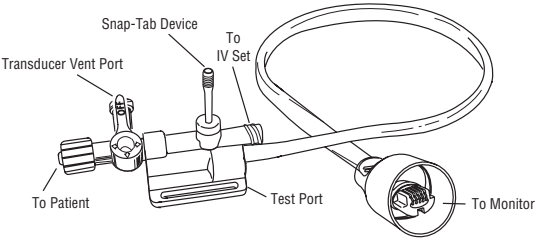
Physiologic Pressure Monitoring

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

TRUWAVE DISPOSABLE PRESSURE TRANSDUCER COMPONENTS



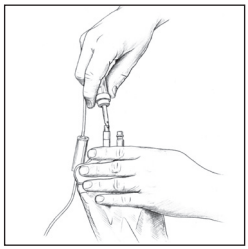
Components of a Physiologic Pressure Measurement System

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

Observation of best practices in set-up, calibration, and maintenance of a physiologic pressure transducer system is crucial in obtaining the most accurate pressure readings from which diagnosis and interventions are made.

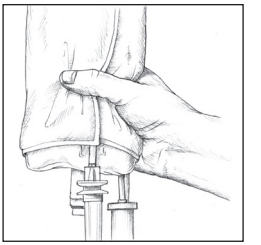
Best Practice in Setting Up a Physiologic Pressure Measurement System for Intravascular Monitoring

1. Wash hands
2. Open TruWave disposable pressure transducer packaging and inspect contents. Replace all caps with non-vented caps and ensure that all connections are tight
3. Remove the TruWave transducer from its packaging and insert into an Edwards Lifesciences mounting back-plate that is secured on an IV pole
4. To de-air and prime IV flush bag and TruWave transducer: Invert normal saline bag (anticoagulation per institution policy). Spike IV bag with fluid administration set, keeping drip chamber upright. While keeping IV bag inverted, gently squeeze air out of bag with one hand while pulling flush (Snap-tab) with the other hand until air is emptied from IV bag and drip chamber is filled to desired level (½ or full)

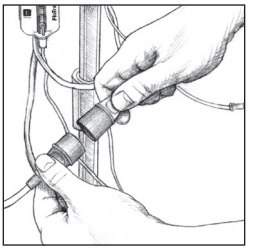


5. Insert flush bag into pressure infuser bag (DO NOT INFLATE) and hang on IV pole at least 2 feet (60cm) above the transducer

6. With gravity only (no pressure in Pressure Bag), flush TruWave transducer holding pressure tubing in upright position as the column of fluid raises through the tubing, pushing air out of the pressure tubing until the fluid reaches the end of the tubing (flushing under pressure creates turbulence and increased occurrence of bubbles)



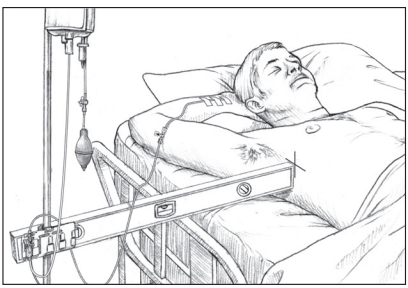
7. Pressurize the pressure bag until it reaches 300 mmHg
8. Fast-flush transducer tubing while tapping on tubing and stopcocks to remove any residual bubbles
9. Connect non-disposable pressure cable that is compatible with bedside monitor to disposable pressure transducer and bedside monitor



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

Best Practice in Leveling and Zeroing a Physiologic Pressure Transducer System

1. Level the transducer's closest stopcock (Vent port) to the physiologic pressure source. Intra-vascular monitoring should be level to the heart or the phlebostatic axis (fourth intercostal space at the chest's anterior-posterior midpoint). This removes the effects of hydrostatic pressure on the pressure transducer
2. Leveling should be performed with a carpenter's level or a laser leveler (PhysioTrac laser leveler). Leveling by visual estimation is not recommended as it is proven to be unreliable with significant inter-user variability



3. Zero referencing eliminates the effects of atmospheric and hydrostatic pressure
4. Open the reference stopcock to air by removing the non-vented cap, keeping sterility intact
5. After removing non-vented cap, turn stopcock off to the patient
6. Initiate "Zero" function on bedside monitor and confirm pressure waveform and numeric value display 0 mmHg
7. Once the "zero" is observed, turn the stopcock back to the vent port and replace the non-vented cap

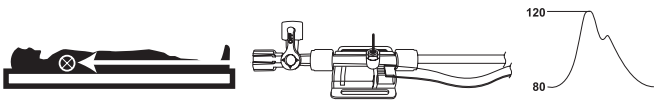
Best Practice in Maintaining Physiologic Pressure Transducer System

- **Keep transducers level:**
  Re-level transducer whenever the patient's height or position changes in relation with transducer
- **Re-zero transducer:**
  Periodic zeroing of physiologic pressure transducer every 8 – 12 hours
- **Check pressure infuser bag:**
  Maintain a pressure of 300 mmHg to assure constant flow of flush solution and system fidelity
- **Check flush bag volume:**
  Change < ¼ full to assure constant flow of flush solution and system fidelity
- **Check system integrity:**
  Assure system is free of bubbles that may develop over time, stopcocks are properly aligned, connections are tight, and catheter is free from kinking
- **Check frequency response:**
  Perform square wave test every 8 – 12 hours to assess for over or under damping of system

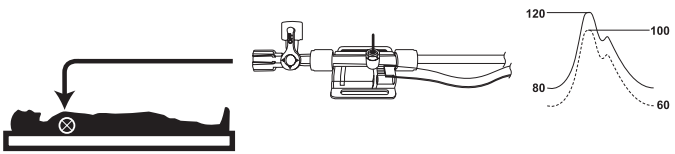
Impact of Improper Leveling on Pressure Readings

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

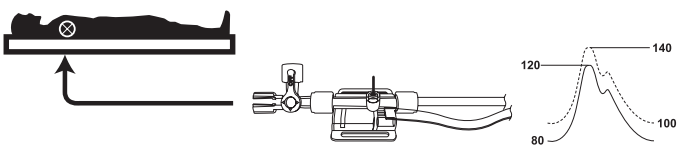
For every inch (2.5 cm) the heart is offset from the reference point of the transducer, a 2 mmHg of error will be introduced.



Heart aligned with transducer = 0 mmHg error



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



Heart 10" (25cm) HIGHER than transducer = Pressure 20 mmHg erroneously HIGH

Waveform Fidelity and Optimal Frequency Response

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

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

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

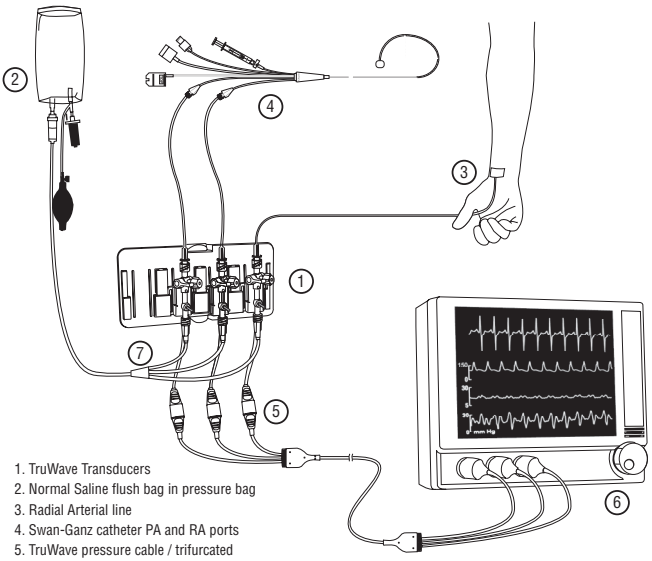
A square wave test can be used as a simple method of evaluating the frequency response at the bedside.

Note: See page 54 for further information and examples of square wave tests.

Pressure Monitoring Systems

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

PRESSURE SYSTEM



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

Determining Dynamic Response

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

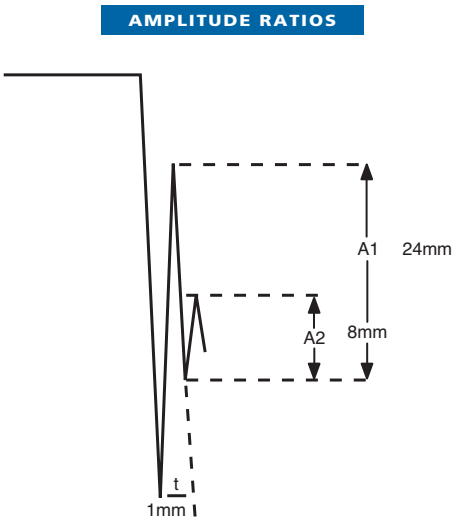
Perform a Square Wave Test

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

Calculate the Natural Response (fn)

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

$$fn = \frac{\text{paper speed (mm/sec)}}{\text{oscillation width/mm}}$$



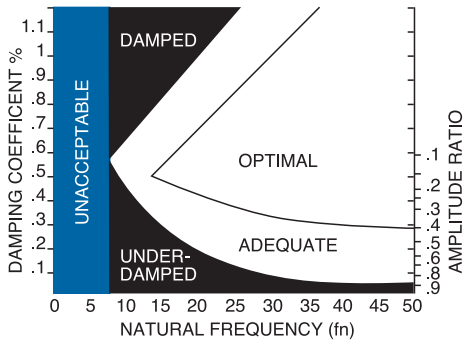
Determine the Amplitude Ratio

Estimate by measuring the amplitudes of two consecutive oscillations to determine an amplitude ratio, A2 / A1.

Plot to Determine Damping Coefficient

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

DYNAMIC RESPONSE GRAPH

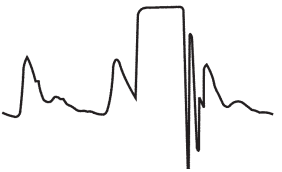


Simple Evaluation of Dynamic Response

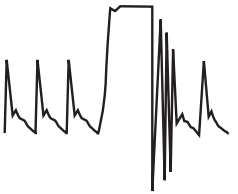
Determining the dynamic response characteristics of a pressure monitoring system by calculating the amplitude ratio and damping coefficient may not be feasible at the bedside when a rapid assessment of the waveform is required. A simple evaluation of dynamic response can be obtained by performing a square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

Square Wave Testing

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



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



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

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

Measuring Technique

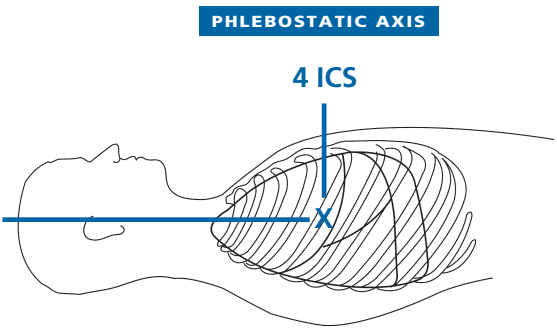
Hydrostatic Zero Reference

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

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

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

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



Mid-Point
A-P Chest Wall

Intra-arterial Monitoring

Components of the Arterial Pulse

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

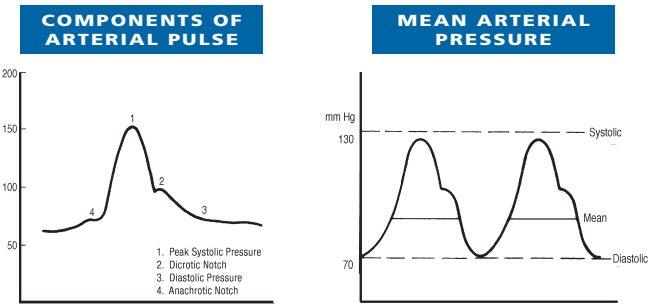
Dicrotic notch: reflects closure of the aortic valve, marking the end of systole and the onset of diastole

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

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

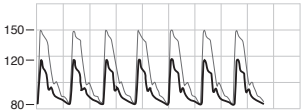
Pulse pressure: difference between systolic and diastolic pressure

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



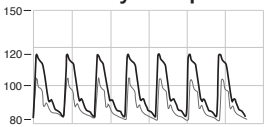
Bedside physiologic monitors use various algorithms to incorporate the area under the curve for determining the mean pressure.

