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QUICK GUIDE TO PEDIATRIC CARDIOPULMONARY CARE

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QUICK GUIDE TO

Pediatric Cardiopulmonary Care

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ISBN 978-0-615-27887-2

Acknowledgements

A special thank you to Pom Chaiyakal, Adriana LeBer, Sheryl Stewart, Susan Willig and Melody Frieda for their guidance and expertise.

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Brian Boville, David Nelson and Caulette Young are paid consultants of Edwards Lifesciences.

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QUICK GUIDE TO

Pediatric Cardiopulmonary Care

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Pertinent Clinical Information Dedicated to the Critical Care Clinician

In 1998, the first *Quick Guide to Cardiopulmonary Care* adult version was published. The intent of the *Quick Guide* was to provide a ready reference for hemodynamic monitoring and oxygenation assessment of the critically ill.

This 1st Edition of the *Pediatric Quick Guide to Cardiopulmonary Care* reflects current practice and changes in technology. Patients cared for in pediatric critical care units can range from newborns to adults, with a variety of illnesses and injuries, acquired or congenital. Caring for such a wide range of ages, sizes and disease states present constant challenges within the pediatric critical care environment as patient acuity has also increased. With advancements in technology, medications, treatment options and practitioners' skill, opportunity for survival has increased with improved monitoring capability.

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. It covers key anatomy and physiology concerns commonly seen in the pediatric critical care unit.

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 venous oxygen saturation assessment, discussed in the next section.

Although less frequently utilized in pediatric critical care units, the Swan-Ganz catheter has been the hallmark of changing critical care practice since the early 1970s. The final section can be used as a quick reference section for calculations and various scoring systems commonly used.

Because the practice of critical care and its related technologies are always changing and improving, the *Quick Guid*e 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.

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Notes

Anatomy and Physiology

ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972

Anatomy and Physiology of Oxygenation

Oxygenation

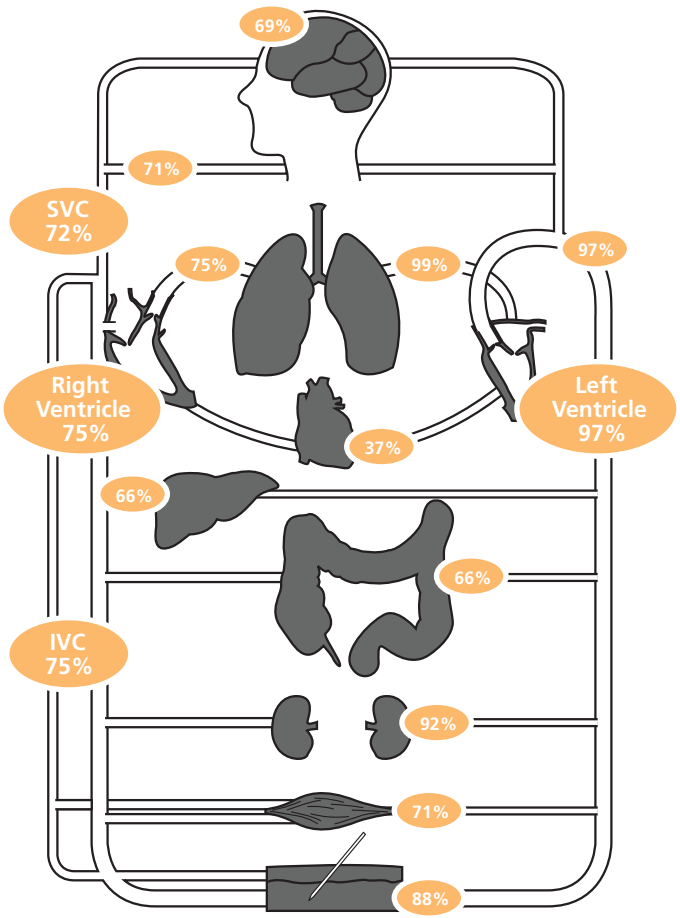
Ensuring that tissues receive adequate oxygen and are able to consume the amount required, is an important part of assessing the critically ill patient. Therefore, the goal of cardiorespiratory monitoring is to evaluate the components of oxygen delivery and consumption, and to assess the utilization 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.

NORMAL OXYGEN SATURATION (%) IN VARIOUS CHAMBERS AND VESSELS

Site	Acyanotic	Cyanotic
SVC (superior vena cava)	75%	55%*
RA / RV (atrium / ventricle)	75%	67%* / 80%*
PA (pulmonary artery)	75%	88%*
Ao (Aorta)	95%	80%*
LA / LV (atrium / ventricle)	95%	90%*
IVC (inferior vena cava)	78%	

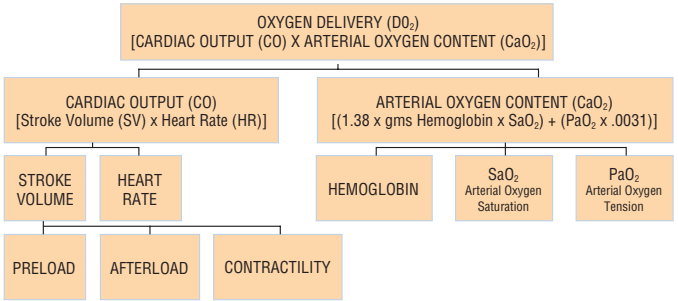
* Values dependent upon lesion type, consult a cardiologist recommended.

APPROXIMATE VENOUS OXYGEN SATURATION PERCENTAGES



Oxygen Delivery (DO_2) ($DO_2 = CO_2 \times CO \times 10$)

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 (CaO_2 or CvO_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 15.6 mL/dL

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

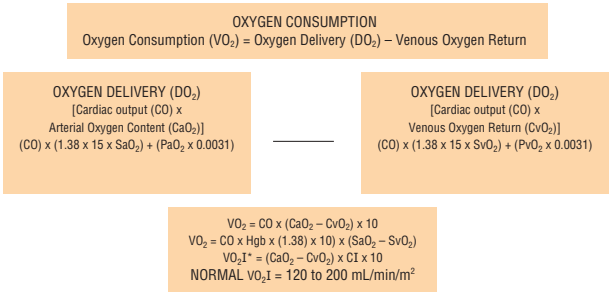
Arterial oxygen delivery (DO_2): $CO \times CaO_2 \times 10$
0.8-4.0 L/min x 20.1 mL/dL x 10 = 160 to 804 mL/min†

Venous oxygen return: $CO \times CvO_2 \times 10$
0.8-4.0 L/min x 15.5 mL/dL x 10 = 124 to 620 mL/min†

**Oxygen carrying capacity has been referenced between 1.34-1.39.*
†Assumes Hgb of 15 gm/dl.
Cardiac output ranges (0.8-4.0 L/min) based on neonatal/pediatric ranges.

Oxygen Consumption (VO_2)

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 index (VO_2I) is calculated using cardiac index rather than cardiac output.



Oxygen Consumption (VO_2)

Arterial Oxygen Transport – Venous Oxygen Transport

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

$$VO_2I = (CaO_2 - CvO_2) \times CI \times 10)$$

Normal $VO_2I = 120 - 200$ mL/min^2

Note: 13.8 = 1.38 x 10

**Usually indexed against body surface area in pediatrics*

ACUTE INCREASES IN SvO_2 IN PEDIATRICS

↑ Cardiac Output	↓ O_2 Demand	↑ Arterial O_2 Content
↑ Heart rate	Analgesia, sedation	Transfusion
↑ Stroke volume	Treatment of fever	Improved oxygenation
Convert dysrhythmia	Anesthesia	Improved ventilation
Volume	NM* blockade	
Inotropic support	Neutral thermal	
Vasodilator therapy	environment	
	Ventilation support	

*NM = neuromuscular

ACUTE DECREASES IN SvO_2 IN PEDIATRICS

↓ Cardiac Output	↑ O_2 Demand	↓ Arterial O_2 Content
Bradycardia	Fever	Anemia
Tachyarrhythmias	Shivering	Hypoventilation
↓ Stroke volume	Pain	Hypoxemia
Hypovolemia	Anxiety	Airway obstruction
Cardiac dysfunction	Cold stress	Pulmonary edema
↑ Afterload	↑ Work of breathing	Atelectasis
Cardiac tamponade	Seizures	Pneumothorax
		ETT dislodgement
		Suctioning
		Intra-cardiac shunting

CONDITIONS AND ACTIVITIES ALTERING DEMAND AND VO_2

Fever (per°C)	10%	Work of breathing	40%
Shivering	50-100%	Post-op procedure	7%
ETT suctioning	7-70%	MODS Multi-organ dysfunction syndrome	20-80%
Sepsis	50-100%	Dressing change	10%
Visitor	22%	Bath	23%
Position change	31%	Chest X-Ray	25%
Weighing patient	36%		

Arterial-Venous Oxygen Difference

An a-vDO_2 difference in the difference between the oxygen content in the arterial (CaO_2) and venous (CvO_2) blood in mL/L, also known as the arterial-mixed venous oxygen content difference (a-vDO_2).

a-vDO_2 = CaO_2 – CvO_2
Normal range = 20-78 mL/L

Based upon Fick's assumption, the amount of oxygen extracted by the body in the blood is equal to the amount of oxygen uptake in the lungs during respiration.

Oxygen Extraction Ratio

O_2ER: normally 22 – 30%
O_2ER: CaO_2 – CvO_2 / CaO_2 x 100
*CaO_2 = 20.1 *CvO_2 = 15.6
O_2ER = 20.1 – 15.6 / 20.1 x 100 = 22.4%

Oxygen Extraction Index

Evaluates the efficiency of oxygen extraction ratio. Reflects the ability of cardiac reserve to increase during an increase in oxygen demand.
Normal range is 20%–30%.
O_2EI = SaO_2 – SvO_2 / SaO_2 x 100 (SaO_2 = 99, SvO_2 = 75)
O_2EI = 99 – 75 / 99 x 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 x CO x 10
CO = VO_2 / C(a – v)O_2
C(a – v)O_2 = VO_2 / (COx10)
S(a – v)O_2 = VO_2 / (COx10)

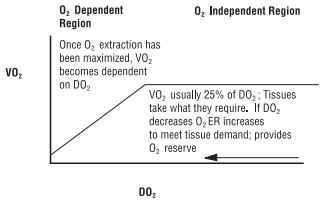
When Fick equation is rearranged, the determinants of SvO_2 are the components of oxygen delivery and consumption:
If SaO_2 = 1.0, then SvO_2 = CvO_2 / CaO_2
SvO_2 = 1 – [VO_2 / (CO x 10 x CaO_2)]
SvO_2 = 1 – (VO_2 / DO_2) x 10

As a result, SvO_2 reflects changes in oxygen extraction and the balance between DO_2 and VO_2.
**See oxygen content section for derived values*

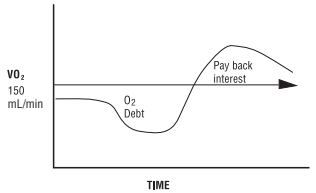
VO_2/DO_2 Relationships

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.

NORMAL RELATION



OXYGEN DEBT CONCEPT



Oxygen debt occurs when the delivery of oxygen is insufficient to meet the body requirements.

Factors Influencing Accumulation of O_2 Debt

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

Cardiac Functional Anatomy

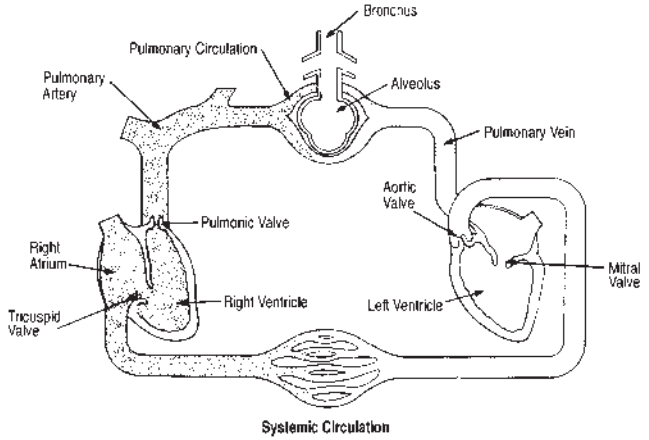
For hemodynamic monitoring purposes, the right and left heart are differentiated as to function, structure and pressure generation. The pulmonary capillary bed lies between the right and left heart. The capillary bed is a compliant system with a high capacity to sequester blood.

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

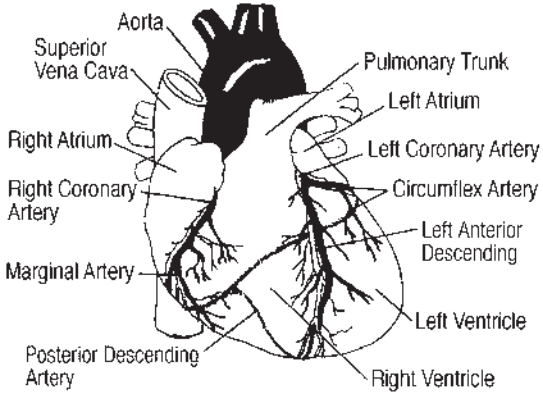
The two major branches of the coronary arteries arise from each side of the aortic root. Each coronary artery lies in the atrioventricular sulcus and is protected by a layer of adipose tissue. Illustrations and descriptions based on normal anatomy and distribution.

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

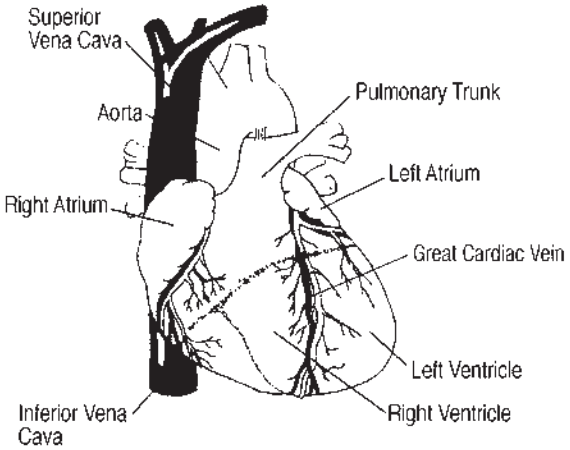
CORONARY ARTERIES

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



CORONARY VEINS

Blood is drained by branches of the cardiac veins.

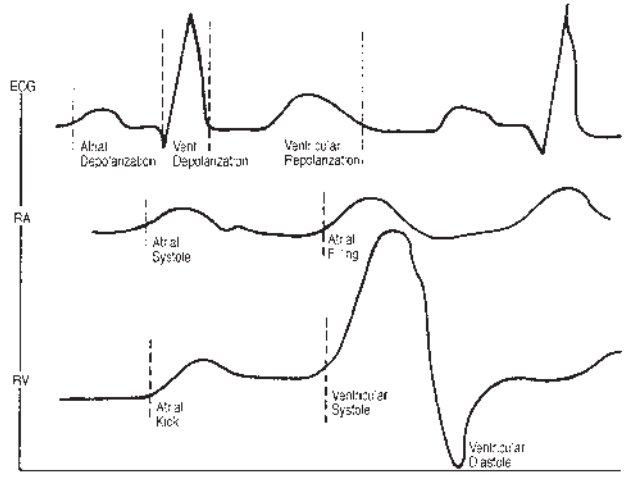


Cardiac Cycle: Electrical Correlation to Mechanical

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

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

ELECTRICAL – MECHANICAL CARDIAC CYCLE

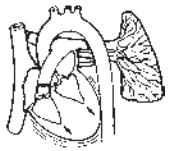


Terminology Relative to Cardiac Cycle

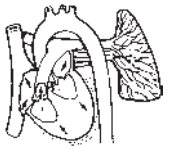
Inotropy: influence on cardiac contractility
Chronotropy: has effect on heart rate
Lusitropy: describes myocardial relaxation
Dromotropy: influences electrical impulses or conduction velocity
Bathmotropy: influence on myocardial excitability or irritability

Mechanical Cardiac Cycle Phases

SYSTOLE



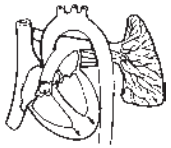
1. Isovolumetric Phase
Follows QRS of ECG
All valves closed
Majority of oxygen consumed



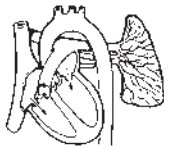
2. Rapid Ventricular Ejection
Aortic valve opens
Occurs during ST segment
2/3 or more of blood volume ejected

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

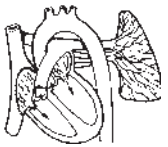
DIASTOLE



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



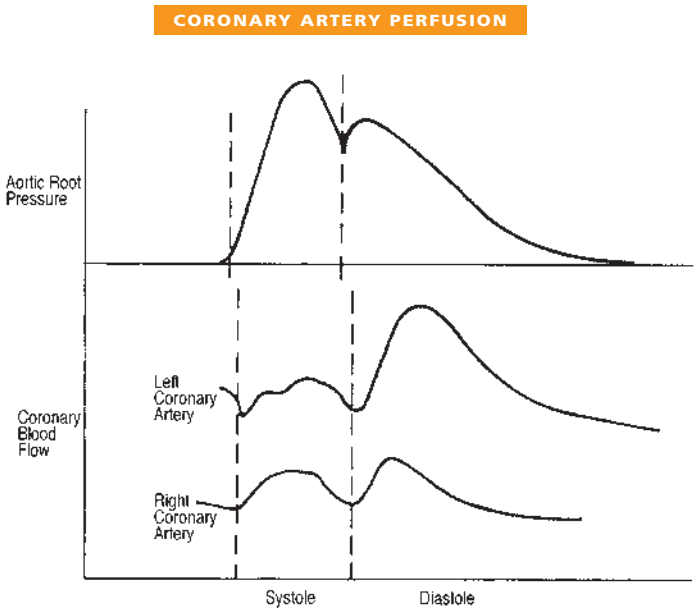
2. Rapid Ventricular Filling
AV valves open
Approximately 70% of blood volume goes into ventricle



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. Due to less resistance, more blood flows through the right coronary artery during systole. Optimal RV performance depends in part on this biphasic perfusion. There must be adequate diastolic pressure in the aortic root for both coronary arteries to be perfused.



Cardiac Output Definition

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

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

Normal Cardiac Output:	0.8 – 1.3 L/min (neonate/infant) 1.3 – 3.0 L/min (child) 4 – 8 L/min (adolescent/adult)

Normal Cardiac Index:	4.0 – 5.0 L/min/m^2 (neonate/infant) 3.0 – 4.5 L/min/m^2 (child) 2.5 – 4 L/min/m^2 (adolescent/adult)
CI	= CO/BSA
BSA	= Body Surface Area

Normal Heart Rate Range:	100 – 180 beats/min (neonate/infant) 70 – 110 beats/min (child) 60 – 100 beats/min (adolescent/adult)

Normal Stroke Volume:	5 – 13 mL/beat (neonate/infant) 13 – 50 mL/beat (child) 60 – 100 mL/beat (adolescent/adult)

Stroke volume: difference between end-diastolic volume (EDV), [the amount of blood in the ventricle at the end of diastole]; and end-systolic volume (ESV), [blood volume in the ventricle at the end of systole].

SV = EDV – ESV

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

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

CIRCULATING BLOOD VOLUME & STROKE VOLUME BY AGE

Age	Circulating Volume (mL/Kg)	Stroke Volume (SV) (mL/beat)
Neonate	85–90mL/Kg	5mL/beat
Infant	75–80mL/Kg	5–13mL/beat
Child	70–75mL/Kg	13–50mL/beat
Adolescent	65–70mL/Kg	50–85mL/beat
Adults	60–65mL/Kg	60–130mL/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 is 54 – 75%.

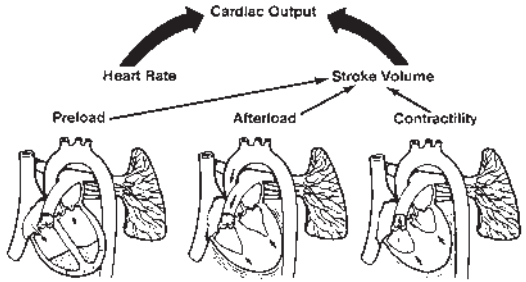
EF = (SV / EDV) x 100

Shortening Fraction (SF)

Represents percent changes in ventricular diameter calculated from end-diastolic (D_ed) and end-systolic (D_es) diameters measured by M-mode echocardiography. Normal values range 30-40%.

$$SF = \frac{D_ed - D_es}{D_ed} \times 100$$

DETERMINANTS OF CARDIAC OUTPUT



DIRECTED BLOOD-FLOW PERCENTAGE OF CARDIAC OUTPUT

Organ	Infants	Adults
Brain	34%	14.3%
Coronary	3%	4.3%
Splanchnic	25%	28.6%
Renal	18%	25.7%
Muscle	10%	11.4%
Fat	5%	10%
Flow to poorly perfused tissue	5%	10%

ASSESSED "PERFUSION PARAMETERS" FOR CARDIAC OUTPUT IN PEDIATRICS

Perfusion Parameter	Adequate	Altered	Poor
Level of consciousness	Appropriate for age	Irritability	Unresponsive
Urinary output	>1 mL/Kg/hr	0.5 mL/Kg/hr	<0.5 mL/Kg/hr
Temperature	36.5–38° C	<36.5–>38° C	<36–>38.5° C
Quality of pulses	+2 (normal)	+3 to +4 (bounding)	+1 to absent
Capillary refill time	<2 seconds	2–4 seconds	>5 seconds

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 it eliminates the influence of ventricular compliance on pressure.

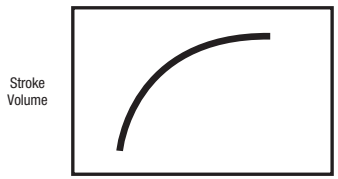
Preload

RAP/CVP:	0 – 8 mmHg (neonate/infant) 2 – 6 mmHg (pediatric/adult)
PADP:*	3 – 12 mmHg
PAOP/LAP:	1 – 10 mmHg
RVEDV:	100 – 160 mL (adult)
LVEDV:	32–52 mL/m^2 (neonate) 62-85 mL/m^2 (pediatric)

Frank–Starling Law

Frank and Starling (1895, 1918) identified the relationship between myocardial fiber length and force of contraction. The larger 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

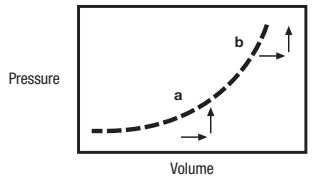


*Pulmonary artery diastolic pressure

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

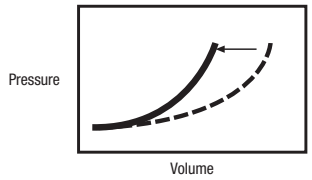


Normal Compliance

Pressure/volume relationship is curvilinear:

a: Large increase in volume = small increase in pressure

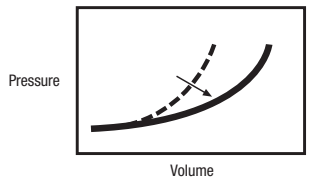
b: Small increase in volume = large increase in pressure



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 formulae 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):

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

<2000–3200 dynes/sec/cm^{-5} (infant)
<40–320 dynes/sec/cm^{-5} (child)
<250 dynes/sec/cm^{-5} (adult)

When measured as Woods units/m^2:
8–10 Woods units/m^2 (<8 weeks)
1–3 Woods units/m^2 (>8 weeks)

Systemic Vascular Resistance (SVR):

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

2800–4000 dynes/sec/cm^{-5} (infant)
1200–2800 dynes/sec/cm^{-5} (child)
800–1200 dynes/sec/cm^{-5} (adult)

When measured as Woods units/m^2:
10–15 Woods units/m^2 (infants)
15–20 Woods units/m^2 (1–2 years)
15–30 Woods units/m^2 (child)

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.

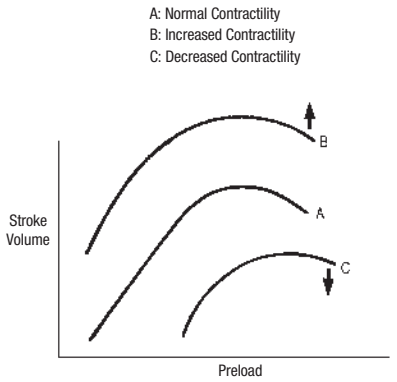
Contractility Definition and Measurements

Inotropy 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 include all determinants of preload and afterload.

Contractility

Stroke Volume	5–13 mL/beat (neonate/infant) 13–50 mL/beat (child) 50–130 mL/beat (adolescent/adult)
SV = (CO x 1000)/HR	
SVI = SV/BSA	30 – 60 mL/beat/m^2
Left Ventricular Stroke Work Index	50 – 62 g/m^2/beat
LVSWI = SVI (MAP – PAOP) x 0.0136	
Right Ventricular Stroke Work Index	6 ± 0.9 g/m^2/beat
RVSWI = SVI (PA mean – CVP) x 0.0136	

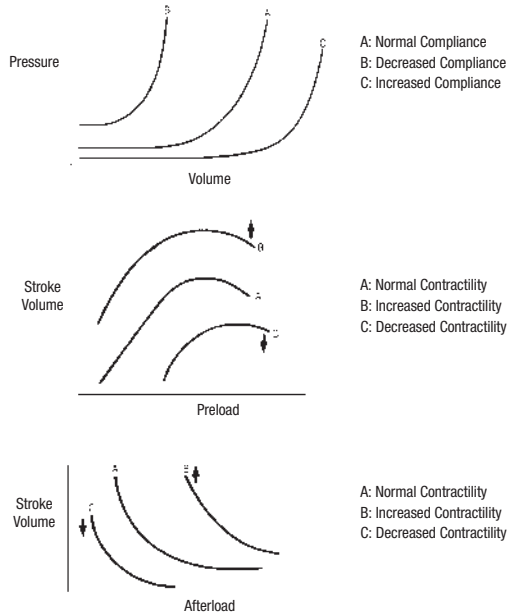
VENTRICULAR FUNCTION CURVES



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.