Elevated systolic pressure



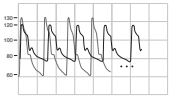
Systemic hypertension
Arteriosclerosis
Aortic insufficiency

Decreased systolic pressure



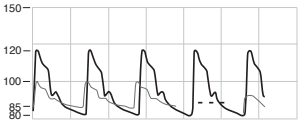
Aortic stenosis
Heart failure
Hypovolemia

Widened pulse pressure



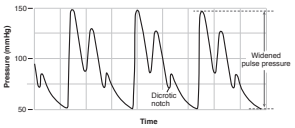
Systemic hypertension
Aortic insufficiency

Narrowed pulse pressure



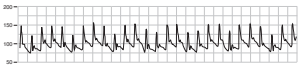
Cardiac tamponade
Congestive heart failure
Cardiogenic shock
Aortic stenosis

Pulsus bisferiens



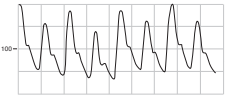
Aortic insufficiency
Obstructive hypertrophic cardiomyopathy

Pulsus paradoxus



Cardiac tamponade
Chronic obstructive airway disease
Pulmonary embolism

Pulsus alternans



Congestive heart failure
Cardiomyopathy

Central Venous Access

Types of Central Venous Access Devices

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

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

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

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

Applications of Central Venous Access Devices

- Rapid fluid administration – for example, in cases of, or at high risk of, high blood loss
    - Multiple trauma
    - Complex orthopedic surgery
    - Large vascular surgery
    - Extensive abdominal surgery
    - Tumor de-bulking
    - Sepsis
    - Burns
- Administration of IV fluids requiring dilution within the central circulation to avoid vascular damage (i.e., chemo-therapy, total parenteral nutrition)
- Administration of vasoactive and/or incompatible drugs
- Frequent blood sampling (in patients without an arterial line) and/or blood administration therapies
- Chronically ill patients in whom peripheral IV access is limited or unavailable
- Central venous pressure (CVP) monitoring for assessment of intravascular fluid status
- Measurement of oxygen saturation levels in blood returning to the heart ($ScvO_2$)
- Monitoring and access for either pre- or post-pulmonary artery catheter insertion (same insertion site)

Relative Contraindications may Include Patients with

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

Complications

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

Mitigating Complications

Mitigating catheter-related bloodstream infections:

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

Mitigating inadvertent carotid puncture/cannulation, multiple sticks

- Ultrasound guided central line placement

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

Central Venous Catheter Specifics

Polyurethane (Commonly Used for Catheter Body):

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

Lumens and Functionality:

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

8.5 Fr Double Lumen Catheter Cross Section

8.5 Fr Quad Lumen Catheter Cross Section

Flow Characteristics

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

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

Length

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

Solution for Excess Catheter, Box Clamp

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

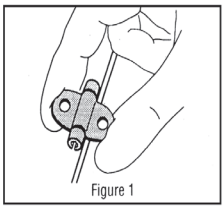


Figure 1

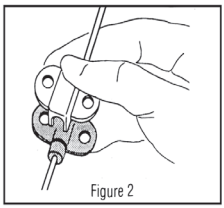


Figure 2

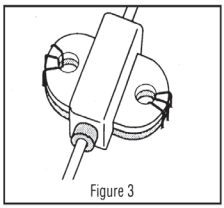


Figure 3

Lumen Designations and Infusion Rates

CVC PORT DESIGNATION

Distal (or largest gauge)	Medial	Proximal
Blood administration	TPN *or* medications	Medication administration
High volume fluids		Blood sampling
Colloid fluid administration		Drug therapy
Drug therapy		
CVP monitoring		

*These are suggestions only.

CVC PORT COLOR DESIGNATION

Port	Double	Triple	Quad
Proximal	White	White	White
Medial (1)	Blue	Blue	Blue
Medial (2)			Gray
Distal	Brown	Brown	Brown

CVC INFUSION RATES

7 Fr Double Lumen and Triple Lumen Polyurethane Multi-Med Catheters AVERAGE PERFORMANCE FLOW RATE			
Catheter	**16 cm Length (mL/hr)**	**20 cm Length (mL/hr)**	**Cross-Section Gauge Equivalence**
Triple Lumen			
Proximal	1670	1420	18
Medial	1500	1300	18
Distal	3510	3160	16
Double Lumen			
Proximal	3620	3200	16
Distal	3608	3292	16

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

Infection Mitigation

Coatings

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

"Oligon" Antimicrobial Catheter Material

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

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

Catheter and Accessory Features

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

Introducers as a Central Line

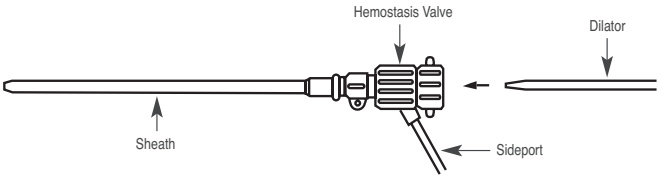
Sometimes an introducer is used for central venous access when rapid volume resuscitation is needed or is left in place following the removal of a pulmonary artery catheter. Components of the introducer system usually include:

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

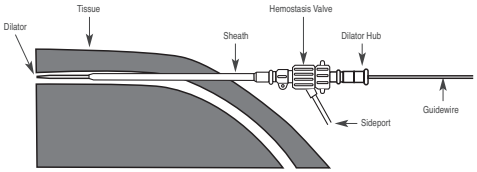
After insertion, the guidewire and dilator are removed, leaving the sheath in place. Fluids may be administered through the side port, while the hemostasis valve prevents bleedback and/or air embolization.

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

AUTOMATIC HEMOSTASIS VALVE



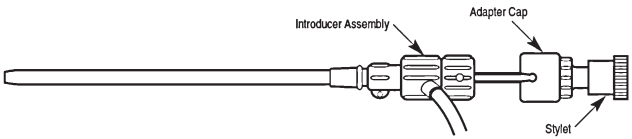
TUOHY-BORST VALVE INTRODUCER (INSERTED)



Infusion Catheter

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

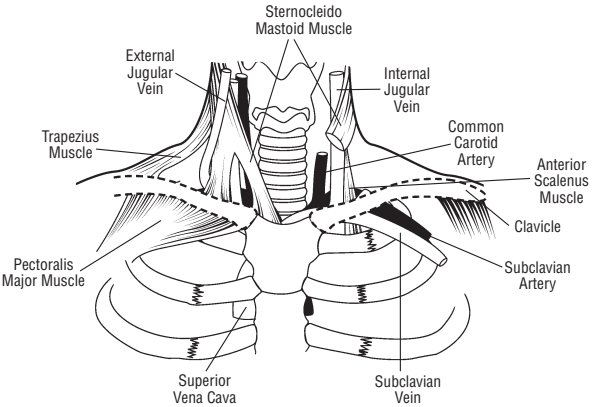
INFUSION CATHETER



Insertion Sites

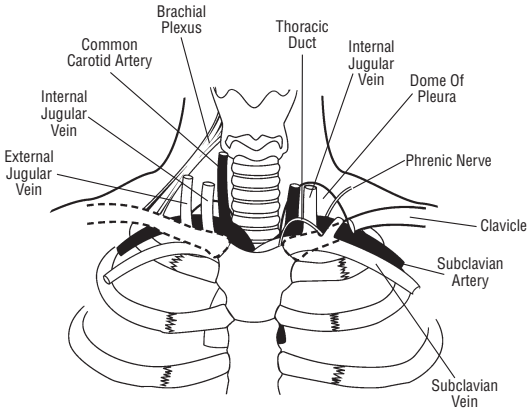
Typically, central venous catheters are inserted via the subclavian or internal jugular (IJ) veins. The subclavian vein begins at the lateral border of the first rib and arches through the space between the first rib and clavicle. It joins the internal jugular to become the innominate (or brachiocephalic)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

RELATIONSHIP OF CLAVICULAR LANDMARKS TO VASCULAR ANATOMY



Note the natural "windows" for supraclavicular venipuncture: 1) supraclavicular triangle formed by the clavicle, trapezius, and sternocleidomastoid muscles; 2) clavicular sternocleidomastoid triangle formed by the two bellies of the sternocleidomastoid muscle and the clavicle.

ANATOMIC ILLUSTRATION OF SIDE PREFERENCE RATIONALE FOR CLAVICULAR APPROACHES



Note the close proximity of arterial and venous structure. Venipunctures in the lateral region of the clavicle are more prone to arterial puncture, brachial plexus injury, and pnemothorax. Note the prominent thoracic duct and higher apex of the lung on the left and the perpendicular entry of the left IJ into the left subclavian vein.

Catheter Tip Placement

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

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

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

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

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

Monitoring Central Venous Pressure

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

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

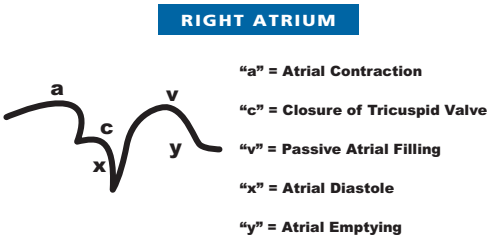
However, there are many factors that influence CVP values, for example, cardiac performance, blood volume, vascular tone, intrinsic venous tone, increased inta-abdominal or intra-thoracic pressures and vasopressor therapy. Therefore using CVP to assess either preload or volume status of the patient may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

CVP INTERPRETATION (CVP RANGE 2-6 MMHG)

Increased CVP	Decreased CVP
Increased venous return from conditions that cause hypervolemia	Decreased venous return and hypovolemia
Depressed cardiac function	Loss of vascular tone caused by vasodilation (sepsis) which contributes to venous pooling and reduced blood return to the heart
Cardiac tamponade	
Pulmonary hypertension	
PEEP	
Vasoconstriction	

Normal CVP Waveform

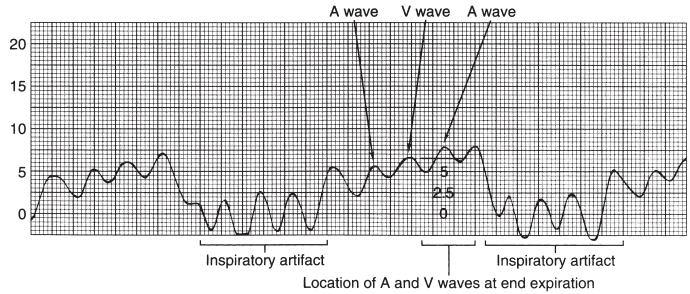
Waveforms seen on the monitor reflect the intracardiac events. The normal CVP waveform consists of three peaks (a, c and v waves) and two descents (x and y). The *a wave* represents atrial contraction and follows the P wave on the ECG trace. This is the atrial kick that loads the right ventricle just prior to contraction. As atrial pressure decreases, a *c wave*, resulting from closure of the tricuspid valve, may be seen. The *x descent* represents the continually decreasing atrial presure. The *v wave* represents the atrial events during ventricular contraction — passive atrial filling — and follows the T wave on the ECG. When the atrial pressure is sufficient, the tricuspid valve opens, and the *y descent* occurs. Then the cycle repeats.



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

CVP WAVEFORM

Reading CVP waveforms with spontaneous inspiratory artifact



Notes

Advanced Minimally Invasive Monitoring

ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972

The FloTrac System Algorithm

Arterial Pressure-Based Cardiac Output

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

Standard Deviation of Arterial Pressure

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

Traditional: CO = HR * SV

FloTrac system:

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

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

σAP = standard deviation of arterial pulse pressure in mmHg is proportional to pulse pressure.