COMPLIANCE CURVES



CARDIAC PHYSIOLOGICAL DIFFERENCES

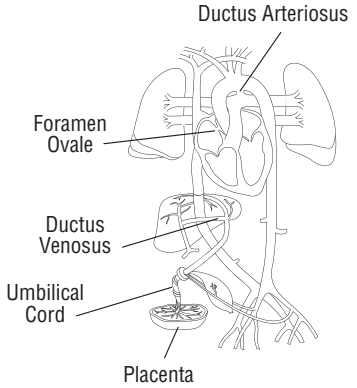
Physiological Differences	Neonate/Infant	Adult
Cardiac Output	Rate dependent	Increased via SV or HR
Contractility	Decreased	Normal
Starling Response	Limited	Normal
Catecholamine Response	Decreased	Normal
Compliance	Decreased	Normal
Afterload Mismatch	Susceptible	Resistant
Ventricular Interdependence	Increased	Normal

Fetal and Neonatal Concerns

Fetal Circulation

Fetal circulation is designed to divert oxygenated blood from the placenta to the brain and away from the lungs via anatomic fetal shunts (foramen ovale and ductus venosus); and then returns the blood to the placenta (ductus arteriosus), where oxygenation and gas exchange take place. In fetal circulation, PVR remains high since the lungs are fluid filled and the alveoli do not participate in oxygenation. The SVR remains low so that blood can return to the placenta, which is a low resistance pathway.

FETAL CIRCULATION



In-utero Oxygenation

Fetus in hypoxic state

Fetal Hb allows for greater O_2 saturation = 60-79%

Arterial O_2 tension = 22-29 mmHg

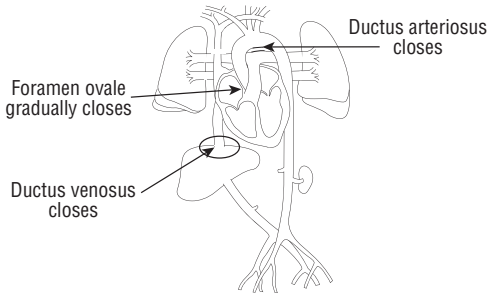
Fetal Hb has less 2, 3 DPG (diphosphoglycerate):
Left-shift in oxyhemoglobin dissociation curve (ODC)

Higher CO (200-300 mL/Kg/min) for adequate tissue perfusion

Peri-natal Circulation Conversion

Structural and physiological changes occur in the newborn period in which the lungs change from being fluid filled, essentially non-functioning during the fetal period, to becoming responsible for oxygenation and gas exchange following delivery. Arterial O_2 tension immediately increases as fluid from the lungs is absorbed by pulmonary capillaries, and then removed by the pulmonary lymphatic system. The ductus arteriosus closes with alveolar ventilation and decrease in PVR. This closure can be delayed in prematurity (<36 weeks gestation), high altitude and congenital heart disease. After separation from the placenta, a sudden increase in SVR occurs. Right heart pressures decrease and left heart pressures increase. Once left heart pressures exceed right heart pressures, the foramen ovale begins to close over a period of a few days to weeks. Left heart pressures continue to increase over several months.

PERI-NATAL CIRCULATION



FETAL/PERINATAL CARDIAC PRESSURES

Cardiac Pressures	Fetal (in-utero)	Perinatal
Right Atrium	4 mmHg	1 mmHg
Right Ventricle	60/5 mmHg	26/2 mmHg
Pulmonary Artery	60/40 mmHg	26/12 mmHg
Left Atrium	2 mmHg	3 mmHg
Left Ventricle	70/50 mmHg	60/3 mmHg
Aorta		70/50 mmHg

Congenital Cardiac Disease and Lesions

Congenital Cardiac Lesions

The first successful repair for a congenital cardiac defect occurred on September 2, 1952, when Dr. F. John Lewis repaired an ASD in a 5-year old girl under direct visualization, using inflow stasis and moderate total body hypothermia. Congenital cardiac surgical repairs are now performed routinely with generally low associated risks.

Adequate tissue oxygenation is the main goal when caring for children with congenital heart disease and is dependent upon adequate cardiac output. These patients can have difficulty achieving adequate cardiac output; if inefficient pump function (i.e. intra- or extra-cardiac shunts, valvular insufficiency or dysrhythmias) or depressed pump function (diminished ventricular contractility) exists. An understanding of the calculations that evaluate the adequacy of cardiac output and tissue oxygen delivery is essential, especially when left-to-right or right-to-left shunting is present. Evaluating pulmonic to systemic flow is an important assessment in shunt dependent lesions.

Intra-cardiac Shunting

Anatomic (intracardiac) shunt:

R ⇨ L	L ⇨ R
blood passing from right side of heart (intracardiac) to left side of heart	blood passing from left side of heart (intracardiac) to right side of heart

Physiologic (extracardiac) shunt:

R ⇨ L	L ⇨ R
systemic venous blood flows to aorta without passing through lungs	pulmonic venous blood flows to PA without passing through systemic circulation

Calculation of Anatomic Shunts

Calculation of anatomic shunts refers to the ratio of pulmonary (Q_p) to systemic (Q_s) ventricular blood flow. In shunt dependent lesions, (single ventricle physiology), maintaining balanced pulmonary to systemic ($Q_p : Q_s$) blood flow is ideal to ensure adequate systemic oxygenation and recognition of pulmonary over-circulation. In single ventricle physiology, mixed venous oxygen saturations can be approximated using $ScvO_2$.

$$Q_p = \frac{VO_2}{1.38 \times 10 \times Hb\ (SpvO_2 - SpaO_2)}$$

$$Q_s = \frac{VO_2}{1.38 \times 10 \times Hb\ (SaO_2 - SvO_2)}$$

$$\frac{Q_p}{Q_s} = \frac{SaO_2 - SvO_2}{SpvO_2 - SpaO_2}$$

Where Q = ventricular flow; $SpvO_2$ represents the O_2 saturation of the pulmonary vein and $SpaO_2$ represents the O_2 saturation of the pulmonary artery.

Calculation of Physiologic Shunts

- Right-to-left, left-to-right shunting

  Q_{ep} = effective pulmonary blood flow (mixed venous blood that ultimately reaches lungs during shunting)

  Q_{es} = effective systemic blood flow

  $Q_{ep} = Q_{es}$ when no shunt exists

- Right-to-left shunting ($Q_{R \rightarrow L}$): $Q_s > Q_{es}$

  $Q_{R \rightarrow L} = Q_s - Q_{es}$ or $Q_{R \rightarrow L} = Q_s - Q_{ep}$

- Left-to-right shunting ($Q_{L \rightarrow R}$): $Q_p > Q_{ep}$

  $Q_{L \rightarrow R} = Q_p - Q_{ep}$

$$SVR = \frac{MAP - RAP}{Q_s} \qquad PVR = \frac{MPAP - MPCWP}{Q_p}$$

In shunt dependent lesions, systemic O_2 saturation is influenced by lung function, pulmonary blood flow and myocardial function.

Vascular Resistance measured during cardiac catheterization is based upon Poiseuille's (constant flow) and Ohm's (vascular resistance) laws. Data measurements must be meticulously collected.

Poiseuille = $Q = (\pi) \times (\Delta P) \times (r^4) / (8) \times (\mu) \times (l)$

Where Q = flow

ΔP = inflow pressure – outflow pressure

r = radius

μ = fluid viscosity (assumed to be constant)

I = length of tube (assumed to be constant)

Ohm = $V = (I)(R)$ or $V = I / R$

Where V = difference in electrical potential (or change in pressure)

I = current (flow)

R = resistance

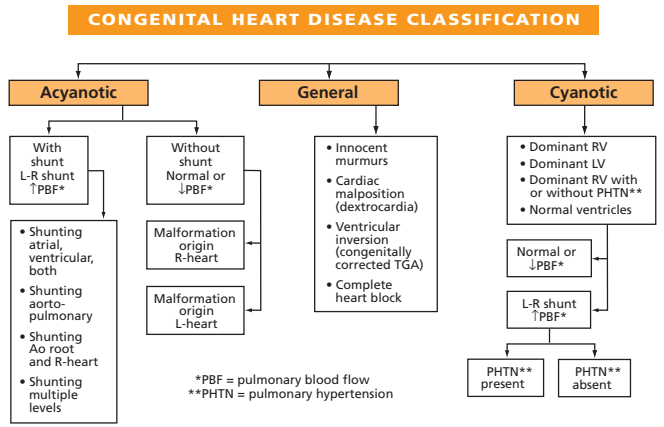
Resistance = pressure / flow:

$$SVR = \frac{\Delta MAP - RAP}{Q_s} = R_s$$

$$PVR = \frac{\Delta MPAP - LAP}{Q_p} = R_p$$

Congenital Heart Disease and Cardiac Lesions

There are over 37 different congenital cardiac lesions requiring various medical and surgical interventions. The most common are illustrated and described later in this chapter.



Shunt Lesions

Aorticopulmonary Window (AP window)
Patent Ductus Arteriosus (PDA)
Atrial Septal Defect (ASD)
Ventricular Septal Defect (VSD)
Arterio-ventricular Canal (A-V canal)
Anomalous Pulmonary Venous Return (TAPVR) total or (PAPVR) partial
Truncus Arteriosus (TA)
Double Outlet Right Ventricle (DORV)

Left Ventricular Outflow Obstruction

Aortic Stenosis (AS) (aortic, subaortic or supravalvular)
Interrupted Aortic Arch (IAA)
Coarctation of the Aorta (Ao Coarc)

Right Ventricular Outflow Obstruction

Tetralogy of Fallot (TOF)
Pulmonary Atresia (PA) with intact ventricular septum (IVS) / pulmonary stenosis (PS)

Single Ventricle Physiology and Considerations

Hypoplastic Left Heart Syndrome	Hypoplastic Right Heart Syndrome
Variations:	Variations:
Mitral Valve Atresia (with Double Outlet LV or Hypoplastic LV)	Tricuspid Valve Artesia (with TGA)
Aortic Valve Atresia (with Hypoplastic LV, or VSD with normal LV)	Double Inlet Left Ventricle (with TGA)
Malaligned Complete A-V Canal (with Hypoplastic LV)	Heterotaxy Syndrome with PA or PS (with asplenia or polysplenia)
Double Outlet Right Ventricle (DORV)	Malaligned Complete A-V Canal (with Hypoplastic RV)
	Ebstein's Anomaly
	Pulmonary Atresia (with intact ventricular septum)

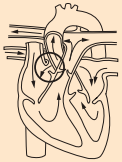
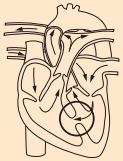
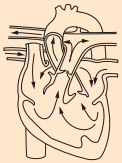
Two Ventricle Hearts with Single Ventricle Physiology

Tetralogy of Fallot with Pulmonary Atresia
Truncus Arteriosus
Total Anomalous Pulmonary Venous Return

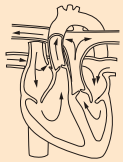
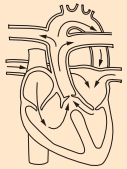
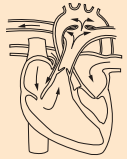
Terminology

- Ambiguous: indeterminate
- Asplenia: congenital absence of a spleen
- Concordant: to agree
- Concordant loop: referring to ventricular loop agreeing with visceral organs (d-loop in situs solitus; l-loop in situs inversus)
- Dextrocardia: heart is located on the right side of the thorax rather than left
- d-loop: rightward bend in embryonic heart tube
- Disconcordant: disagreeing, inappropriate
- Ectopic cordis: displaced location of the heart
- Heterotaxy: different arrangement or order
- Infundibulum: ventriculo-arterial segment which is normally subpulmonary
- Inversion: referring to right and left reversal without change in anterio-posterior or superio-inferior
- Inversus: reversed position
- Isomerism: equal part, referring to bilateral structures that normally are not equal
- Left isomerism: referring to bilateral structures that have a left morphological characteristic
- l-loop: leftward bend in embryonic heart tube
- Levocardia: heart located on left side of thorax
- Polysplenia: numerous spleens
- Right isomerism: referring to bilateral structures that have a right morphological characteristic
- Situs: site or position
- Situs ambiguous: indeterminate position
- Situs inversus: opposite or reversed of normal
- Solitus: normal position
- Ventriculo-arterial concordance: referring to the appropriate connection of the ventricles to the appropriate outflow tracts (RV-PA; LV-Ao)

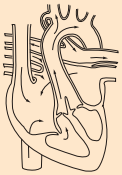
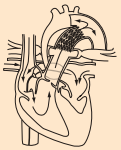
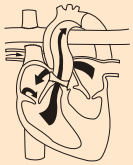
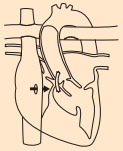
CARDIAC DEFECTS AND LESIONS