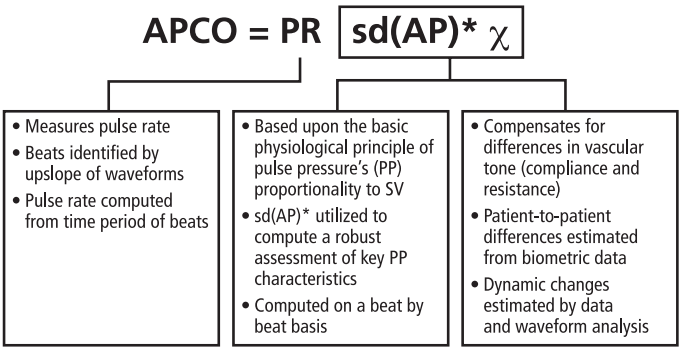
Khi (χ) = scaling multivariate parameter proportional to the effects of vascular tone on pulse pressure.

M = multivariate polynomial equation.

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

MAP = mean arterial pressure calculated by taking sum of sampled pressure point values over 20 seconds and dividing it by the number of pressure points.

μ = statistical moments determined by skewness (symmetry) and kurtosis (distinctness of a peak) calculated along several mathematical derivatives.



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

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

- **Pulse rate:** The patient's pulse rate is calculated by counting the number of pulsations in a 20-second period and extrapolated to a per minute value.
- **Mean arterial pressure (MAP):** An increase in average pressure often indicates an increase in resistance, and vice versa.
- **Standard deviation of arterial pressure (σ_{AP}):** Pulse pressure is proportional to σ_{AP} and to stroke volume. Increases and decreases in the standard deviation also provide information on pressure amplitude. When this pressure amplitude is correlated with kurtosis, it compensates for differential compliance and wave reflectance that vary from one arterial location to another. This then allows the monitoring of cardiac output from different arterial locations.
- **Large vessel compliance:** Work reported by Langewouters found a direct correlation among age, gender, and MAP with respect to aortic compliance. An equation was derived from these studies by which a patient's compliance could be estimated with the inputs of age and gender. According to Langewouters et al, the arterial compliance (C), as a function of pressure, could be estimated using the following equation:

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

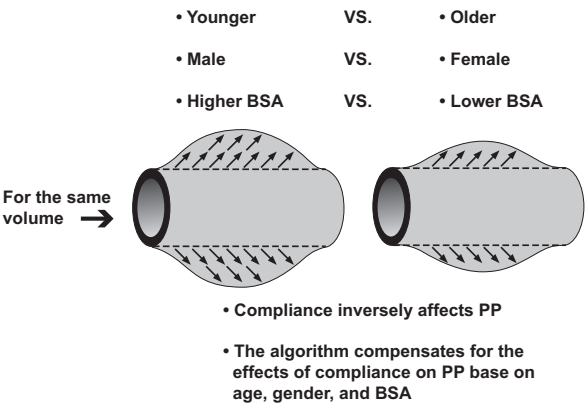
L = estimated aortic length

A_{max} = aortic root cross sectional area maximum

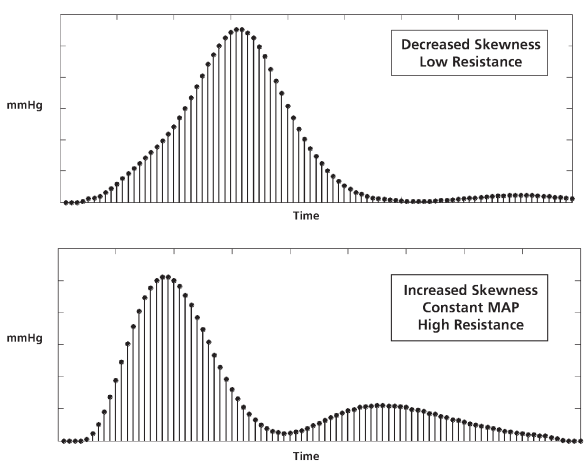
P = arterial pressure

P_0 = pressure at which compliance reaches its maximum

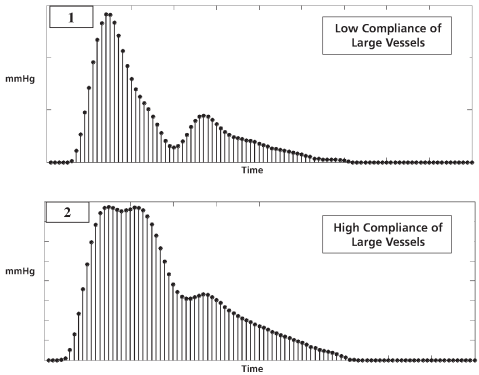
P_1 = the width of compliance curve at half of maximum compliance. Additional measures of weight and height (BSA) were also found to correlate with vascular tone and were added to enhance the calculation of aortic compliance



- **Skewness (a measure for lack of symmetry, μ_{3ap}):** Symmetry characteristics on arterial pressure can indicate a change in vascular tone and/or resistance. Two different functions may have the same mean and standard deviation but will rarely have the same skewness. For example, an arterial pressure waveform in which the data points increase quickly in systole and fall slowly can result as an increase in vasoconstriction and would have increased skewness.



- **Kurtosis (a measure of how peaked or flat the pressure data points are distributed from normal distribution, μ_{4ap}):** Pressure data with high kurtosis has the pressure rise and fall very quickly relative to the normal pulse pressure and can be directly associated with large vessel compliance.
  1) A high kurtosis value will indicate a distinct peak near the mean, with a drop thereafter, followed by a heavy "tail."
  2) A low kurtosis value will tend to indicate that the function is relatively flat in the region of its peak and suggests decreased central tone, as is often seen, for example, in the neonatal vasculature.



Khi (χ) mmHg to mL/beat

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

Stroke Volume (mL/beat) = σ_{AP} (mmHg)* χ (mL/mmHg)

No Manual Calibration Needed

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

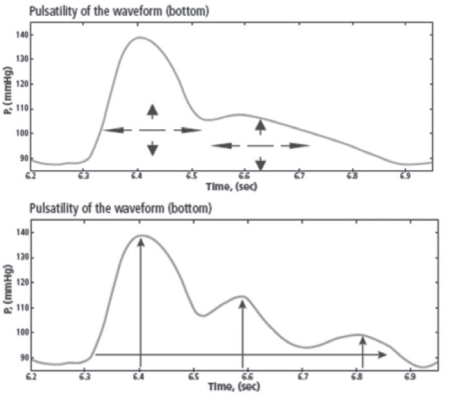
Technical Considerations

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

The FloTrac System 4.0

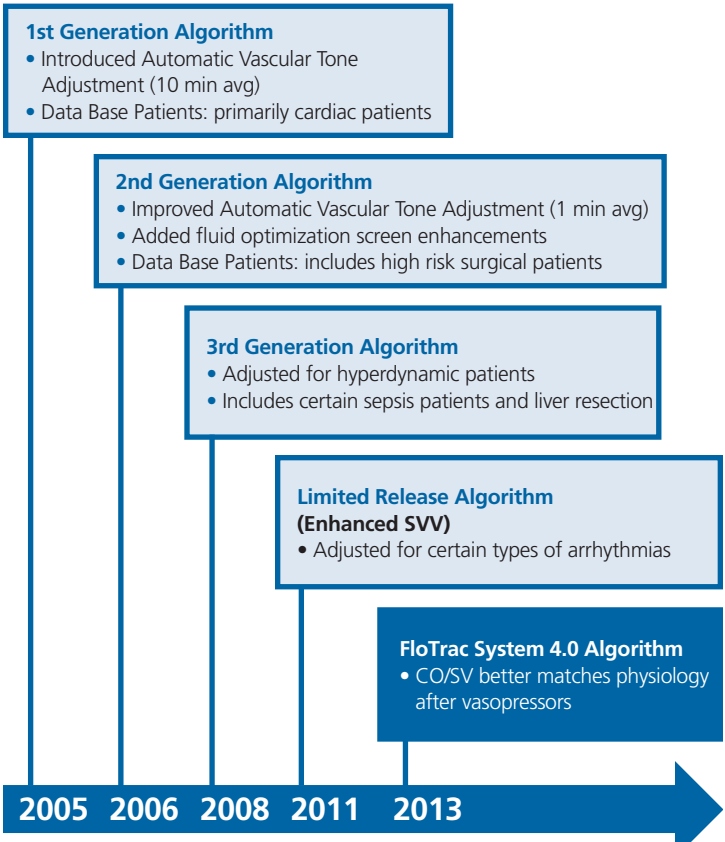
The FloTrac system algorithm has evolved based on a broad and expanding patient database that allows ongoing system performance improvements. In this latest evolution (v.4.0), Edwards continues to expand the database to include a more diverse surgical patient population in order to continuously inform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

These updates are in addition to changes made in FloTrac systems 3rd generation software which continuously assess the arterial waveform for characteristic changes associated with hyperdynamic and vasodilated conditions. As part of this effort, additional physiologically-based variables (see image below) were added to the algorithm's vascular tone Khi factor in order to adjust automatically for hyperdynamic and vasodilated patients. Once identified it accesses a specially designed algorithm to account for such conditions.



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

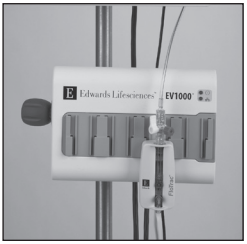
FloTrac System Algorithm Evolution



FloTrac Sensor Setup

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

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

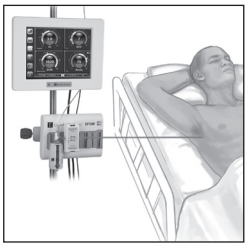


3. **To de-air and prime I.V. bag and FloTrac sensor:** Invert normal saline I.V. bag (anticoagulation per institution policy). Spike I.V. bag with fluid administration set, keeping drip chamber upright. While keeping I.V. bag inverted, gently squeeze air out of bag with one hand while pulling flush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

4. Insert I.V. bag into the **Pressure Bag** and hang on I.V. pole **(do not inflate)**.
5. With gravity only **(no pressure in Pressure Bag)**, flush FloTrac sensor holding pressure tubing in upright position as the column of fluid raises through the tubing, pushing air out of the pressure tubing until the fluid reaches the end of the tubing.

6. Pressurize the **Pressure Bag** until it reaches 300 mmHg.
7. Fast-flush the FloTrac sensor and tap on tubing and stopcocks to remove any residual bubbles.
8. Connect the **green** FloTrac connecting cable to the **green** capped connector on the FloTrac sensor. Then connect the opposite end of the cable to the FloTrac connection on the back of the Edwards monitor.

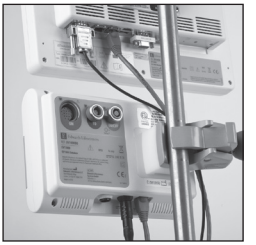
9. Connect the bedside monitor's arterial pressure cable to the **red** cable connector on the FloTrac sensor.
10. Connect tubing to arterial catheter, then aspirate and flush system to assure no residual bubbles remain.
11. Level the FloTrac sensor to the phlebostatic axis. **Note: It is important to keep the FloTrac sensor level to the phlebostatic axis at all times to ensure accuracy of cardiac output.**
    - Open the stopcock to atmospheric air.
    - Select **Zero Arterial Pressure**, then select and press **Zero.**
    - **Zero** the arterial channel on the bedside monitor.



12. **Cardiac output will display within 40 seconds and will update every 20 seconds thereafter.**
13. Inspect arterial pressure trace on bedside monitoring screen or the waveform confirmation screen on the Edwards monitor.

FloTrac Sensor EV1000 Clinical Platform Setup

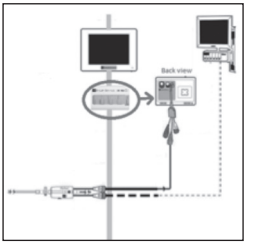
1. Connect the power adapter and ethernet cable for both EV1000 panel and databox. Press the ⏻ button on the panel.



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

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

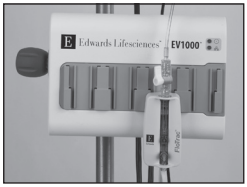
4. Connect the FloTrac trifurcated databox cable to the back of the EV1000 databox. Then connect the green FloTrac connecting cable to the green capped connector on the FloTrac sensor.



5. Connect the bedside monitor's arterial pressure cable to the red cable connector on the FloTrac sensor.

6. Touch **Clinical Actions** and then touch **Zero & Waveform.**

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



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

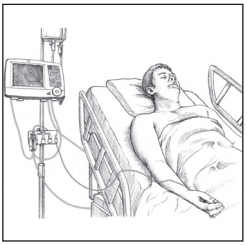
9. Monitor patient in real-time with one of the available screens.

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

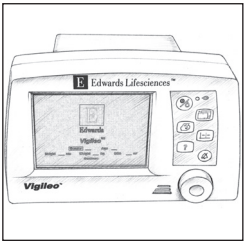
11. Visual targets and alarms can be set by touching inside parameters globe.

Vigileo Monitor Setup and Zeroing

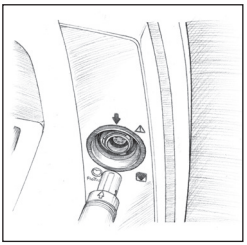
1. Press the ⏻ button on the front panel to turn the Vigileo monitor **ON.** The screen will display an opening message, indicating that a **Power-On** Self-Test (POST) is being performed.



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



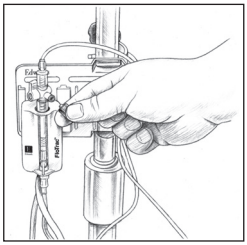
3. Use the navigation knob to select and enter values. Press **Continue** to continue selection and open the **Home** screen.
4. Connect the FloTrac connecting cable to the FloTrac cable connector at the back of the Vigileo monitor. Align the arrows at the top of the cable connector on the monitor to the arrow on the FloTrac connecting cable.



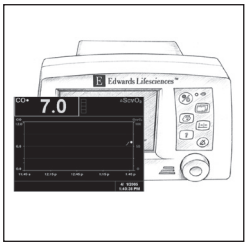
5. Connect the other end of the FloTrac cable to the green capped FloTrac sensor.
6. Rotate navigation knob until the **CO** frame is outlined in yellow and then **press** the knob to open CO menu.

7. From the **CO Menu**, rotate the navigation knob until **Zero Arterial Pressure** is highlighted and then press the knob. The Zero Arterial Pressure screen will appear.

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



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



Stroke Volume Variation

Trending Dynamic Parameters

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

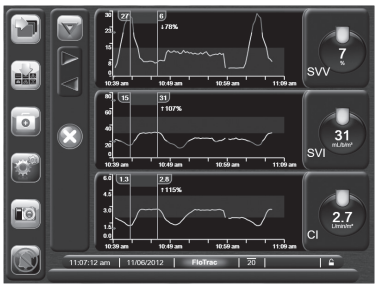
HEMODYNAMIC PARAMETERS FOR ASSESSING VOLUME STATUS AND FLUID RESPONSIVENESS

Static Parameters	Dynamic Parameters
Arterial pulse pressure (NIBP)	Systolic pressure variation (SPV)
Mean arterial pressure (MAP)	Arterial pulse pressure variation (PPV)
Central venous pressure (CVP)	Stroke volume variation (SVV)
Pulmonary artery occlusion pressure (PAOP)	
Heart rate	
Urine output	

Advantages of Trending SVV with Cardiac Output

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

FLOTRAC SYSTEM – ADVANCED SVV TRENDING SCREENS



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

SVV is increasingly used to determine fluid responsiveness and to monitor the effects of volume therapy. Successful optimization is linked to improved patient outcomes including shorter hospital stays and lower morbidity rates. As a result, tools such as the FloTrac system are being adopted to provide insight into fluid optimization, blood flow and oxygen delivery.

The FloTrac system provides dynamic insight using an existing arterial catheter. The system includes advanced SVV trending screens that provide vital information enabling early action while complementing the clinical workflow.

FLOTRAC SYSTEM – ADVANCED SVV TRENDING SCREENS

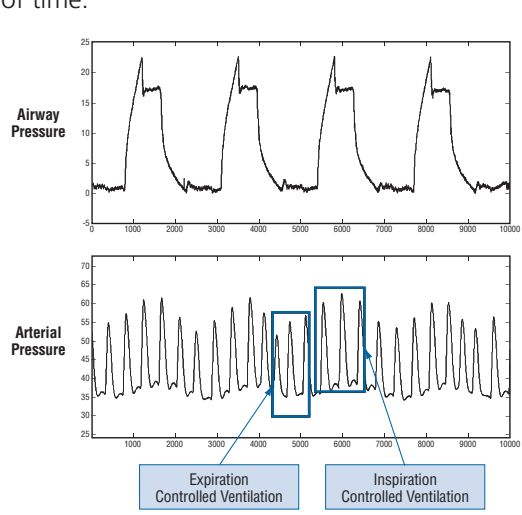
Using Fluid to Improve Hemodynamics

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

Calculating Stroke Volume Variation

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

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



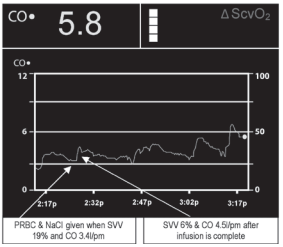
SVV and Assessing Fluid Response

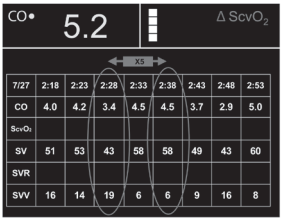
SVV and its comparable measurement, pulse pressure variation (PPV), are not indicators of actual preload but of relative preload responsiveness. SVV has been shown to have a very high sensitivity and specificity when compared to traditional indicators of volume status (HR, MAP, CVP, PAD, PAOP), and their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

Study	Patients	Volume	Tidal Volume mL/Kg	Parameters Tested (Artery)	R^2	Def. of Responder	Sensitivity	Specificity
Michard	Sepsis	500 mL	8 to 12	Δ PP (R or F)	0.85	Δ CO ≥ 15%	94	96
Berkenstadt, et al	Neuro Surgery	100 mL	10	Δ SVV	0.53	Δ SV ≥ 5%	79	93
Reuter, et al	Cardiac	10 x BMI	10	Δ SVV	0.64	Δ SV ≥ 5%	79	85

Application of SVV

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





Stroke Volume Variation Limitations

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

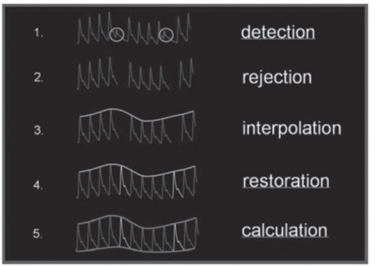
- Mechanical ventilation: Currently, literature supports the use of SVV on patients who are 100% mechanically (control mode) ventilated with tidal volumes of more than 8 mL/kg and fixed respiratory rates.
- Spontaneous ventilation: Currently, literature does not support the use of SVV with patients who are spontaneously breathing.
- Arrhythmias: Historically arrhythmias have dramatically affected SVV and its ability to be used to guide fluid resuscitation. SVVxtra limits this limitation with the exception of severe arrhythmias such as atrial fibrillation.
- Other considerations while using SVV to guide fluid resuscitation:
  - Heart rate (HR) <150 beats per minute
  - Heart rate to respiratory rate ratio below 3:1
  - Respiratory rate (RR) of <35
  - Chest must be closed
  - No right ventricular failure
  - Good arterial waveform required
  - Raised intra-abdominal pressure may exaggerate the cardio-pulmonary interaction
  - Raised intra-thoracic pressure may exaggerate the cardio-pulmonary interaction

SVVxtra

Limiting Limitations with the FloTrac Algorithm

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

The SVVxtra algorithm is based on five consecutive steps:



If the frequency of arrhythmias has exceeded the algorithms ability to filter these arrhythmias then a "Yellow Heart" icon will appear.

Interventional Effects on SVV

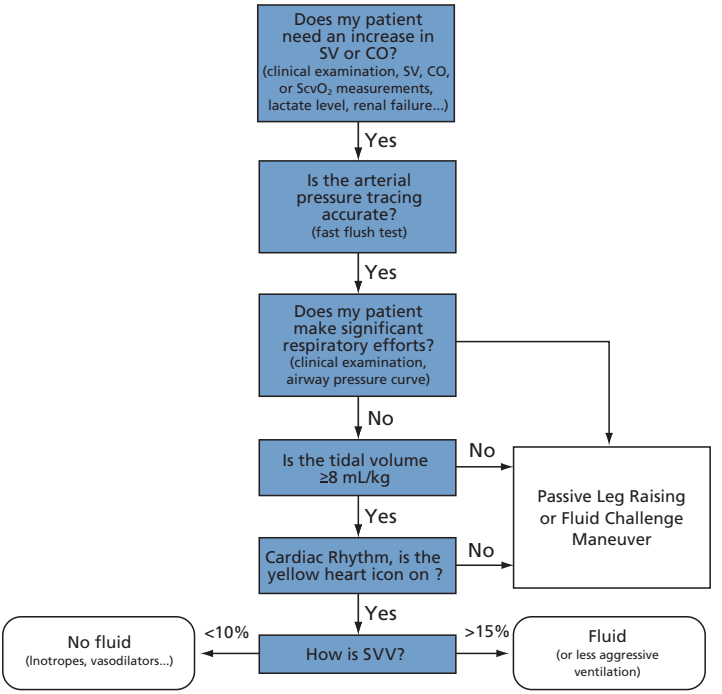
- PEEP
  Increasing levels of positive end expiratory pressure (PEEP) may cause an increase in SVV, the effects of which may be corrected by additional volume resuscitation if warranted.
- Vascular Tone
  The effects of vasodilatation therapy may increase SVV and should be considered before treatment with additional volume.

Summary

When used within its limitations SVV is a sensitive tool that can be used to guide the appropriate management of the patient's preload to achieve optimal DO_2 to assist with fluid optimization. SVV is an available parameter with the FloTrac sensor and Vigileo monitor.

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

FloTrac/Vigileo System SVV Algorithm



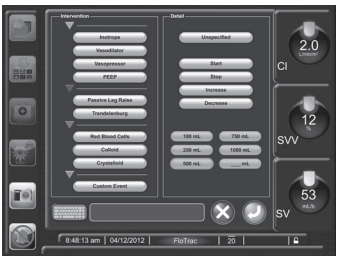
Modified from Michard. Anesthesiology 2005;103:419-28.