Septal Cardiac Lesion	Description	Surgical Repair	Additional Considerations
Atrial Septal Defect (ASD) 	• Opening in the atrial septal wall allows communication between RA and LA • Association with additional lesions and syndromes	• Atrial patch closure with pericardium tissue or polytetrafluoroethylene (PTFE) • Percutaneous transcatheter closure available using occlusive devices	• Types: Ostium secundum (most common), ostium primum (near AV valve) and sinus venosus (SVC or IVC to intratrial communication)
Ventricular Septal Defect (VSD) 	• Opening in the ventricular septal wall allowing communication between RV and LV • Association with additional lesions and syndromes	• Ventricular patch closure with PTFE or Dacron via transatrial approach or ventriculotomy • Percutaneous transcatheter closure available using occlusive devices	• Single or multiple lesions • Types: perimembranous (most common), subarterial (beneath PV), inlet (involves AV valves), muscular (usually multiple lesions in muscular septum) • Post op considerations: PHTN and 3º block • Risk for Eisenmengers complex with untreated large VSD
Atrio-ventricular Septal Defect (AVSD) aka AV Canal (AVC) or Endocardial Cushion Defect 	• Defects in the atrial and ventricular septum above or below the AV valves • Can be complete or partial • Association with additional lesions and syndromes, particularly Trisomy 13 (Down syndrome)	• Single or double patch closure	• Classifications: Rastelli A, B, C based on anatomy of AV valve • Post-op considerations: PHTN, mitral insufficiency, cardiac conduction disturbances and dysrhythmias

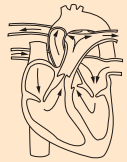
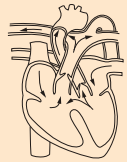
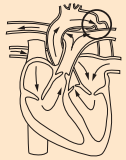
CARDIAC DEFECTS AND LESIONS [CONT]

Conotruncal Cardiac Lesion	Description	Surgical Repair	Additional Considerations
Transposition of the Great Arteries (TGA) aka Transposition of the Great Vessels (TGV) 	• Pulmonary and systemic circulations act in parallel versus series • Shunting vital for survival pre-op via PDA, ASD, or VSD • Ventricular to arterial (aorta and pulmonary) discordance • Association with additional lesions and syndromes	• Emergent atrial balloon septostomy (BAS) may be required prior to repair • Transection of Ao and PA for reattachment to appropriate ventricular chambers. LCA and RCA excised from PA (neoAo) for reattachment to Ao (neoPA) • ASD and or VSD patch closure	• Types: D-TGA, L-TGA (anatomic ventricular inversion with vessels transposed), CCTGA (see below) • Post-op considerations: Low cardiac output syndrome (LCOS), PHTN in D-TGA • Mustard or Senning repairs common before mid 1980's • Congenitally Corrected Transposition of the Great Arteries (CCTGA) has atrial to ventricular and ventricular to arterial discordance
Truncus Arteriosus (TA) 	• Common great arterial vessel receives blood from both ventricles and supplies blood to Ao, PA and coronary arteries, VSD • Association with additional lesions and syndromes	• Separation of branch PAs from truncal artery. RV-PA valved homograft conduit constructed. Ao reconstructed with sutures in an end-to-end fashion generally without additional materials • VSD patch closure (Dacron)	• Classification I-IV based upon origin of PA • Care during surgical procedure to not affect coronary arteries • Post-op considerations/ risks: PHTN, RV dysfunction, RVOTO
Single Ventricle Lesions	**Description**	**Surgical Repair**	**Additional Considerations**
Hypoplastic Left Heart Syndrome (HLHS) 	• Can be anatomical or physiologic (See single ventricle physiology listed in previous section) • Complete mixing of systemic and pulmonary circulations at atrial, ventricular or both levels • Association with additional lesions and syndromes	• Norwood, modified Norwood, Sano or Damus-Kaye-Stansel (DKS) can be performed with CPB • Hybrid performed off CPB with cardiac surgeon and cardiac interventionalist	• Balancing parallel circulation (Qp:Qs) goal of pre-/post-operative management • Attention to adequate systemic circulation to maintain perfusion to other organ systems

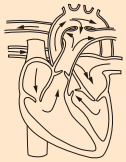
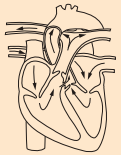
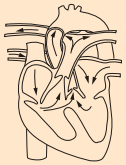
CARDIAC DEFECTS AND LESIONS [CONT]

Single Ventricle Lesions	Description	Surgical Repair	Additional Considerations
Stage 1 Palliation (S1P): Norwood, Damus-Kaye-Stansel or Sano Procedures 	• Norwood to provide unobstructed systemic outflow and balanced systemic to pulmonary circulation • DKS for SV with subaortic stenosis and distal arch obstruction • Sano ↓ incidence of ventricular ischemia preventing aortopulmonary runoff results in ↑ coronary and systemic pressures	• Norwood: PDA ligation, BTS right subclavian to MPA and ASD created • DKS: PDA ligation, BTS MPA to Ao, ASD created • Sano: PDA ligation, RV to MPA conduit via ventriculotomy, ASD created, and Ao arch anastomosis to proximal MPA	• Post-op objectives: Q_p:Q_s = 1:1 ratio; A-vO_2 difference = 0.25; O_2ER <50%, ScvO_2 ≥50% • Post-op considerations/ risks: PHTN, LCOS, hypoxemia, excessive SaO_2, necrotizing enterocolitis (NEC) • Thrombosis formation in SVC can impact further stages
Hybrid Procedure 	• Option to stage 1 palliation with cardiac surgeon and cardiac interventionalist without CPB	• Bilateral PA banding, with transpulmonary ductal stenting for arterial patency • Ao arch reconstruction performed later	• May not be appropriate in stenosis for retrograde Ao arch • Can be performed in premature and low birth weight neonates
Stage 2 Palliation: Bi-directional Glenn or Hemi-Fontan 	• Stage 2 palliation single ventricle, cavo-pulmonary shunt for volume unloading of single systemic ventricle, reduce wall stress and AV valve insufficiency	• Bi-directional Glenn: anastomosis of SVC to ipsilateral pulmonary artery and takedown of PA band (may be performed off CPB) • Hemi-Fontan: SVC connected to PA without disconnection from RA and patch closure at atrial end of SVC	• Risk of SVC syndrome post-operative • Veno-venous collaterals develop with increase SVC pressures, AV malformations lead to intrapulmonary shunting
Stage 3 Palliation: Fontan Procedure 	• Final stage palliation for single ventricle physiology	• Lateral tunnel: intra-atrial baffle connecting IVC to PA, with or without fenestration to RA • Extracardiac: graft conduit connects IVC to PA, with or without fenestration to RA	• Risk of protein-losing enteropathy (PLE), CHF, hypoxemia, dysrhythmias and thromboembolism post-operatively

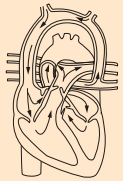
CARDIAC DEFECTS AND LESIONS [CONT]

Single Ventricle Lesions	Description	Surgical Repair	Additional Considerations
Pulmonary Atresia with Intact Ventricular Septum (PA/IVS) Pulmonary Stenosis (PS) 	• PA/IVS: atretic pulmonary valve with normal RV or hypoplastic RV (HRHS) • Complete membranous RV outflow tract obstruction (RVOTO) • PDA/ASD present and abnormal tricuspid valve (TV) • Ebstein-like variant hypertrophied RV • PS at PV with normal TV and RV • Association with additional lesions and syndromes	• Repair either bi- or uni-ventricular based upon RV. Placement systemic-to-pulmonary artery (BT) shunt as neonate • Bi-ventricular: PDA ligation, pulmonary valvotomy, staged ASD closure, removal BT shunt • Uni-ventricular: bi-directional cavo-pulmonary anastomosis (Glenn) • PS as an independent lesion can be repaired with balloon valvotomy percutaneously or surgically	• HRHS staged repair similar to HLHS • Post-op consideration/ risks: shunt thrombosis, TV growth, coil embolization of aortopulmonary collaterals
Aortic Cardiac Lesion	**Description**	**Surgical Repair**	**Additional Considerations**
Interrupted Aortic Arch (IAA) 	• Interruption of ascending to descending Ao. PDA necessary and commonly VSD present • Association with additional lesions and syndromes	• Ao end-to-end anastomosis, PDA ligation and VSD patch closure • Damus-Kaye-Stansel (DKS) based on size and location of VSD • Modified Norwood if two ventricle repair unobtainable	• Classifications: Type A, B, C based on location with L-subclavian and carotid artery • Post-op consideration/ risks: stenosis of anastomosed area, arch obstruction, L-bronchial compression
Coarctation of the Aorta (Ao) 	• Aortic obstruction from narrowing of the Ao at various levels. Ao arch hypoplasia common • Association with additional lesions and syndromes	• Removal of stenotic area and end-to-end aortic anastomosis, Dacron, PTFE or Gortex patch augmentation; subclavian flap aortoplasty; extended resection with primary anastomosis via throacotomy	• Post-op consideration/ risks: re-coarctation can occur, phrenic nerve damage, thoracic duct injury (chylothorax)

CARDIAC DEFECTS AND LESIONS [CONT]

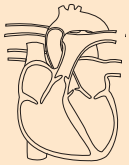
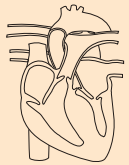
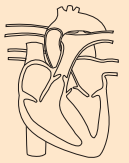
Abnormal Vessel Connection or Shunt Lesion	Description	Surgical Repair	Additional Considerations
Patent Ductus Arteriosus (PDA) 	• Persistent patency of fetal ductus arteriosus. Communication between MPA and descending Ao • Association with additional lesions and syndromes • If additional lesions, may be require PDA to be kept open with PGE_1 until complete repair performed	• Ductal ligation via thoracotomy or thorascopy • Percutaneous device closure (older children)	• At risk for PHTN post-op when associated with AP window • Post-op consideration/ risks: thoracic duct injury (chylothorax), phrenic nerve damage
Aorticopulmonary (AP) Window 	• Communication Ao-to-PA usually at the level of the ascending Ao • Association with additional lesions and syndromes	• Transaortic closure with prosthetic patch or pericardium	• Classification: Types I-III depending on location of the window to the Ao • At risk for PHTN post-op when associated with PDA
Tetralogy of Fallot (TOF) (with or without PA) 	• PA stenosis, VSD, Ao deviation, RV hypertrophy • CXR with "boot-shaped" cardiac image • Association with additional lesions and syndromes	• BT shunt palliative to improve pulmonary blood flow (PBF) • Transannular patch of RV-MPA and patch VSD closure • TOF with PA requires valved conduit (RV-MPA) in neonatal period	• Post-op consideration/ risks: volume loading of heart, dysrhythmias • Repair in neonate increase incidence of valved conduits and reoperation

CARDIAC DEFECTS AND LESIONS [CONT]

Abnormal Vessel Connection or Shunt Lesion	Description	Surgical Repair	Additional Considerations
Total Anomalous Pulmonary Venous Return (TAPVR) 	• Pulmonary veins (PV) do not connect to LA; can be complete (TAPVR) or partial (PAPVR), obstructed or unobstructed • Lesions can be supra-cardiac (PV to innominate vein via vertical vein or SVC; intra-cardiac (PV to coronary sinus or RA); infra-cardiac (common PV to portal system to IVC via ductus venosus); mixed (each four PV can drain anomalously) • Association with additional lesions and syndromes particularly heterotaxy	• Anastomosis of pulmonary veins to LA in all types • Supra-cardiac: vertical vein ligated; intra-cardiac: patch at foramen ovale and coronary sinus; infra-cardiac: descending vein ligated	• Classifications: supra-, intra-, infra-cardiac, mixed depending on location of pulmonary venous return • Obstructed TAPVR is a medical emergency, may require ECMO • Post-op consideration/ risks: PHTN and crisis, LCOS, pulmonary venous obstruction, supraventricular dysrhythmias
Inflammatory States and Myopathies	**Description**	**Surgical Repair or Treatment**	**Additional Considerations**
Congestive Heart Failure (CHF)	• Clinical syndrome of increase (preload), structural and/or functional myocardial abnormalities, neurohormonal activation	• Diuretics, digoxin, inotropes, ACE inhibitors, angiotensin II receptor and beta blockers, mechanical assist device • Surgical repair for cardiac lesions, heart transplantation if irreversible	• Causes: Structural heart disease in unrepaired lesions, LCOS post-op, dysrhythmias, inflammatory process, non-cardiac causes (PHTN, syndromes, toxemias, infections)
Pericarditis	• Inflammation of the pericardial sac • Resulting from an infectious process (bacterial, viral, tuberculosis) or non-infectious	• Dependent upon causative factor • Supportive therapy • Pericardiocentesis for pericardial effusion	• Can be chronic (>3 months), recurrent (after therapy stopped) or constrictive (thickened pericarditis) • Development of pericardial effusion can develop into pericardial tamponade
Infective Endocarditis (IE)	• Infectious process of endocardial surface, bacterial or fungal	• Anti-microbial therapy and IE prophylaxis	• High risks: prosthetic valves and unrepaired cyanotic CHD

ANATOMY AND PHYSIOLOGY

CARDIAC DEFECTS AND LESIONS [CONT]