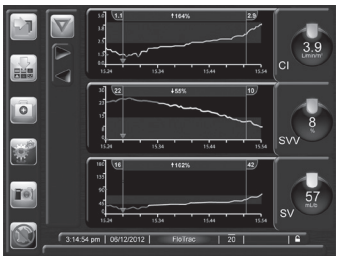
Fluid Challenges and FloTrac/Vigileo System

FloTrac/Vigileo System Passive Leg Raising (PLR) Maneuver

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

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





Concerns or Limitations

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

FloTrac/Vigileo System Fluid Challenge Maneuver

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

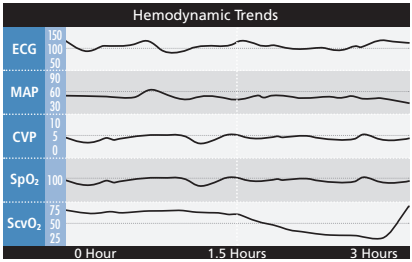
1. Note FloTrac system SV – T1 time on % change calculator
2. Infuse bolus of 250-500 mL
3. Note FloTrac system SV – T2 time on % change calculator
4. If SV % increase > 10-15% = preload responsive
5. Consider additional fluids
6. Repeat FloTrac/Vigileo system fluid challenge maneuver
7. If SV % < 10-15%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

Venous Oximetry Physiology and Clinical Applications

Physiology and Venous Oximetry

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

$ScvO_2$ = EARLY WARNING AND PREVENTION



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

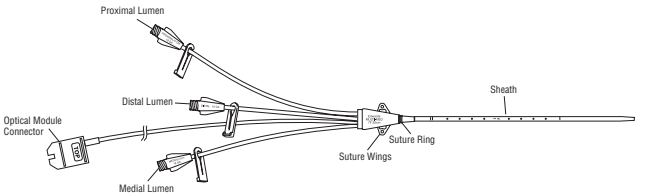
Continuous fiberoptic venous oximetry is a valuable tool for monitoring the balance between oxygen delivery and consumption at the bedside. Continuous venous oximetry is a sensitive real-time indicator of this balance, which can be applied as a global or regional indicator – with mixed venous oxygen saturation (SvO_2) and central venous oxygen saturation ($ScvO_2$) being the most commonly monitored. SvO_2 is a true reflection of the global balance between oxygen delivery and consumption since it is measured in the pulmonary artery, where venous blood returning to the right heart from the superior

vena cava (SVC), inferior vena cava (IVC) and the coronary sinus (CS) have mixed. SvO_2 has been extensively studied and used clinically to monitor the global balance between DO_2 and VO_2. SvO_2 monitoring has been available through either co-oximetry laboratory analysis or through continuous fiberoptic monitoring with advanced technology pulmonary artery catheters since the 1970s and mid-1980s, respectively.

Continuous fiberoptic $ScvO_2$ monitoring became available in 2003 on an 8.5 Fr central venous catheter platform (Edwards PreSep catheter). With the tip of the PreSep central venous catheter placed in the SVC, $ScvO_2$ can be measured and displayed on either a Vigileo or Edwards Vigilance II monitor. This capability is also available via 4.5 Fr and 5.5 Fr central venous oximetry catheters (Edwards PediaSat catheter) for pediatric use.

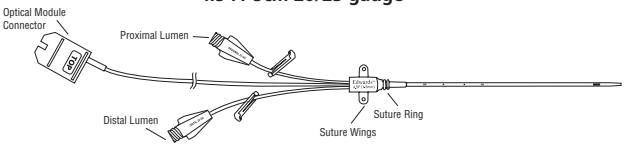
PRESEP OXIMETRY CATHETER

8.5 Fr 20cm 18/18/16 gauge with Oligon* antimicrobial material



PEDIASAT OXIMETRY CATHETER

4.5 Fr 5cm 20/23 gauge



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

Difference Between SvO_2 and $ScvO_2$

Since SvO_2 and $ScvO_2$ are affected by the same four factors (cardiac output, hemoglobin, oxygenation, and oxygen consumption), and trend together clinically, they are considered clinically interchangeable.The exception is when calculating global physiologic profiles that use SvO_2, such as VO_2.

SvO_2 is a global indicator of the balance between DO_2 and VO_2 as it is a reflection of all venous blood; IVC, SVC, and CS. $ScvO_2$ is a regional reflection (head and upper body) of that balance. Under normal conditions $ScvO_2$ is slightly lower than SvO_2 due in part to the mixing and amount of venous blood returning. In hemodynamically unstable patients, this relationship changes with $ScvO_2$ being higher than SvO_2 by approximately 7%. This difference can widen in shock states, up to 18%, but the values trend together more than 90% of the time.

Global Venous Oximetry

SvO_2 – mixed venous oximetry

Regional Venous Oximetry

$ScvO_2$ – head and upper extremities

$SpvO_2$ – peripheral venous oximetry

Organ Specific Venous Oximetry

$SjvO_2$ – cranial jugular bulb oximetry

$ShvO_2$ – hepatic venous oximetry

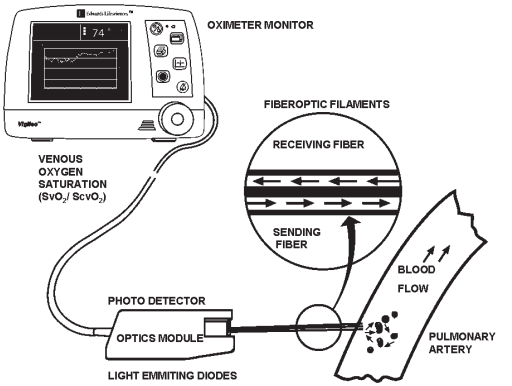
$ScsO_2$ – coronary sinus oximetry

Continuous $ScvO_2$ Monitoring Technology

All venous oximetry is measured through reflection spectrophotometry. Light is emitted from an LED through one of the two fiberoptic channels into the venous blood; some of this light is reflected back and received by another fiberoptic

channel, which is read by a photodetector. The amount of light that is absorbed by the venous blood (or reflected back) is determined by the amount of oxygen that is saturated or bound to hemoglobin. This information is processed by the oximetry monitor, and updated and displayed every two seconds as a percent value on the monitor.

FIBEROPTIC VENOUS OXIMETRY SYSTEM



Accuracy of Edwards Fiberoptic Continuous $ScvO_2$ Compared to Co-oximetry

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

Interference with $ScvO_2$ Readings

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

Interpreting Venous Oximetry (SvO_2 and $ScvO_2$) Values

Normal range values for SvO_2 are 60-80% and 70% for $ScvO_2$. $ScvO_2$ usually runs 7% higher than SvO_2 in critically ill patients. Low oximetry readings usually indicate either low oxygen delivery (DO_2) or an increase in consumption (VO_2). Significantly elevated levels (> 80%) may indicate:

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

When Change is Significant

$ScvO_2$ and SvO_2 values are not static and fluctuate approximately ± 5%. These values may show significant changes with activities or interventions such as suctioning; however, the values should recover within seconds. Slow recovery is an ominous sign of the cardiopulmonary system's struggle to respond to a sudden increase in oxygen demand. When monitoring $ScvO_2$, clinicians should look for changes of ± 5 -10% that are sustained for more than 5 minutes and then investigate each of the four factors that influence $ScvO_2$:

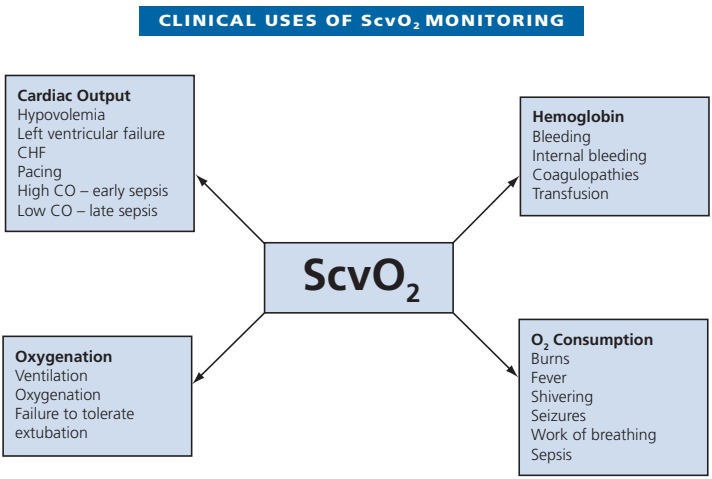
- Cardiac output
- Hemoglobin
- Arterial oxygen saturation (SaO_2) and
- Oxygen consumption

The first three (above) are indicators of DO_2, while the fourth is an indicator of VO_2.

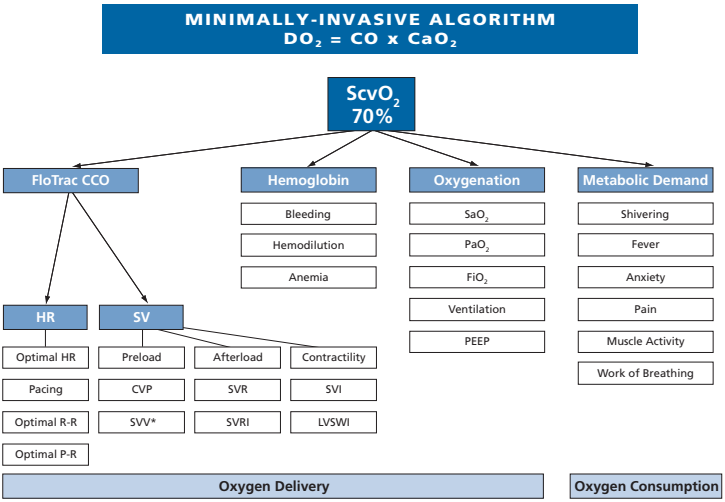
Clinical Applications of $ScvO_2$

$ScvO_2$ and SvO_2 are affected by the same four factors and trend together more than 90% of the time. Thus most of the research and clinical applications documented for SvO_2 should apply to $ScvO_2$.

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



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



Minimally-invasive algorithm breaking down components of oxygen delivery and consumption followed by sub-components investigating root cause of imbalance

Summary

Continuous venous oximetry ($ScvO_2$) monitoring is an early, sensitive, and real-time indicator of the balance between DO_2 and VO_2 that can alert clinicians to an imbalance when traditional vital signs may not. $ScvO_2$ monitoring with the PreSep or PediaSat catheter is a practical tool which is no more invasive than a traditional central venous catheter. Venous oximetry is best used in conjunction with cardiac output monitoring. Moreover, keeping $ScvO_2$ values above 70% has been proven to lead to better patient outcomes.

** SVV is an indicator of preload responsiveness.*

VolumeView System

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

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

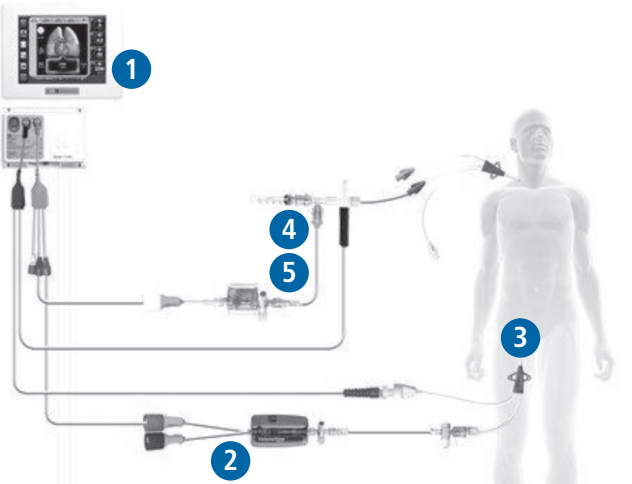
Transpulmonary thermodilution with the VolumeView system allows for the measurement and derived calculations of the elements that affect oxygen delivery through:

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

In addition:

- Extra-Vascular Lung Water
- Pulmonary Vascular Permeability Index

VolumeView System Setup

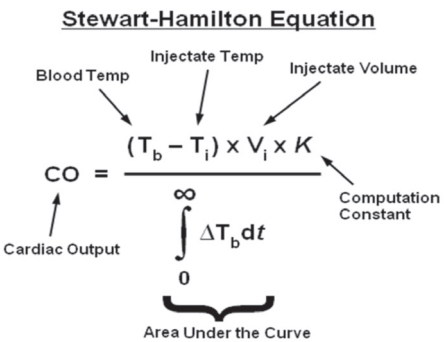


System Setup

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

Intermittent Cardiac Output Calculation with the VolumeView System

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



After injection, the computer analyzes the area under the transpulmonary thermodilution curve to calculate cardiac output. The area under the curve is inversely proportional to the cardiac output. A series of boluses are performed and edited to obtain an average value. Once edited the measured and derived calculations are displayed and time stamped for retrospective review.

- Blood Temperature is monitored and collected through an embedded thermistor on the VolumeView femoral arterial catheter.
- The injectate temperature is collected and monitored through an in-line thermistor on the VolumeView thermistor manifold.
- The volume of the injectate is entered into the computer by the clinician.
- The area under the curve is calculated and analyzed by the computer by measuring the change in temperature over time in the femoral artery.

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

Continuous Cardiac Output with VolumeView

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

Calculating Global End Diastolic Volume

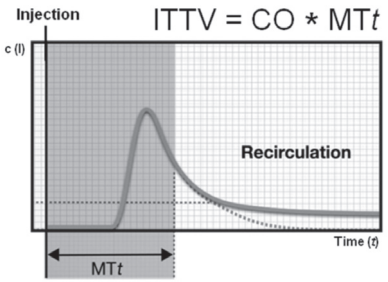
The transpulmonary thermodilution measurement used to calculate cardiac output can also be used to calculate other physiologic parameters such as Global End Diastolic Volume, Global Ejection Fraction, and Extra Vascular Lung Water. These parameters are useful in evaluating and guiding volume resuscitation,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

Global End Diastolic Volume is closely related to the volume within all four chambers at the end of diastole. It can be used to assess preload and manage a patient's volume resuscitation.

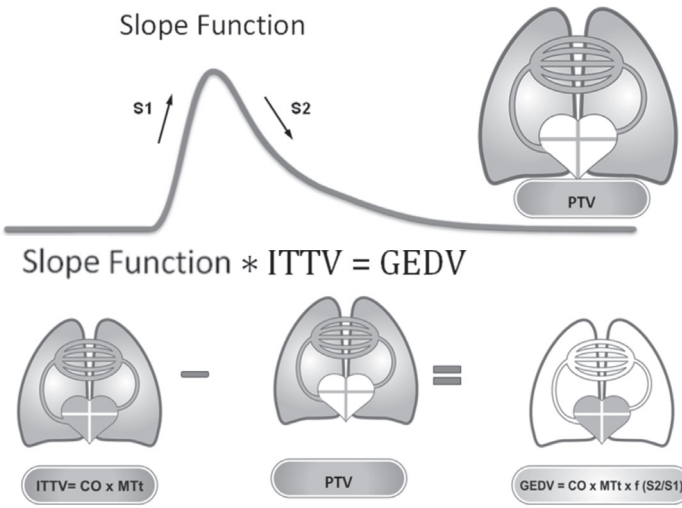
GEDI 680 – 800 ml/m²

In order to calculate GEDV, Intra Thoracic Thermal Volume (ITTV) is calculated by identifying the beginning of the injection cycle from a pressure spike measured in the central venous pressure from the VolumeView CVC manifold. The VolumeView system's TPTD algorithm then identifies the peak indicator concentration followed by its immediate downslope which is an indication of the mean transit time. Once cardiac output is known and the mean transit time is known, Intra Thoracic Thermal Volume can be calculated by multiplying cardiac output times the mean transit time. The downslope time is representative of the flow time through the lungs.

Intra Thoracic Thermal Volume is the first calculation of the cardiopulmonary volumes calculated from the TPTD procedure. It represents the total dilution volume within the thorax, which consists of the heart, lungs, and vasculature, that is calculated by the VolumeView TPTD algorithm.



The GEDV is a reflection of the volume within all four chambers at the end of diastole. The rate of change of the upslope and downslope of the thermodilution waveform is used to calculate the slope function which appropriately scales down ITTV to account for Pulmonary Thermal Volume in order to calculate GEDV. GEDV is computed by calculating ITTV and multiplying it against a scale that accounts for PTV.



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

Global Ejection Fraction

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

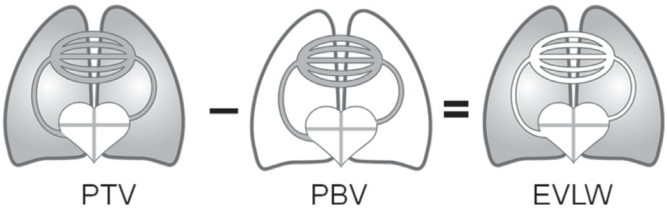
Cardiac Output / Pulse Rate = **Stroke Volume**

Stroke Volume * 4 / GEDV = **Global Ejection Fraction**

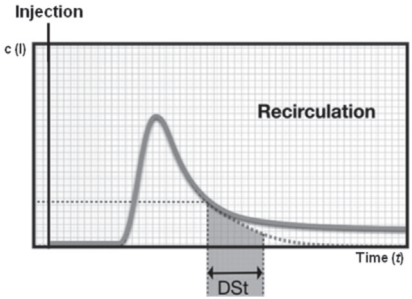
Global Ejection Fraction normal range is between **25-35%**

Extra Vascular Lung Water

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

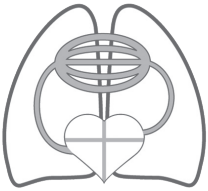


Extra Vascular Lung Water is calculated by subtracting the Pulmonary Blood Volume from the Pulmonary Thermal Volume, leaving the thermal volume within the lungs. EVLW can be used to assess the level of pulmonary edema which may be the result of heart failure, volume overload, or lung injury and can interfere with the ability of the lungs to oxygenate the blood. This is indexed against the patient's predicted body weight to obtain Extra Vascular Lung Water Index or EVLWI. EVLW can be used to assess the level of pulmonary edema.

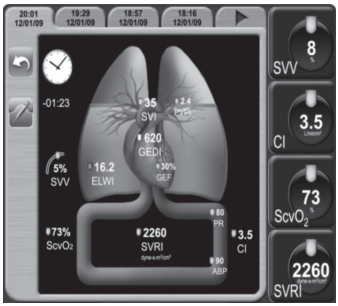


PTV

PTV = CO * DSt



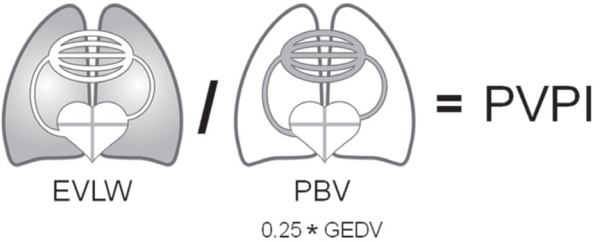
PBV = 0.25 * GEDV



The "normal" value for EVLWI is reported to be 3–7 mL/kg. Values above 10 mL/kg indicate pulmonary edema and values as high as 15-20 ml/kg indicate severe pulmonary edema. EVLW is a useful indicator of pulmonary edema and challenges with oxygenation.

Pulmonary Vascular Permeability Index

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



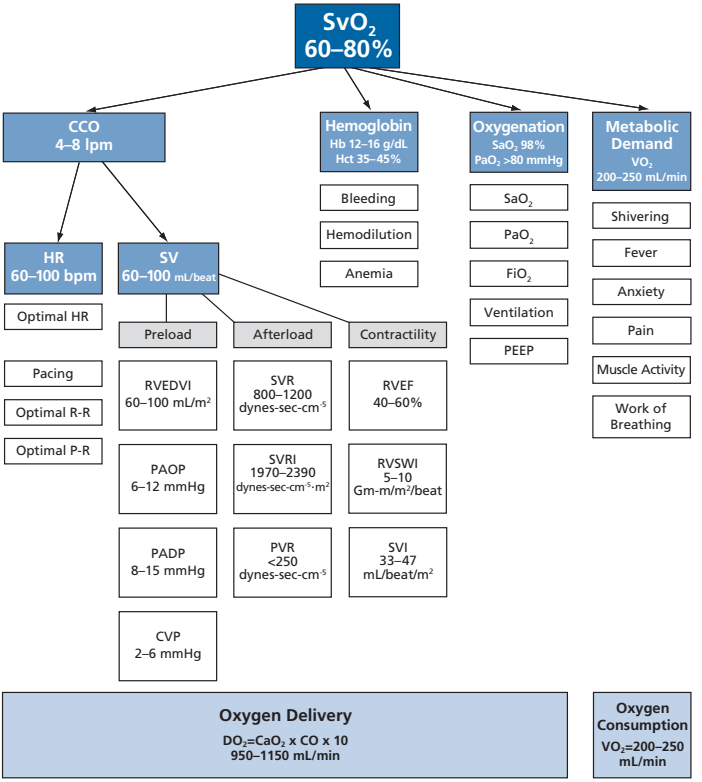
PVPI helps the clinician to differentiate which mechanisms are responsible for increased EVLW: PVPI is increased (> 3) in patients with increased pulmonary permeability due to lung injury and normal in patients with hydrostatic and cardiogenic pulmonary edema.

Quick Reference

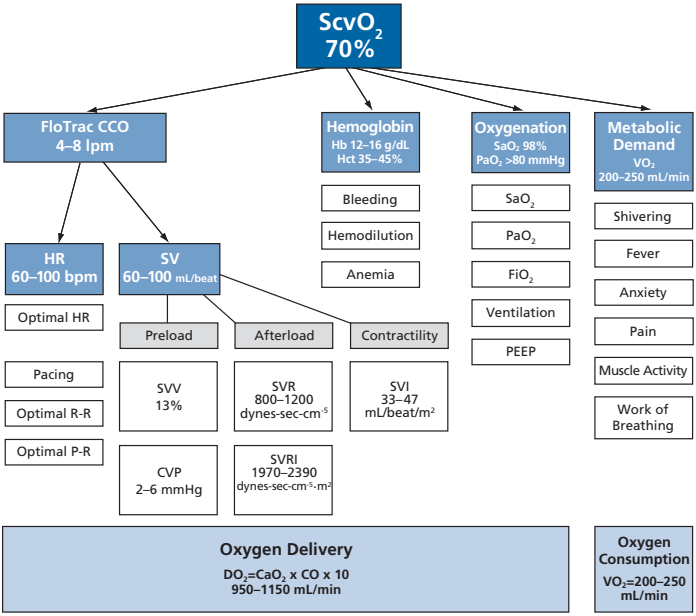
ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972

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

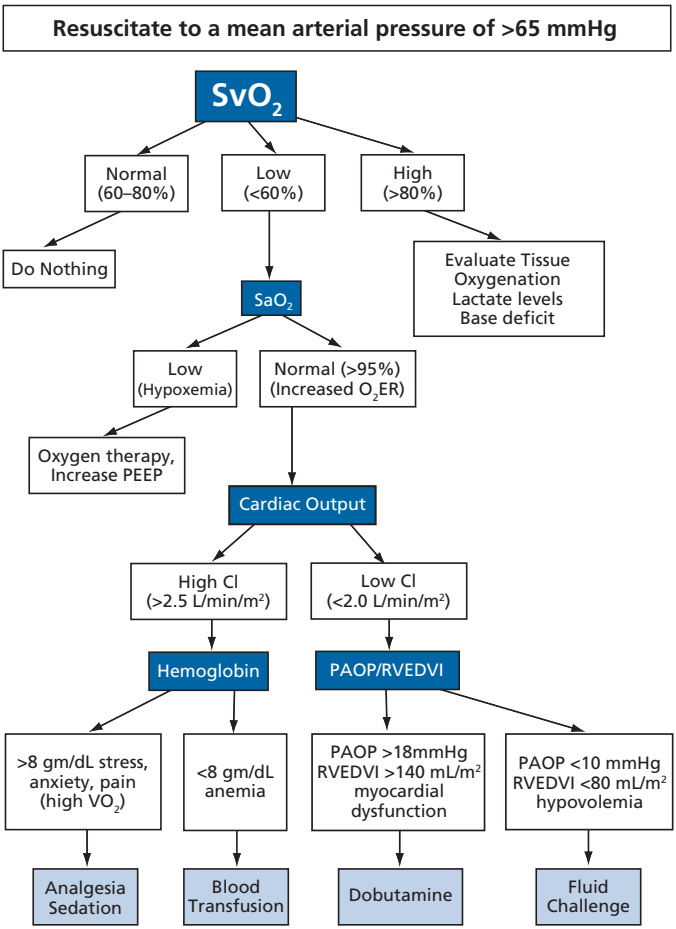
Advanced Technology Swan-Ganz Catheter Algorithm