Inflammatory States and Myopathies	Description	Surgical Repair or Treatment	Additional Considerations
Myocarditis	• Inflammatory process of myocardium (usually viral) • Suspected with unexplained congestive heart failure (CHF) and ventricular tachycardia (VT)	• Positive inotropes support C.O.	• Apoptosis and DCM • Preventable with childhood vaccinations
Dilated (DCM) 	• Ventricular hypertrophy and decrease systolic function • Association with syndromes and inflammatory processes	• Medical management: inotrope and vasodilator support, catecholamine IV, digoxin, diuretics, β-blockers, β-type natriuretic peptides, anti-thrombolytics, anti-dysrrhythmics, VAD and cardiac resynchronization • Surgical management: cardiac transplantation	• Myocyte death from apoptosis • Dilation results in increase wall stress and mismatch of myocardial O_2 supply and demand
Hypertrophic (HCM) 	• Thickened, non-dilated LV with mild or without LV outflow obstruction • Genetic cardiac disorder • Association with syndromes	• Medical management: β-adrenergic blocking agents, β-blockers, diuretics and/or VAD • Surgical management: septal myectomy or cardiac transplantation • Other: ICDs in high-risk HCM, dual chamber pacing (refractory to drugs, alcohol septal ablation)	• Common cause of sudden death in young children/adolescents • Common symptoms: exertional dyspnea, fatigue, syncope, palpitations, angina
Restrictive (RCM) 	• Restrictive ventricular filling and decreased diastolic volume (uni- or bi- ventricular), normal systolic function and ventricular wall thickness • Association with syndromes	• Medical management: supportive to symptoms • Surgical management: cardiac transplantation	• Clinical presentation commonly pulmonary in origin • Frequently familial

BTS: Blalock-Taussig Shunt
CBP: cardiopulmonary bypass
C.O.: cardiac output
CXR: chest radiograph
ECMO: extracorporeal membrane oxygenation
ICD: internal cardioversion defibrillator
LCOS: low cardiac output syndrome
PHTN: pulmonary hypertension
VAD: ventricular assist device

NORMAL INTRA-CARDIAC PRESSURES

Site	Newborn (mmHg)	Child (mmHg)	L→R Shunt	R→L Shunt	Adult (mmHg)
	(non-cardiac)	(non-cardiac)	(cardiac lesion)	(cardiac lesion)	(non-cardiac)
Right atrium (RAP) mean	0-4	2-6	5	0-2	0-8
Right ventricle (RVP)	65-80 / 0-6	15-23 / 3-7	25 / 5	110 / 8	25-30 / 0-8
Pulmonary artery (PA)	65-80 / 35-50	15-23 / 10-16	18-20 /8-10	20 / 8	15-30 / 6-12
Pulmonary wedge (PW) – mean	6-9	8-11	2	3-8	4-12
Left atrium (LA) mean	0-6	5-10	8	2	4-12
Left ventricle (LV)	65-80 / 0-6	90-110 / 7-9	100 / 8	96 / 8	100-130 / 4-12
Aorta (Ao)	65-80 / 45-60	90-110 / 65-75	140 / 80	96 / 8	100-130 / 60-90
CVP	0-8	2-6	5	0-2	0-8

Low Cardiac Output Syndrome (LCOS)

LCOS occurs within the first 24-36 hours generally following congenital heart surgery, especially in post-op Norwood or DKS repair for HLHS; and the arterial switch operation for TGA. Symptoms include: tachycardia, hypotension, oliguria, metabolic acidosis, A-vO_2 difference >40-50%, changes in neurological assessment, valve regurgitation and increased Q_p:Q_s ratio. Monitoring and maintaining SVC saturations ($ScvO_2$) >50 to 60% depending upon lesion and lactate measurements have been helpful in predicting risk for anaerobic metabolism.

Non-specific causes

Inflammation

Ischemia-reperfusion injury

Atrio-ventricular dysynchrony
- Complete heart block (3° block)

Brady- or tachy-dysrhythmias
- Junctional ectopic tachycardia (JET)

Tamponade

Hypothyroidism

Lesion-specific causes

Changes in loading conditions
- Decrease preload and increase afterload (systemic AV valve repair for regurgitation)
- Increase volume load (systemic-PA shunt, pulmonary regurgitation)

Ventriculotomy

Coronary re-implantation (Ross procedure, TGA repair)

Dennervation after heart transplant

Pulmonary Arterial Hypertension (PAH)

PAH is defined as mean pulmonary artery pressures >25mmHg at rest or >30mmHg with activity and results from an increase in pulmonary flow or resistance. PAH can be acute or chronic, reversible or irreversible.

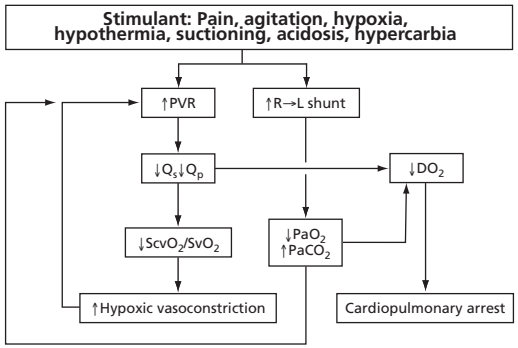
PAH Causes

- Cardiac disease (congenital or left-sided obstruction/dysfunction)
- Lung disease
- Thromboembolic disease (pulmonary vascular obstruction)
- Connective tissue, granulomatous diseases
- Idiopathic
- Other (infection, overdose, toxins, liver disease)

INTERVENTIONS TO MANIPULATE PVR

Increase PVR	Decrease PVR
Hypoxia	Oxygen
Hypercarbia/acidosis	Hypocarbia/alkalosis
Hyperinflation	Normal FRC (functional residual capacity)
High hematocrit (polycythemia)	Low hematocrit (anemia)
Atelectasis	Nitric oxide
	Prostacyclin
	Inhaled anesthetics

PULMONARY HYPERTENSION IN POST-OP CARDIAC SURGERY



Adapted from Critical Heart Disease In Infants And Children 2nd Ed.

Syndromes*

There are at least 4,500 syndromes or disorders identified in pediatrics. Although for the most part, these syndromes or disorders can be considered rare, between 30-45% of patients admitted to a PICU have an associated syndrome. Many patients admitted with cardiac disease or defect often have an associated syndrome. An abbreviated list of more commonly seen syndromes or disorders in congenital heart disease can be found in the reference section.

Adult Congenital Heart Disease (ACHD)

Adults living with congenital heart disease equal or exceed infants/children with congenital cardiac lesions and continue to increase worldwide. Adults who have undergone previous repair(s) in infancy/childhood, or who have unrepaired lesions, often experience long-term complications, residual abnormalities and may require re-operation. Eisenmenger's syndrome is of concern in unrepaired left-to-right shunts, as survival decreases drastically beyond adolescent years. If repair is not an option, heart-lung transplantation may be considered.

Possible complications:

- Dysrhythmias (ventricular dysfunction, sudden cardiac death)
- Vascular lesions (aneurysm formations)
- Valvular disease (regurgitation, insufficiency, stenosis)
- Residual or unrepaired shunts (left-to-right)
- Heart failure (right or left, pulmonary hypertension (PHTN), outflow tract obstruction, left-heart lesions)

UCLA Adult Congenital Heart Disease Classification

Class 1	Asymptomatic all activity levels
Class 2	Symptomatic but do not affect activities
Class 3	Symptomatic, affects activities
Class 4	Symptomatic with activities and at rest

**See the reference section for list of syndromes commonly seen in pediatrics with congenital heart disease.*

Extracorporeal Circulatory Devices

Extra-Corporeal Membrane Oxygenation (ECMO)

ECMO is utilized for neonates, infants and children in severe refractory cardiac and/or pulmonary failure. It is commonly used for low cardiac output syndrome in pre- and post-operative repair of congenital cardiac defects, or when unable to be weaned from cardiopulmonary bypass. ECMO is a proven therapy in neonates and premature infants with respiratory disease. It can be performed as a veno-arterial (VA) or veno-venous (VV) circuit.

DIFFERENCES BETWEEN VV AND VA ECMO

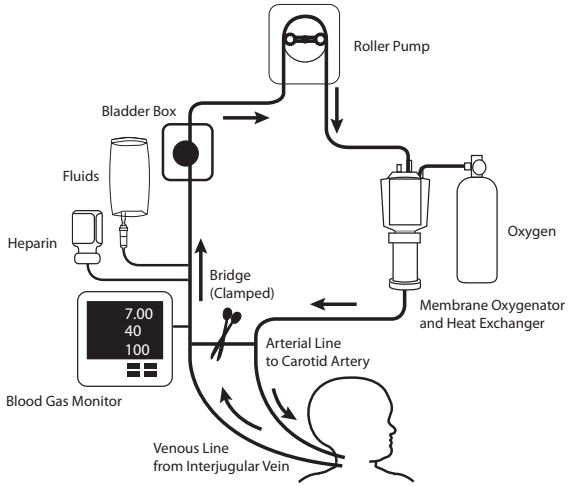
Hemodynamic Differences	VV ECMO	VA ECMO
Systemic perfusion	Cardiac output only	Circuit flow and cardiac output
Arterial BP	Full pulse contour	Dampened pulse contour
CVP	Accurate guide of volume status	Not useful
PA pressure	No effect by flow	Decreased flow in proportion to ECC* flow
Right-to-Left shunt	No effect	Mixed venous in perfusate blood
Left-to-Right shunt, PDA+	No effect on ECC* flow, usual PDA physiology	Pulmonary hyperperfusion, may require increase flow
Brain perfusion, selective right arm	No occurrence	Occurs
Gas Exchange Differences	**VV ECMO**	**VA ECMO**
Typical blood flow (full gas exchange)	100-120 mL/Kg/min	80-100 mL/Kg/min
Arterial oxygenation	80-95% on maximum flow	Saturation controlled by ECC* flow
CO_2 removal	Dependent on sweep gas, membrane lung size	Dependent on sweep gas, membrane lung size
Oxygenator	0.6 or 0.8	0.4 or 0.6
Decrease in vent settings initially	Slowly	Rapid

*ECC = extracorporeal circuit

+PDA = patent ductus arteriosus

TYPICAL ECMO FLOW AND GAS EXCHANGE REQUIREMENTS

Weight (Kg)	Membrane Surface Area (m^2)	Rated Flow (mL/min)	Gas Exchange (mL/min)
3-10 Kg	0.4 m^2	500 mL/min	30 mL/min
	0.8 m^2	1,000 mL/min	60 mL/min
	1.5 m^2	2,000 mL/min	120 mL/min
10-30 Kg	2.5 m^2	3,000 mL/min	180 mL/min
	3.5 m^2	4,000 mL/min	240 mL/min
>30 Kg	4.5 m^2	5,000 mL/min	300 mL/min



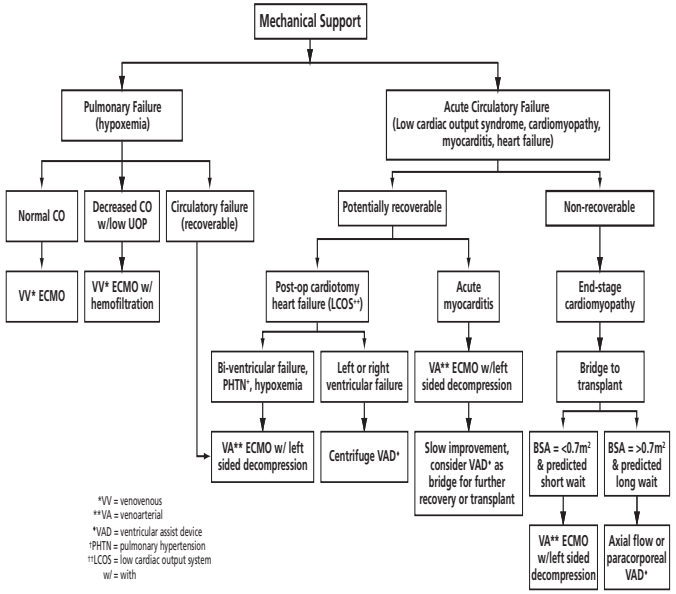
Mechanical Circulatory Support

Mechanical circulatory support devices have been used in children since the first successful use of the adult heart-lung machine in the 1950's for an ASD repair. These devices have been important in support of cardiovascular compromise during the peri-operative period, reversible myocardial failure and/or as a bridge to transplantation in congenital heart disease. Depending upon the device, it can be utilized either short or long-term.

MECHANICAL CIRCULATORY SUPPORT DEVICES (MCSD) & VENTRICULAR ASSIST DEVICES (VAD)

Support Device	Mechanical Type	Support	Age Limitations
ECMO	Extracorporeal, centrifugal	Short-term	All ages
Bio-pump	Extracorporeal, centrifugal	Short-term	All ages
Intra-aortic Balloon pump (IABP)	Extracorporeal, counterpulsation	Short-term	Older child
Abiomed BVS 5000	Extracorporeal, pneumatic	Short-term	Older child
Thoratec	Paracorporeal, pneumatic	Long-term	Older child
Heartmate V-E	Implantable, electric	Long-term	Older child
Medos HIA-VAD	Paracorporeal, pneumatic	Long-term	All ages
Berlin Heart EXCOR	Paracorporeal, pneumatic	Long-term	All ages
DeBakey VAD Child	Implantable, axial flow	Long-term	5-12 years

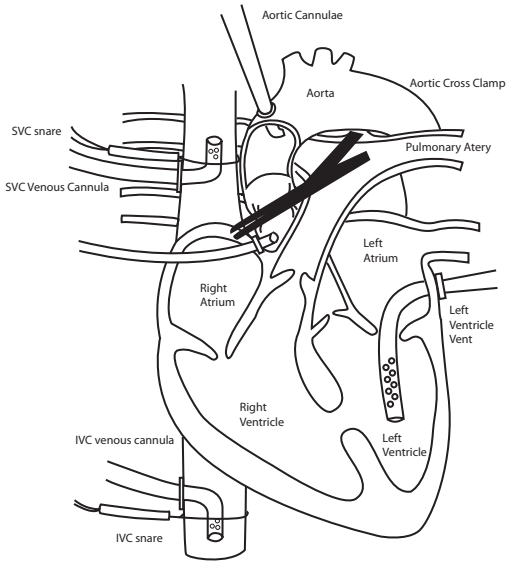
MECHANICAL SUPPORT ALGORITHM



Cardiopulmonary Bypass (CPB)

Cardiopulmonary bypass in pediatrics can have profound physiological effects, especially in neonates and infants. The degree of hypothermia, hemodilution and perfusion flow rates can have extreme effects in these patients. Many organ systems, such as the brain, lungs, coagulation and endocrine may still be developing in the neonate. Since infants have higher metabolic rates, they require a higher bypass flow per body surface area. Cardiac anatomical features, (aortopulomonary connection, interrupted aortic arch and intracardiac shunts), can require alterations in bypass cannulation, hypothermia and circulatory arrest. Arterial cannulation is generally in the ascending aorta. However, in interrupted aortic arch repair, the arterial cannula can be placed in the innominate artery and ductus arteriosus.