Advanced Minimally-Invasive Algorithm

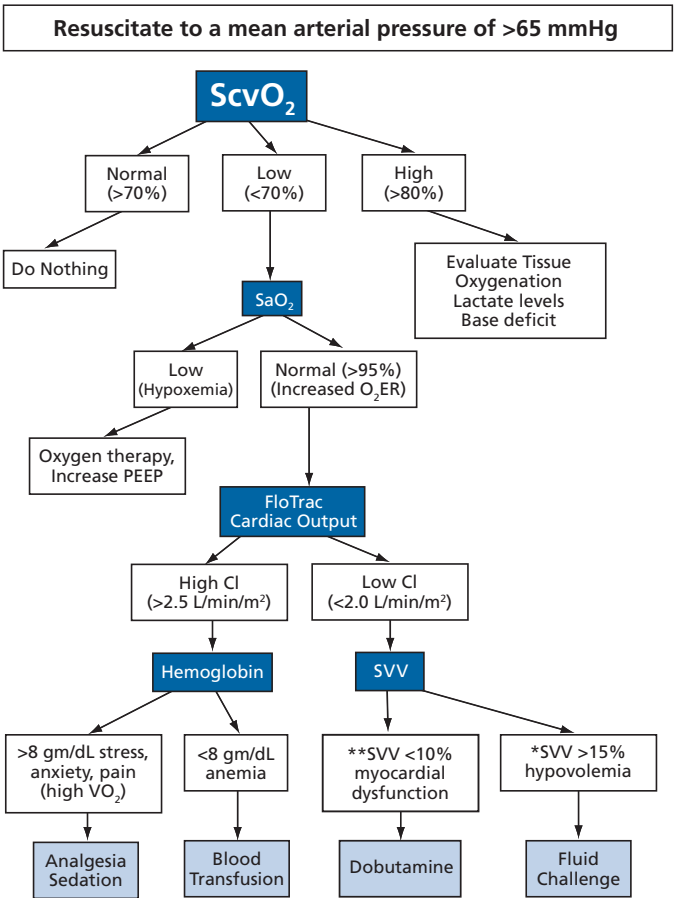


Advanced Swan-Ganz Catheter Goal-Directed Protocol



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

Advanced Minimally-Invasive Goal-Directed Protocol

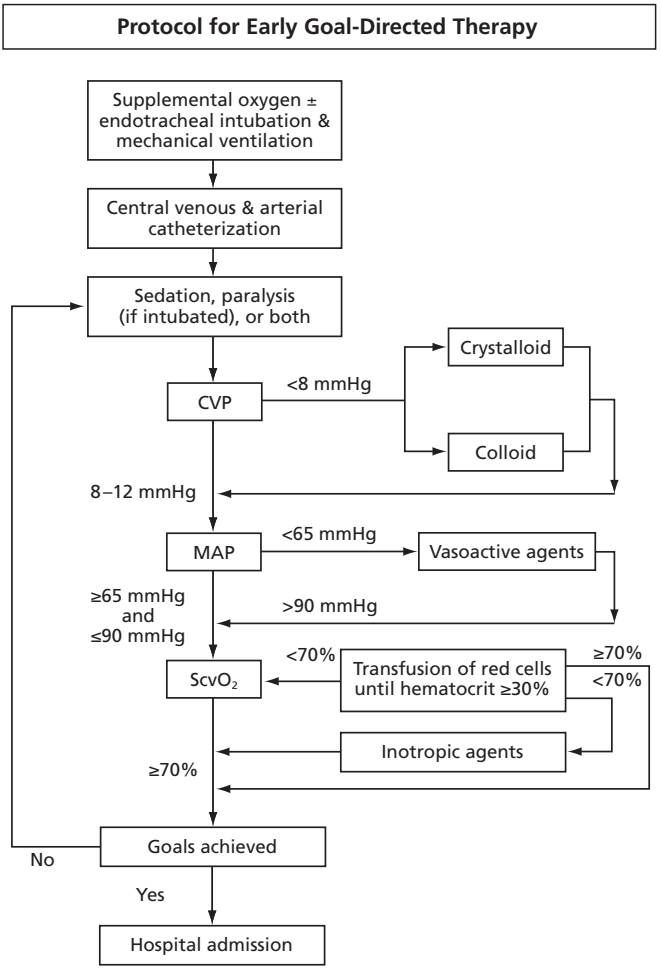


* *Used within the limitations of SVV as a guide for fluid responsiveness.*

** *Cardiac Output response to fluid challenge or passive leg raising when SVV cannot be used.*

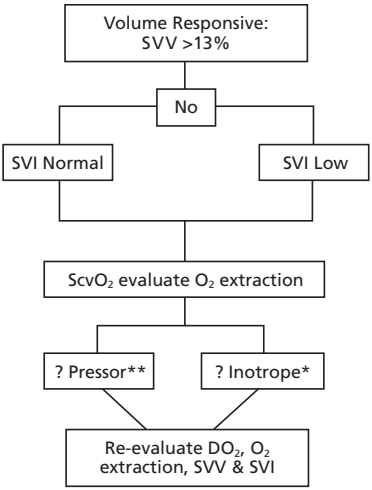
Modified from Pinsky & Vincent. Critical Care Med. 2005;33:1119-22.

EGDT In the Treatment of Sepsis or Septic Shock



Rivers, Emanuel, Nguyen, Bryant, et al; Early Goal-Directed Therapy in the Treatment of Severe Sepsis and Septic Shock: N Engl J Med, Vol. 345, No. 10, 2001.

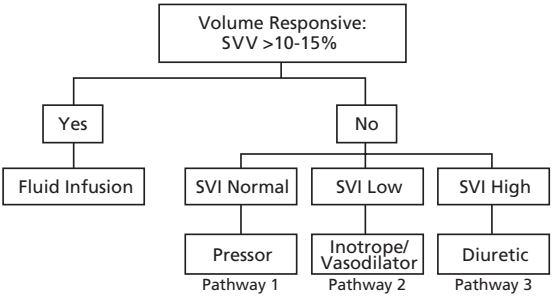
Physiologic Algorithm Using SVV, SVI and $ScvO_2$



* *If O_2 extraction is high, an inotrope may be required to provide perfusion support.*

** *As individual organ perfusion may also depend on blood pressure, a MAP target > 60-65 mmHg may require a vasopressor even when O_2 extraction is normal.*

Physiologic Optimization Program Using SVV and SVI In Hypotensive and/or Oliguric Patients



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

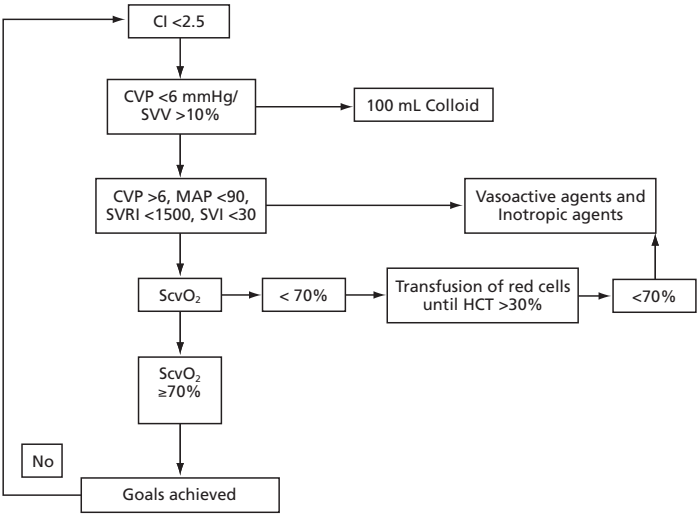
For non-volume responsive patients, SVV < 10-15% the physiology is interrogated at the level of cardiac performance on a beat to beat basis. Ultimately with this approach, many patients will develop a SVI ≥ normal (pathway 1). This represents resuscitated septic shock, and these patients may be safely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

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

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

McGee WT, Mailloux PT: Applied physiology and the hemodynamic management of septic shock utilizing the Physiologic Optimization Program. Chapter 12 In: Severe Sepsis and Septic Shock. INTECH Publisher, Croatia. 2012, pp. 255-272.

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



Malholtra PK, Kakani M, Chowdhury U, Choudhury M, Lakshmy R, Kiran U. Early goal-directed therapy in moderate to high-risk cardiac surgery patients. Ann Card Anaesth 2008;11:27-34.

Typical Hemodynamic Profiles in Various Acute Conditions

Condition	HR	MAP	CO/ CI	CVP/ RAP	PAP/PAOP	Notes
Left Ventricular Failure	↑	↓	↓	↑	↑	
Pulmonary Edema (Cardiogenic)	↑	N,↓	↓	↑	↑PAOP >25 mmHg	
Massive Pulmonary Embolism	↑	↓	↓	↑ N	↑PAD > PAOP by >5 mmHg	↑PVR
Acute Ventricular Septal Defect	↑	↓	↓	↑	↑giant "v" waves on PAOP tracing	O_2 step up noted in SvO_2
Acute Mitral Valve Regurgitation	↑	↓	↓	↑	↑giant "v" waves on PAOP tracing	No O_2 step up noted in SvO_2
Cardiac Tamponade	↑	↓	↓	↑	↑CVP, PAD and PAOP equalized	↓RVEDVI
Right Ventricular Failure	↑,V	↓,V	↓	↑	PAP ↑, PAOP N/↓	↑RVEDVI
Hypovolemic Shock	↑	↓	↓	↓	↓	↑Oxygen extraction ↑SVR
Cardiogenic Shock	↑	↓	↓	N,↑	↑	↑Oxygen extraction ↑SVR
Septic Shock	↑	↓	↓	↓,N	↓,N	SVR changes, ↓Oxygen extraction ↓SVR

Charts, Classifications, Scales and Systems

NEW YORK HEART CLASSIFICATION OF CARDIOVASCULAR DISEASE

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

FORRESTER CLASSIFICATION HEMODYNAMIC SUBSETS OF ACUTE MYOCARDIAL INFARCTION

Subset	Clinical Description	Cardiac index L/min/m^2	PAOP mmHg	Therapy
I	No Failure	2.7 ± 0.5	12 ± 7	Sedate
II	Isolated Pulmonary Congestion	2.3 ± 0.4	23 ± 5	Normal BP: Diuretics ↑ BP: Vasodilators
III	Isolated Peripheral Hypoperfusion	1.9 ± 0.4	12 ± 5	↑ HR: Add volume ↓ HR: Pacing
IV	Both Pulmonary Congestion and Hypoperfusion	1.6 ± 0.6	27 ± 8	↓ BP: Inotropes Normal BP: Vasodilators

GLASGOW COMA SCALE

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

ATLS CHART

Estimated Fluid and Blood Requirements in a 70kg Male INITIAL PRESENTATIONS				
	Class I	Class II	Class III	Class IV
Blood loss (mL)	<750	750–1500	1500–2000	>2000
Blood loss (% blood volume)	<15%	15%–30%	30%–40%	>40%
Pulse rate (bpm)	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (bpm)	14–20	20–30	30–40	>35
Urine output (mL/hr)	30 or more	20–30	5–15	Negligible
CNS-Mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid + blood	Crystalloid + blood

FLUID CHALLENGE GUIDELINE CHART

BASELINE VALUES		
PAOP* mmHg	**Challenge Volume Amount/10 Minutes**	**CVP* mmHg**
<12 mmHg	200 mL or 20 cc/minute	<6 mmHg
12–16–18 mmHg	100 mL or 10 cc/minute	6–10 mmHg
>16–18 mmHg	50 mL or 5 cc/minute	>10 mmHg

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

Optional Baseline RVEDVI Value Guidelines:

- If RVEDVI <90 mL/m^2 or mid-range 90-140 mL/m^2, administer fluid challenge
- If RVEDVI >140 mL/m^2, do not administer fluid challenge

** References differ on PAOP and CVP ranges*

APACHE II SEVERITY OF DISEASE CLASSIFICATION SYSTEM

	High Abnormal Range					Low Abnormal Range			
	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature- rectal (°C)	≥41°	39–40.9°		38.5°–38.9°	36°–38.4°	34°–35.9°	32°–33.9°	30°–31.9°	≤29.9°
Mean Arterial Pressure - mmHg	≥160	130–159	110–129		70–109		50–69		≤49
Heart Rate (ventricular response)	≥180	140–179	110–139		70–109		55–69	40–54	≤39
Respiratory Rate (bpm) (non-ventilated or ventilated)	≥50	35–49		25–34	12–24	10–11	6–9		≤5
Oxygenation A-aDO_2 or PaO_2 (mmHg) a. FIO_2 ≥0.5 record A-aDO_2 b. FIO_2 ≤0.5 record only PaO_2	≥500	350-499	200-349		<200 PO_2>7	PO_2 61-70		PO_2 55-60	PO_2 <55
Arterial pH	≥7.7	7.6–7.69		7.5–7.59	7.33–7.49		7.25–7.32	7.15–7.24	<7.15
Serum Sodium (mMol/L)	≥180	160–179	155–159	150–154	130–149		120–129	111–119	≤110
Serum Potassium (mMol/L)	≥7	6–6.9		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9		<2.5
Serum Creatinine (mg/100 mL) (Double point score for acute renal failure)	≥3.5	2–3.4	1.5–1.9		0.6–1.4		<0.6		
Hematocrit (%)	≥60		50–59.9	46–49.9	30–45.9		20–29.9		<20
White Blood Count (total/ mm3) (in 1,000s)	≥40		20–39.9	15–19.9	3–14.9		1–2.9		<1
Glasgow Coma Scale (GCS) Score = 15 minus actual GCS									
A. Acute Physiology Score (APS): Sum of the 12 individual variable points from the chart above.									
Serum HCO_3 (venous-mMol/L) [Not preferred, use if no ABGs]	≥52	41–51.9		32–40.9	22–31.9		18–21.9	15–17.9	<15

B. Age Points:

Assign points to age as shown in chart at right:

Age (years)	Points
≤44	0
45–54	2
55–64	3
65–74	5
≥75	6

C. Chronic Health Points:

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

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

Definitions

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

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

Cardiovascular: New York Heart Association Class IV.

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

Renal: Receiving chronic dialysis.

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

APACHE II Score

Sum of A + B + C

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

Total Apache II

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

Recommendations for Pulmonary Artery Catheter Monitoring:

Class I

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

Class IIa

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

Class III

1. Pulmonary artery catheter monitoring is not recommended in patients with STEMI without evidence of hemodynamic instability or respiratory compromise.

Recommendations for Intra-arterial Pressure Monitoring:

Class I

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

Class II

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

Class IIb

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

Class III

1. Intra-arterial pressure monitoring is not recommended for patients with STEMI who have no pulmonary congestion and have adequate tissue perfusion without use of circulatory support measures.

Normal Hemodynamic Parameters and Laboratory Values

NORMAL HEMODYNAMIC PARAMETERS – ADULT

Parameter	Equation	Normal Range
Arterial Blood Pressure (BP)	Systolic (SBP) Diastolic (DBP)	100–140 mmHg 60–90 mmHg
Mean Arterial Pressure (MAP)	SBP + (2 x DPB)/3	70–105 mmHg
Right Atrial Pressure (RAP)		2–6 mmHg
Right Ventricular Pressure (RVP)	Systolic (RVSP) Diastolic (RVDP)	15–30 mmHg 0–8 mmHg
Pulmonary Artery Pressure (PAP)	Systolic (PASP) Diastolic (PADP)	15–30 mmHg 8–15 mmHg
Mean Pulmonary Artery Pressure (MPAP)	PASP + (2 x PADP)/3	9–18 mmHg
Pulmonary Artery Occlusion Pressure (PAOP)		6–12 mmHg
Left Atrial Pressure (LAP)		4–12 mmHg
Cardiac Output (CO)	HR x SV/1000	4.0–8.0 L/min
Cardiac Index (CI)	CO/BSA	2.5–4.0 L/min/m^2
Stroke Volume (SV)	CO/HR x 1000	60–100 mL/beat
Stroke Volume Index (SVI)	CI/HR x 1000	33–47 mL/m^2/beat
Stroke Volume Variation (SVV)	SVmax–SVmin/SVmean x 100	<10–15%
Systemic Vascular Resistance (SVR)	80 x (MAP–RAP)/CO	800–1200 dynes-sec-cm^{-5}
Systemic Vascular Resistance Index (SVRI)	80 x (MAP–RAP)/CI	1970–2390 dynes-sec-cm^{-5}•m^2
Pulmonary Vascular Resistance (PVR)	80 x (MPAP–PAOP)/CO	<250 dynes-sec-cm^{-5}
Pulmonary Vascular Resistance Index (PVRI)	80 x (MPAP–PAOP)/CI	255–285 dynes-sec-cm^{-5}•m^2
Left Ventricular Stroke Work Index (LVSWI)	SVI x (MAP–PAOP) x 0.0136	50–62 g/m^2/beat
Right Ventricular Stroke Work Index (RVSWI)	SVI x (MPAP–CVP) x 0.0136	5–10 g/m^2/beat
Coronary Artery Perfusion Pressure (CPP)	Diastolic BP–PAOP	60–80 mmHg
Right Ventricular End-Diastolic Volume (RVEDV)	SV/EF	100–160 mL
Right Ventricular End-Diastolic Volume Index (RVEDVI)	RVEDV/BSA	60–100 mL/m^2
Right Ventricular End-Systolic Volume (RVESV)	EDV–SV	50–100 mL
Right Ventricular Ejection Fraction (RVEF)	SV/EDV x 100	40–60%

OXYGEN PARAMETERS – ADULT

Parameter	Equation	Normal Range
Partial Pressure of Arterial Oxygen (PaO_2)		75–100 mmHg
Partial Pressure of Arterial CO_2 ($PaCO_2$)		35–45 mmHg
Bicarbonate (HCO_3)		22–26 mEq/L
pH		7.34–7.44
Arterial Oxygen Saturation (SaO_2)		95–100%
Mixed Venous Saturation (SvO_2)		60–80%
Central Venous Oxygen Saturation ($ScvO_2$)		70%
Arterial Oxygen Content (CaO_2)	(0.0138 x Hgb x SaO_2) + 0.0031 x PaO_2	16–22 mL/dL
Venous Oxygen Content (CvO_2)	(0.0138 x Hgb x SvO_2) + 0.0031 x PvO_2	15 mL/dL
A-V Oxygen Content Difference ($C(a-v)O_2$)	$CaO_2 - CvO_2$	4–6 mL/dL
Oxygen Delivery (DO_2)	CaO_2 x CO x10	950–1150 mL/min
Oxygen Delivery Index (DO_2I)	CaO_2 x CI x 10	500–600 mL/min/m^2
Oxygen Consumption (VO_2)	$C(a-v)O_2$ x CO x 10	200–250 mL/min
Oxygen Consumption Index (VO_2I)	$C(a-v)O_2$ x CI x 10	120–160 mL/min/m^2
Oxygen Extraction Ratio (O_2ER)	($CaO_2 - CvO_2$)/CaO_2 x 100	22–30%
Oxygen Extraction Index (O_2EI)	($SaO_2 - SvO_2$)/SaO_2 x 100	20–25%
Extra Vascular Lung Water (EVLW) Extra Vascular Lung Water Index (ELWI)	CO x DSt - 0.25 GEDV EVLW/PBW Predicted Body Weight (PBW): Female: 45.5 = 0.91 x (Height-152.4) Male: 50 = 0.91 x (Height-152.4)	3-7 mL/kg
Global End Diastolic Volume (GEDV) Global End Diastolic Volume Index (GEDI)	CO x MTt x f(S1/S2) CI x MTt x f(S1/S2)	680-800 mL/m^2
Global Ejection Fraction (GEF)	SV x 4 / GEDV	>20%
Cardiac Function Index (CFI)	1000 x CO / GEDV	4.5-6.6 1/min
Intra Thoracic Blood Volume (ITBV) Intra Thoracic Blood Volume Index (ITBI)	ITBV = 1.25 x GEDV ITBI = 1.25 x GEDI	850-1000 mL/m^2
Pulmonary Vascular Permeability Index (PVPI)	EVLW/0.25 x GEDV	<3
Cardiac Power (CPO) Cardiac Power Index (CPI)	CO x MAP x *K* CI x MAP x *K*	0.5-0.7 W/m^2

NORMAL BLOOD LABORATORY VALUES

Test	Conventional Units (Reference Values)	SI Units
Chemistry Studies		
Sodium (Na)	135–145 mEq/L	135–145 mmol/L
Potassium (K)	3.5–5.0 mEq/L	3.5–5.0 mmol/L
Chloride (CI)	100–108 mEq/L	100–108 mmol/L
Carbon Dioxide (CO_2)	22–26 mEq/L	22–26 mmol/L
Glucose (BS)	70–100 mg/dL	3.9–6.1 mmol/L
Blood Urea Nitrogen (BUN)	8–20 mg/dL	2.9–7.5 mmol/L
Creatine kinase (CK)	Males: 55–170 U/L Females: 30–135 U/L	Males: 0.94–2.89 μkat/L Females: 0.51–2.3 μkat/L
Creatinine	0.6–1.2 mg/dL	53–115 μmol/L
Calcium (Ca)	8.2–10.2 mEq/L	2.05–2.54 mmol/L
Magnesium (Mg)	1.3–2.1 mg/dL	0.65–1.05 mmol/L
Bilirubin (direct/indirect)	<0.5–1.1 mg/dL	<6.8–19 μmol/L
Amylase	25–85 U/L	0.39–1.45 μkat/L
Lipase	<160 U/L	<2.72 μkat
Anion Gap	8–14 mEq/L	8–14 mmol/L
Lactate	0.93–1.65 mEq/L	0.93–1.65 mmol/L
Alanine Aminotransferase (ALT, GPT)	8–50 IU/L	0.14–0.85 μkat/L
Aspartate Aminotransferase (AST, GOT)	7–46 U/L	0.12–0.78 μkat/L
Hemotologic Studies		
Red Blood Cells	Males: 4.5–5.5 million/μL Females: 4–5 million/μL	4.5–5.5 x 10^{12}/L 4–5 x 10^{12}/L
White Blood Cells (WBC)	4,000–10,000/μL	4–10 x 10^9/L
Hemoglobin (Hgb)	Males: 12.4–17.4 g/dL Females: 11.7–16 g/dL	124–174 g/L 117–160 g/L
Hematocrit (Hct)	Males: 42%–52% Females: 36%–48%	0.42–0.52 0.36–0.48

Test	Conventional Units (Reference Values)	SI Units
Lipids/Lipoproteins Studies		
Total Cholesterol: Desirable Range	Males: <205 mg/dL Females: <190 mg/dL	<5.3 mmol/L <4.9 mmol/L
LDL Cholesterol: Desirable Range	<130 mg/dL	<3.36 mmol/L
HDL Cholesterol: Desirable Range	Males: 37–70 mg/dL Females: 40–85 mg/dL	0.96–1.8 mmol/L 1.03–2.2 mmol/L
Triglycerides	Males: 44–180 mg/dL Females: 11–190 mg/dL	0.44–2.01 mmol/L 0.11–2.21 mmol/L
Coagulation Studies		
Platelet Count	150,000–400,000/mm^3	
Prothrombin Time (PT)	10–13 sec	
International Normalized Ratio (INR)	2.0–3.0 for pts. on warfarin therapy; 2.5–3.5 for pts. with mech. prosthetic heart valves	
Plasma Thrombin Time (PTT)	60–70 sec	
Activated Partial Thromboplastin Time (APTT)	35–45 sec	
Activated Clotting Time (ACT)	107 ± 13 sec	
Fibrin Split Product (FSP)	<10 μg/mL	<10 mg/L
D-dimer	Neg. or <250 μg/L	
Fibrinogen	200–400 mg/dL	2–4 g/L

SI Units = International Units

NORMAL BLOOD LABORATORY VALUES [CONT.]

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

SI Units = International Units

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

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Notes

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