CARDIOPULMONARY BYPASS CANNULATION



Oxygenator

Membrane oxygenators are used that consist of a micro-porous hollow fibers (3-5 mm) that allow for gas exchange over a small surface area and have low prime volumes. This helps to eliminate how blood is exposed to the artificial surface, which helps to decrease hemolysis, platelet aggregation and release of the inflammatory mediators. In pediatrics, the oxygenators must be able to provide a wide range of temperature (10°C - 40°C), flow (0-200 mL/min), hematocrits (15%- 40%), line pressures and gas flow because of the wide range of weights and ages in congenital cardiac surgery.

COMMONLY USED PEDIATRIC CPB OXYGENATORS

Oxygenator Type	Prime (30)	Flow (LPM)	Surface Area (m^2)
Medtronic Minimax	149	2.3	0.80 m^2
Sorin Lilliput I	60	0.8	0.34 m^2
Sorin Lilliput II	105	2.3	0.60 m^2
Terumo BabyRx	43	1.5	0.50 m^2
Terumo Sx10	135	4.0	1.00 m^2
Terumo Rx10	135	5.0	1.50 m^2

WHOLE BODY VO_2 BASED UPON BODY TEMPERATURE

Temperature (°C)	Oxygen Consumption (%)
37°C	100%
32°C	60%
30°C	50%
28°C	40%
25°C	25-30%
20°C	20%
10°C	10%

Pump

The two types of pump are roller head or centrifugal. Although the roller head is the most widely used, new advances in the centrifugal have addressed the issues of requiring higher priming volumes. Roller pump CPB machines use two roller head pumps, oriented 180 degrees from each other. Blood is displaced in a forward, continuous and non-pulsatile manner. The second pump acts as a valve and minimizes backward flow. Centrifugal pumps entrap blood via a vortex action using spinning curved blades. This minimizes trauma to the red blood cells and can be used in pulsatile flow.

CARDIOPULMONARY BYPASS PUMPS

Pump Type	Pro	Con
Roller head	• Delivers precise volume at low flow (<0.500 L/min) • Promotes forward flow when set at "just-occlusive" • Reduces hemolysis and trauma to blood components	• Roller pumps may be in fixed position at base of machine
Centrifugal	• Non-occlusive • Mobile • Less traumatic on blood components • Reduces flow if obstruction of the outlet occurs	• May have higher priming volumes • May be less accurate at low flow rates (<0.500 L/min)

PUMP FLOW RATES

Patient weight (Kg)	Pump flow rate (mL/Kg/min)
0-7 Kg	120-200 mL/Kg/min
7-10 Kg	100-175 mL/Kg/min
10-30 Kg	80-120 mL/Kg/min
30-50 Kg	75-100 mL/Kg/min
>50Kg	50-75 mL/Kg/min

In-Line Monitoring

In-line continuous monitoring available on CPB machines includes pH, pO_2, pCO_2, K^+, SaO_2, BE and HCO_3^-.

BLOOD VOLUME PER WEIGHT

Patient weight (Kg)	Blood volume (mL/Kg)
<10 Kg	85 mL/Kg
11-20 Kg	80 mL/Kg
21-30 Kg	75 mL/Kg
31-40 Kg	70 mL/Kg
>40 Kg	65 mL/Kg

Tubing

Heparin bonded tubing has been useful in improving the blood-surface interface, reducing anticoagulation in infants undergoing CPB surgery and limiting the degree of inflammatory response. Bolus doses of heparin at the initiation of CPB can range 300-400units/Kg. ACTs (activated clotting times in seconds) are frequently followed during bypass time and after discontinuation of bypass. ACTS >350-400 sec is generally an accepted range, however can differ from institutional guidelines and if non-heparin bonded tubing is used.

CPB TUBING VOLUME & MAXIMAL FLOW RATES

Tubing size	Arterial (mL/ft)	Venous Flow Rate (mL/min)	Internal Diameter (ID) Return Rate (mL/min)
1/8	2.4 mL/min	<450 mL/min	250-300 mL/min
3/16	5.4 mL/min	<1,300 mL/min	50-650 mL/min
1/4	9.6 mL/min	<3,000 mL/min	1,200 – 1,600 mL/min
3/8	21.6 mL/min	>5,000 mL/min	4,000 – 4,500 mL/min
1/2	38.4 mL/min	>5,000 mL/min	>5,000 mL/min

TUBING INTERNAL DIAMETER (ID) SELECTION BASED ON WEIGHT

Patient Weight (Kg)	Arterial ID	Boot ID	Venous ID
0-3 Kg	1/8 – 3/16	3/16	3/16
4-7 Kg	3/16	1/4	3/16
8-12 Kg	3/16	1/4	1/4
13-17 Kg	1/4	1/4	1/4
18-27 Kg	1/4	3/8	1/4
27-35 Kg	1/4	3/8	3/8
>35 Kg	3/8	1/2	3/8

The thymus is removed to allow for better visualization of the operative field and visualization of PDA and/or previously placed shunts. When CPB is initiated, blood is drained from the heart. Venous cannulae (at the SVC and IVC), are "snared" (see diagram on page 43) to compress the vessel around the cannulae and prevent air from entering the circuit. Venous drainage is dependent upon the height difference between patient and circuit, venous cannula internal diameter and vacuum (if used). Elevated arterial cannula pressure indicates malpositioned or kinking of the arterial cannula. The heat exchanger can be used to cool the patient's core temperature to desired degree. All venous drainage is directed into the oxygenator. Once on CPB, ventilatory support is stopped and the lungs are collapsed.

Modified Ultrafiltration

Modified ultrafiltration is often employed to remove excess free water, cytokines and promote hemoconcentration of blood. Blood is directed from the aortic cannula to the hemofilter and heat exchanger. Once the blood has been hemoconcentrated, it is returned via the venous reservoir. This helps to decrease the need for RBC transfusion, improve cardiac function post-bypass and helps to reduce the incidence of post-operative pulmonary hypertension.

CPB ORGAN SYSTEM EFFECTS AND RELATED CAUSES

Organ system	Effect	Related Causes
Heart	• Decreased metabolic needs with cessation of mechanical and electrical activity	• Hypothermia reduces whole body oxygen consumption
	• Cessation of contractile and electrical activity	• Cardioplegia offers protection via depolarization or hyperpolarization of membranes and mechanical cardiac arrest
	• Ischemic - reperfusion injury	• Myocardial edema leads to decreased ventricular compliance, increased ventricular stiffness and diastolic dysfunction
	• Depolarization arrest decreases metabolic demands of myocytes	• Hyperkalemia from potassium depolarization during cardioplegia
Brain	• Neuronal loss following cerebral ischemia	• Liberation of inflammatory cytokines (IL – 1β and TNF – α)
	• Reperfusion injury following hypoxia – ischemia	• Resumed capillary blood flow with inflammatory mediators, activated and primed leukocytes. • Neurophils exhibit oxidative burst and influx of cytosolic calcium into cells
	• Dysfunctional cerebral autoregulation	• Post-operative hypoxemia from prolonged deep hypothermic circulatory arrest (DHCA)
Endocrine	• Elevated stress hormones: epinephrine/ norepinephrine at initiation of CPB	• Cardiopulmonary bypass • Hypothermia
	• Cortisol increases at initiation of CPB and for 24 hours post-bypass. (Levels may decrease during actual CPB time) • Vasopressin increases and may remain elevated up to 72 hrs post-bypass	• Hemodilution may cause the decrease of circulating hormones during CPB
	• Decrease in insulin and peripheral response to insulin	• Deep hypothermic CPB due to decrease insulin effect and decease metabolic rate
Renal	• Decrease in renal blood flow and glomerular filtration rate • Oliguria which gradually improves 24-48 hours post-bypass	• Surgical stress • Altered renal blood flow or redistribution of blood flow • Elevated stress hormones
	• Fluid retention	• Activation of renin-angiotensin system leads to increased aldosterone production
Pulmonary	• Diffuse endothelial injury resulting in increased capillary permeability, pulmonary edema, atelectasis	• Leukocyte degranulation and complement activation
	• Decrease in compliance, functional residual capacity, ventilation/perfusion mismatch	• Activation of the inflammatory system
	• Pulmonary hypertension	
Hematological	• Higher flow rates and priming volumes cause more stress on blood cell components	• Priming volumes are more than total blood volumes in neonates/infants leading to dilution of RBCs, clotting factors and plasma proteins

Pulmonary

Pulmonary Function Tests Definitions

Total Lung Capacity (TLC):
maximal amount of air within the lung at maximal inspiration. (60–80 mL/KgL)

Vital Capacity (VC):
maximal amount of air that can be exhaled after a maximal inspiration. (30–40 mL/Kg infants; 45–55m/Kg adults)

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

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

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

Functional Residual Capacity (FRC):
amount of air remaining in the lungs at the end of normal expiration. (30 mL/Kg)

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

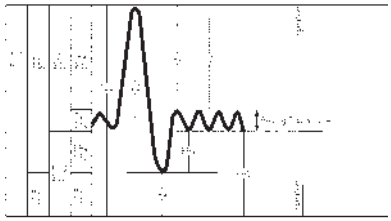
Tidal Volume (V_T):
volume of gas in and out of lungs per breath. (6–8 mL/Kg)

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.

NORMAL SPIROGRAM



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

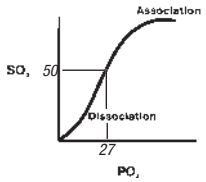
NORMAL BLOOD GAS VALUES

Component	Arterial	Venous	Capillary
pH	7.35 – 7.45	7.32 – 7.42	7.35 – 7.45
PO_2 (mmHg)	70 – 100	24 – 48	60 – 80
PCO_2 (mmHg)	35 – 45	38 – 52	35 – 45
HCO_3 (mEq/L)	19 – 25	19 – 25	19 – 25
TCO_2 mEq/L)	19 – 29	23 – 33	19 – 29
SO_2 (%)	90 – 95	40 – 70	90 – 95
Base excess/deficit (mEq/L)	-2 to +2	-2 to +2	-2 to +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-binding 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 molecule.

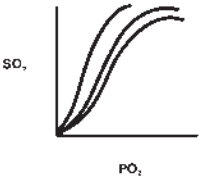
NORMAL OXYHEMOGLOBIN DISSOCIATION CURVE



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, evaluate the diffusion of oxygen across the pulmonary capillary unit, and 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.
$PAO_2 = [(PB - PH_2O) \times FiO_2] - PaCO_2 / 0.8$

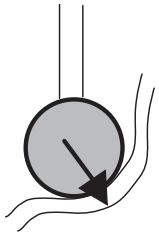
Alveolar–arterial Oxygen Gradient (A–a Gradient or P(A–a)O_2)

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

$$(\text{Barometric Pressure} - \text{Water Vapor Pressure}) \times \text{Patient's } FiO_2 - \frac{PaCO_2}{0.8} - \text{Patient's } PaO_2$$

$$(760 - 47) \times 0.21 - \frac{40}{0.8} - 90$$

$$713 \times 0.21 - 50 - 90$$

$$99.73 - 90 = 9.73$$

A–a Gradient $\cong$ 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 a FiO_2 of 1.0. Venous admixture which produces a physiologic shunt can be determined when the patient is on a FiO_2 of < 1.0. Both determinations require pulmonary artery saturation values to complete the calculation.

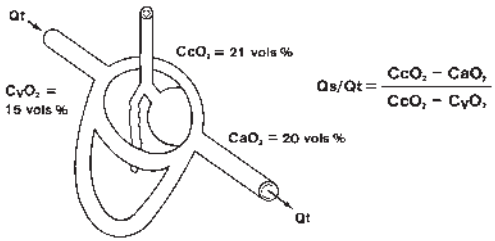
$$Qs/Qt = \frac{CcO_2 - CaO_2}{CcO_2 - CvO_2}$$

CcO_2 = Capillary oxygen content
$(1.38 \times Hgb \times 1) + (PAO_2 \times 0.0031)$

CaO_2 = Arterial oxygen content
$(1.38 \times Hgb \times SaO_2) + (PaO_2 \times 0.0031)$

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

QS / QT



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

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

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

Dead Space, Shunts and Ventilation/Perfusion Mismatch (V/Q)

Dead space:	airflow to alveolus, but no blood flow. Dead space leads to hypercarbia.
Shunt:	blood flow to the alveolus, but no air flow. Shunts lead to hypoxia.
Mismatch:	V/Q = 1 normally thus ventilation and perfusion are equal. When V/Q <1, decrease in PaO_2. If V/Q >1 increase in PaO_2.

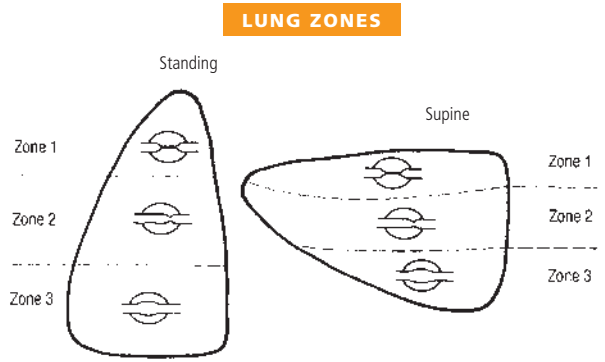
West Zone of the lungs

Lungs divided into three vertical zones and represents relationship between pressure in alveoli (P_A), pulmonary arteries (P_a) and pulmonary veins (P_v). It compares ventilation to perfusion.

Zone 1 generally is considered alveolar dead space and does not normally exist except if positive pressure ventilation is applied. Blood vessels are collapsed by alveolar pressure.

Zone 2 is above the level of the heart and has pulsatile flow. Flow initially obstructed at venous end of capillary bed until arterial pressure build-up in cardiac cycle exceeds alveolar pressure and blood flow resumes.

Zone 3 takes up the majority of the lungs and has continuous blood flow throughout the cardiac cycle.



Zones	Standing	Supine
Zone 1 (V/Q >1) $P_A > P_a > P_V$	Apex of lungs	Anterior aspect of lungs
Zone 2 (V/Q = 1) $P_a > P_A > P_V$	Middle of lungs	Middle aspect of lungs
Zone 3 (V/Q <1) $P_a > P_V > P_A$	Base of lungs	Posterior aspect of lungs

Mechanical Ventilation (MV) and Ventilatory Assist

Mechanical ventilation is used in the treatment of chronic and/or acute respiratory failure and ventilatory support during surgical procedures. Mechanical ventilation administers forced breaths with control of pressure, volume and flow. The control variables are related to the elastic and resistive forces to allow for gas flow in and out of the lungs.

$$\text{Pressure} = \frac{\text{Volume}}{\text{Compliance}}$$

$$\text{Flow} = \frac{\text{Pressure}}{\text{Resistance}}$$

$$\text{Volume} = \text{Pressure} \times \text{Compliance}$$

Pressure is related to the patient's compliance to the change in lung volume. It is affected by both elastic and resistive forces. Volume resistive forces include compliance of the lungs, airways and chest wall. Gas flow resistive forces include airway, ventilator tubing, and ETT.

VENTILATORY THERAPIES

Inhaled Gases/IV Medications	Description	Uses	Modes of Delivery	Adverse Effects
Isoflurane	• Anesthetic agent	• Pulmonary vasodilator for PHTN and bronchodilator • Systemic vasodilator • Refractory status epilepticus	• Inhaled gas in conjunction with mechanical ventilation	• Reversible choreiform movements with prolonged exposure
Nitric oxide (iNO)	• Selective pulmonary vasodilator	• Pulmonary hypertension (PHTN) • ARDS: V/Q mismatch, intrapulmonary shunting, hypoxemia	• Inhaled gas in conjunction with mechanical or high frequency ventilation	• Methemoglobinemia • Rebound PHTN when iNO discontinued • Coagulation abnormalities
Heliox	• Inert gas of helium and oxygen mixture. Helium: oxygen ratios of 80/20 or 70/30	• Reduce work of breathing (WOB) and expiratory wheezing in obstructive lung disease	• Nebulizer with MV or HFOV • Non-rebreather mask	• Helium can interfere with pneumotachometer on some ventilators
CO_2	• Inhaled gas via MV, permissive hypercarbia	• Reduce PVR in HLHS	• Inhaled gas in conjunction with MV	
Hypoxic gas mixtures	• Nitrogen dilution with oxygen to lower inspired FiO_2 <21%	• Used in HLHS to increase PVR	• Low-flow bleed into ventilator circuit	• Non-uniform delivery system • Additional organ hypoxia
Exogenous surfactant	• Synthetic recombinant of phospholipids and protein preparation • Animal lung (bovine, porcine)	• Treatment respiratory distress syndrome (RDS) premature infants • Meconium aspiration • ARDS / ALI • Surfactant deficiency syndromes	• Via endotracheal tube with or without MV	• Associated with ETT insertion and management • Pulmonary hemorrhage
Sildenafil	• Pulmonary vasodilator	• Decrease PVR in PHTN	• Intravenously	• Increase intrapulmonary shunting post CHD repair
Iloprost	• Selective pulmonary vasodilator, stable, Prostacyclin (PGI_2) analogue	• Pulmonary hypertension (PHTN) ARDS post CHD repair	• Conventional nebulizer • Used with iNO	
Hyperbaric chamber	• 100% O_2 at pressures >1 ATA (atmospheric absolute) • Hyperbaric oxygenation (HBO) increases O_2 dissolved in plasma	• Smoke inhalation, carbon monoxide poison • Crush injuries, compartment syndrome • Acute traumatic ischemia, necrotizing fasciitis • Arterial gas embolism (AGE)	• Hyperbaric chamber for invasive and non-invasive O_2 delivery	• Related to pressure/volume changes • Oxygen toxicity

VENTILATION MODES AND O_2 SUPPORT

Ventilation/O_2 Therapy	Description	Modes/Settings
Mechanical Ventilation (MV)	• Regulation of pressure, volume and flow to generate a forced, mechanical breath and displacement of gas in and out of the lungs	• IMV: intermittent mandatory ventilation or (SIMV) synchronized IMV • Controlled ventilation: AC (assist control), PC (pressure control), VC (volume control), PAV (proportional assist ventilation), APRV (airway pressure release ventilation) • Support ventilation: PS (pressure support), VS (volume support) • Airway pressure release ventilation (APRV) is timed cycle ventilation switching between two pressure levels in flow circuit • Hybrid modes: PRVC (pressure regulated volume control), VAPS (volume-assured pressure support) • Closed loop: Automode (VC/VS, PC/PS or PRVC/VS), ATC (automatic tube compensation) • PIP (positive inspiratory pressure); PEEP (positive end-expiratory pressure) • I:E ratio: (inspiratory : expiratory time)
High Frequency Ventilation (HFV)	• HFV extremely exceeds normal RR with low tidal volume (V_T) approximating for anatomical dead space • HPPV and HFJV, V_T >dead space with passive expiration • HFOV, V_T < dead space with active expiration, maximize V/Q matching without impairing C.O., unpairs O_2 and CO_2 elimination • High MAP recruit alveoli and maintain lung volume above FRC	• HPPVP: (high frequency positive pressure ventilation) 60-150 breaths/min • HFJV: (high frequency jet ventilation) 100-600 breaths/min • HFOV: (high frequency oscillation ventilation) 180-1,500 breaths/min or 3-25 Hz • HFPV: (high frequency percussion ventilation) • HFFIV: (high frequency flow interruption ventilation)
Non-Invasive Positive Pressure Ventilation (NPPV)	• Positive pressure ventilation administered via tight-fitting face mask, NC or mouthpiece	• CPAP: continuous positive airway pressure • IPAP: inspiratory positive pressure ventilation • EPAP: expiratory positive airway pressure • Bi-PAP: bi-level positive airway pressure (IPAP and EPAP)

VENTILATION MODES AND O_2 SUPPORT [CONT.]

Ventilation/O_2 Therapy	Description	Modes/Settings
High-Flow Nasal Cannula (HFNC)	• Deliver flow rates >6 - 15 L/min O_2 with humidification • Support for infants with respiratory distress, prematurity apnea and post-extubation respiratory support	• NCPAP: nasal continuous positive airway pressure • HHHFNC: heated, humidified, high-flow nasal cannula
Liquid Ventilation (Perfluorocarbon)	• Inert biochemical compound with high solubility to O_2 and CO_2 to increase gas exchange in the lungs, recruit alveoli and improve V/Q matching in the treatment of severe acute respiratory distress syndrome (ARDS) • Has high density with low surface tension	• PLV: partial liquid ventilation. Perfluorocarbon administered via side port ETT and eliminated via exhalation
Extra-corporeal Membrane Oxygenation (ECMO) or Extra-corporeal Life Support (ECLS)	• Used for temporary and prolonged support for heart and/or lung function • VV (venovenous) ECMO advantage is avoidance of cannulation and artery ligation, but does not support direct circulatory support • VA (venoarterial) ECMO advantage is partial or complete circulatory support and high O_2 delivery	• VV ECMO blood is drained via cannula in a vein and returned to a vein • VA ECMO blood is drained from a large vein and returned via an artery • Extra-corporeal lung assist (ECLA) or Extracorporeal CO_2 Removal (ECCOR) when used via extra-thoracic cannulation for respiratory support • Cardiopulmonary Support (CPS) or Extra-corporeal Cardiopulmonary Resuscitation (ECPR) when used with extra-thoracic cannulation for emergency cardiac support • Ventricular Assist Device (VAD) can be used with an oxygenator to support left (LVAD), right (RVAD) or bi-ventricular (Bi-VAD) support

Disease, Disorder, Diagnosis	Causes	Clinical Manifestations	Treatment/ Prevention	Other Considerations
Acute Lung Injury (ALI)	• Acute inflammatory reaction in the lung, epithelial-endothelial permeability with pulmonary edema • Via direct or indirect lung injury (pneumonia, aspiration, sepsis, trauma, massive transfusion	• Acute onset, bilateral pulmonary infiltrates, PA occlusion pressure ≤ 18 mmHg, PaO_2: FiO_2 ≤ 300 • Decrease lung compliance, increase V/Q mismatch, poor gas exchange)	• O_2 support with or without CMV or HFV adjunct therapies (inhaled vasodilators, corticosteroids, surfactant), prone positioning, nutritional support and hydration	• VILI (ventilator induced lung injury) can be dependent on V_T
Acute Respiratory Distress Syndrome (ARDS)	• More severe form of ALI • Via direct or indirect lung injury (inflammatory process i.e. tumor necrosis syndrome (TNF), burns, sepsis, trauma, massive transfusion)	• Acute onset, bilateral pulmonary infiltrates, PA occlusion pressure ≤ 18 mmHg, PaO_2: FiO_2 ≤ 200	• CMV or HFV, adjunct therapies (inhaled vasodilators, corticosteroids, surfactant), prone positioning, nutritional support and hydration	• ECMO may be used as a rescue therapy
Asthma	• Reactive airway disease • Increased airway resistance to inspiratory and expiratory gas flow	• Inflammatory-mediated edema, hyper-mucus secretions, airway plugging, bronchospasm, wheezing • Hypoxemia, V/Q mismatch, alveolar hyperinflation	• Supportive care with O_2, fluids, ß-agonists, bronchodilators, anticholinergics, corticosteroids, methylxanthines, heliox, ketamine, MV	• Frequent viral infections common • Status asthmaticus can lead to respiratory failure and MV
Bronchiolitis (upper respiratory tract inflammation)	• RSV common cause • *Mycoplasma* • Viral: parainfluenza, influenza, rhinovirus, adenovirus, paramyxovirus (measles)	• Acute respiratory tract infection • Inflammation, obstruction of peripheral airways	• Supportive care with O_2, fluids, ß-agonists, racemic epinephrine, steroids (inhaled and systemic), aerosolized recombinant human DNAse • MV, HFV, iNO NPPV, ECMO	• Prophylaxis with certain vaccinations

RESPIRATORY DISEASES, DISORDERS & DIAGNOSIS IN PEDIATRICS

Disease, Disorder, Diagnosis	Causes	Clinical Manifestations	Treatment/ Prevention	Other Considerations
Bronchopulmonary Dysplasia (BPD)	• Chronic lung disease seen in premature and very low birth weight (VLBW) infants of hyaline membrane disease after O_2 exposure and mechanical ventilation	• Respiratory distress syndrome • Post-neonatal period, unable to maintain SpO_2 ≥90% (non-congenital heart disease (CHD))	• Preventative: minimizing ventilatory support and over-inflation maintain FRC with optimal PEEP, optimize nutrition and diuretics, bronchodilators • Permissive hypercapnia to minimize barotraumas and volutrauma with MV	• Severe BPD rare in infants >32 weeks gestation • Seen in infants <28 weeks gestation or <1000 gms
Congenital Diaphragmatic Hernia (CDH)	• Pleuroperitoneal folds fail to close during gestation • Associated with syndromes	• Respiratory distress syndrome • Pulmonary hypoplasia	• Ventilatory support, pulmonary vasodilation with iNO • ECMO	• Usually left-sided and associated with ipsilateral lung hypoplasia • May be diagnosed *in-utero*
Coronavirus (SARS)	• Atypical pneumonia caused by coronavirus (SARS-CoV) • Four types; OC43, 229E, NL63, HKU1	• Incubation 5-7days • Asymptomatic, mild-moderate-severe respiratory illness • Febrile, CXR indicates pneumonia pattern, + SARS-CoV antibody	• Detected by RT-PCR or antibody analysis • Infection precautions	• Coronaviruses cause respiratory and gastrointestinal diseases in humans and domestic animals • Bi- or tri-phasic pattern
Croup (Laryngotracheo-bronchitis)	• Inflammation of subglottic area • Viral pathogens: adenovirus, *parainfluenzae*, influenzae A/B, RSV, herpes varicella, enteroviruses, *mycoplasma pneumoniae*	• Stridor, "barking cough", low-grade fever, respiratory distress, nasal flaring, lethargy, cyanosis • Viral prodrome: rhinorrhea, cough, sore throat (1-2 days)	• Humidified supplemental O_2, racemic epinephrine	• Typically affects ages 6 mo - 3 yrs • Differential diagnosis of congenital anomalies, additional inflammatory/ infectious processes, foreign body aspiration (FBA) or inhalation injuries
Cystic fibrosis (CF)	(See CF section on page 66)	(See CF section on page 66)	(See CF section on page 66)	(See CF section on page 66)

ANATOMY AND PHYSIOLOGY

Disease, Disorder, Diagnosis	Causes	Clinical Manifestations	Treatment/ Prevention	Other Considerations
Epiglottitis	• Associated *with Haemophilus influenzae B* • Other bacterial agents: *Strep, Staph,* and *Candida albicans*	• Rapid, progressive illness • Symptoms: fever, respiratory distress, stridor, drooling • Elevated leukocyte count • Lateral neck x-ray "thumbprint" appearance	• Hib-conjugated vaccine • Priority for airway protection • Broad spectrum antibiotics (sensitive to ß–lactamase producing pathogens) • Steroids IV	• High potential for complete airway obstruction • Avoid agitation until secure airway • Experienced personal to attempt ETT intubation • Rule out FBA and anaphylaxis
Foreign Body Aspiration (FBA)	• Aspiration of foreign items into airway passages	• Air obstruction and air trapping	• Cervical and CXR for diagnosis • Bronchoscopy for visualization and removal	• Left bronchus FBA more common in younger children, right bronchus FBA more common in older children
Human metapneumo virus (HMPV)	• Viral pathogen *Paramyxovirus*	• Acute respiratory tract infection of upper and lower tracts • Similar to RSV	• Supportive care with O_2, fluids, ß-agonists, racemic epinephrine, steroids (inhaled and systemic), ribaviran • MV, HFV, iNO NPPV, ECMO	• Currently a vaccine is not available • Often co-infected with other respiratory viruses or bacteria
H1N1 (Swine flu)	• Influenza A virus • Viral pathogen H1N1	• Severe flu symptoms (cough, fever, fatigue, chills, congestion, headache) with vomiting and diarrhea • Dyspnea, dizziness, confusion warrants emergent medical treatment	• Vaccination as preventative measure • Treatment with anti-viral agents: oseltamivir and zanamivir • Droplet nuclei isolation precautions, (N95 isolation mask recommended for caregivers)	• Should avoid contact with others for 24 hours after fever resolved, highly contagious • Is not contracted by eating pork • Household pets (cats and dogs) reported to contract H1N1)

RESPIRATORY DISEASES, DISORDERS & DIAGNOSIS IN PEDIATRICS

Disease, Disorder, Diagnosis	Causes	Clinical Manifestations	Treatment/ Prevention	Other Considerations
H5N1 (Avian flu)	• Influenza A virus • Variable forms (H5N1, H7N7, H9N2, H7N2, H7N3)	• Flu-like symptoms, with eye infections can develop conjunctivitis, acute respiratory distress syndrome (ARDS) and neurologic changes possible • Symptoms can develop within 7 days of exposure	• Vaccination as preventative measure • Treatment with anti-viral agent ribiviran • Prophylactic anti-viral agent during suspected outbreaks	• H5N1 most commonly seen in humans • Infection commonly following contamination from infected poultry or birds • Observe for anti-viral resistance
Inhalation injuries	• Inhalation from smoke and/or burns, hot liquid, toxic ingestions	• Facial burns, singed nasal and oral mucosa, wheezing, blistering, hoarseness, airway edema, pneumonia, airway obstruction • Risk for acute respiratory distress syndrome (ARDS)	• Cardiopulmonary supportive care • Sepsis prevention • Hyperbaric chamber for CO_2 poisoning	• Carbon monoxide (CO) poisoning leading cause of death in smoke inhalation • CO >200-300 affinity to O_2 • Pulse ox can't differentiate oxyhemoglobin and carboxyhemoglobin
Meconium aspiration syndrome (MAS)	• Aspiration of meconium *in-utero*	• Pneumonitis, respiratory distress syndrome (RDS) • primary pulmonary hypertension (PPHN)	• Initial treatment at delivery according to NRP (neonatal resuscitation program) • Supportive therapy for primary pulmonary hypertension (PPHN) (iNO or ECMO)	• Meconium can inactivate surfactant
Neonatal pneumonia	• Most common pathogen *Listeria monocytogenes* • Nosocomial from gram-negative enteric bacteria • *Chlamdyia trachomatis* from genital tract of infected mothers	• *L monocytogenes:* tachypnea, grunting, retractions, irritability, poor feeding, fever or hypothermia • *C trachomatis:* staccato cough, tachypnea, afebrile, crackles, bilateral diffuse infiltrates	• *L monocytogenes:* Broad spectrum antibiotics, supportive care • *C trachomatis:* erythromycin po, sulfonamides po if >2months	• *L monocytogenes:* generally not community-acquired • *C trachomatis:* diagnosed 2-19 weeks after birth

ANATOMY AND PHYSIOLOGY

Disease, Disorder, Diagnosis	Causes	Clinical Manifestations	Treatment/ Prevention	Other Considerations
Pertussis	• Viral pathogen *Bordetella pertussis or paratussis*	• Rhinorrhea, fever, coughing spasm to whooping cough (after 10 days)	• Antibiotic therapy, humidified O_2, supportive respiratory care with MV • Exchange transfusion if WBC >100 x 10^9/L	• Poor prognosis if MV required • Risk for encephalopathy and PHTN if not treated
Pneumonia (lower respiratory tract)	Common bacterial or viral pathogens *S pneumonia* (<4 years) *M pneumonia* (5-9 years) *C pneumonia* (10-16 years	*S pneumonia:* fever, malaise, rales, non-productive cough, decrease breath sounds, lobar consolidation, empyema *M pneumonia, C pneumonia:* infiltrates, tachypnea, cyanosis, retractions, lung hyperinflation, impaired cilia, mucus plugs	*S pneumonia:* antibiotic therapy, cephalosporins (2nd generation) *M pneumonia, C pneumonia:* • Macrolide agents • Tetracyclines (>8 years of age) • Fluoquinolones (>16 years of age)	• Neonatal pneumonia may have other causes
Pneumonia (atypical)	• Bacterial organisms:, *Chlamydia, Legionella pneumophilia, Bordetella pertussis* • Other viral pathogens	• *Legionella pneumophilia:* acute and severe symptoms of respiratory distress	• MV, supportive therapy, antimicrobial or antiviral agents	• See H1N1 and H5N1 for additional influenza pneumonias
Pneumocystis	• *Pneumocystis jirovci* (*p. carinii*)	• Pneumonia	• Trimethoprim-sulfamethozazole (TMP-SMZ) IV/PO • Corticosteroids • Prophylaxis (TMP-SMZ) at risk pts	• Common in HIV and immunodeficiency disorders
Primary pulmonary hypertension (PPHTN)	• PVR fails to decrease at birth • Meconium aspiration syndrome (MAS)	• Increased pulmonary pressure and hypoxemic respiratory failure	• Pulmonary vasodilation (iNO) and prostacycline • ECMO • Surfactant • Lung transplantation if persistent	

Disease, Disorder, Diagnosis	Causes	Clinical Manifestations	Treatment/ Prevention	Other Considerations
Respiratory Syncitial Virus (RSV)	• Pneumovirus (A and B) • Incubation ~5 days • Most common early winter-late spring • Transmission droplet nuclei	• *Upper respiratory:* fever, cough, rhinorrhea, wheezing • *Lower respiratory:* wheezing, retractions, flaring • *Severe infection:* hypoxemia, cyanosis, tachypnea-apnea, respiratory failure • *Other symptoms:* bronchiolitis, pneumonia, sepsis-like syndrome, myocardial injury (myocarditis)	• Diagnosis on history, physical exam • Viral detection (immuno-fluorescence or enzyme immunoassay) • Prevention of dehydration • Supplemental O_2, monitor O_2 status • Medications for consideration - Bronchodilators (clinical response) - Corticosteroids - Antibiotic therapy (prophylactic) - Antiviral agents i.e. Ribaviran (severe cases) - RSV prophylaxis (Palivizumab)	At risk: • Prematurity (33-35 weeks gestation) • Term infants born 6 months prior to "RSV season" • Chronic lung disease • Congenital heart disease • Neuromuscular disease • Immunologic deficiencies

CMV: conventional mechanical ventilation
CXR: chest radiograph
ECMO: extracorporeal membrane oxygenation
ETT: endotracheal tube
HFV: high frequency ventilation
iNO: inhaled nitric oxide
IV: intravenous
MV: mechanical ventilation
NPPV: Non-invasive positive pressure ventilation
po: by oral route

Cystic Fibrosis

Cystic fibrosis is an exocrine gland dysfunction with multi-system involvement. It is an inherited autosomal recessive trait with a 1:4 chance of receiving the recessive gene from both parents. The mutated gene exists on long arm of chromosome 7 and has protein product: cystic fibrosis transmembrane regulator (CFTR). Although CF is not primarily a pulmonary disease, many of the clinical manifestations and therapies surround optimizing pulmonary function. Newer advances in therapies and lung transplantation offer greater life expectancies than years ago. There is a focus to transfer care from pediatricians to adult practitioners. Many adults can experience a greater quality of life than before with CF. Carrier screening is recommended and reliable for siblings and family members of an individual with CF.

Diagnosis

- Quantitative sweat chloride test (pilocarpine iontophoresis)
  - Stimulation of the production of sweat, collection and analysis of sweat electrolytes.
  - Minimum 50 mg required, in two different samples
  - May be difficult to obtain in infants (do not have active sweat glands)
  - Sweat Cl- values

    Normal: <40 mEq/L. Mean 18 mEq/L
    CF values: >60 mEq/L
    Highly suggestive: 40-60 mEq/L
- Elevated immunoreactive trypsinogen (newborns)
- Genetic analysis

CYSTIC FIBROSIS (CF)

	Pulmonary	Gastrointestinal	Reproductive	Integumentary
Pathophysiology	• Increase mucus gland secretion and viscosity, bronchi and bronchiole obstruction, decrease gas exchange (O_2:CO_2)	• Decrease pancreatic HCO_3^- and Cl^- secretion, pancreatic fibrosis, biliary obstruction and fibrosis, decrease absorption fat soluble vitamins (A, D, E, K)	• Viscous cervical secretions inhibit fertility • Vas deferens blockage or abnormal development	• Elevated sweat electrolytes (Na^{++} and Cl^-)
Diagnosis	• Family history, CXR, pulmonary function tests (PFT)	• Stool samples, dietary intake record, enzyme analysis and studies, radiographic studies		• Quantitative sweat Cl^- (pilocarpine iontophoresis)
Clinical manifestations	• Wheezing, non-productive cough, CO_2 retention, dyspnea, obstructive emphysema, atelectasis, chronic respiratory infections, clubbing, cyanosis, hemoptysis, cor pulmonale, PHTN, pneumothoraces	• Meconium ileus (newborn), rectal prolapse (infancy), steatorrhea, azotorrhea, diabetes mellitus, distal intestinal obstruction, failure to thrive (FTT)	• Delayed puberty, amenorrhea, inhibited fertility, sterility (low sperm production)	• Generalized edema, hypoalbunemia, decrease protein absorption, "salty" taste to skin, emaciated, sallow, drooping skin, easy bruising
Therapy and Treatment	• Pulmonary toileting - Aerosolized treatment DNase - Chest physiotherapy (CPT) - Flutter mucus clearance device - Therapy vest • Aerosolized antibiotic, partial pleurectomy, pleurodesis, lung transplantation	• Pancreatic enzyme supplements (trypsin, chymotrypsin, amylase, lipase), vitamin supplements, high-protein, high-caloric diet, salt supplements, insulin therapy, liver transplantation	• Growth hormone therapy	
Common pathogens	• *Pseudomonas aeruginosa* • *Burkholderia cepacia* • *Staphylococcus aureus* • *Haemophilius influenzae* • *Escherichia coli* • *Klebsiella pneumoniae*			

Additional Neonatal Considerations

Neonatal Period

Neonatal period is described as the first 30-90 days post 40 weeks gestation. Premature infants are at greater risk, since lung and pulmonary development is not complete until after 36 weeks gestation.

Common Concerns During Neonatal Period

- Gestational age
- Fetal Hb
- Hyperbilirubinemia
- Lung immaturity
- Immature immunities
- Congenital heart disease
- Persistent fetal circulation (primary pulmonary hypertension)
- Cold stress effects
  - VO_2
  - Hypoxia
  - Pulmonary vasoconstriction
  - Respiratory distress
  - Metabolic rate
  - Surfactant consumption
  - Metabolic acidosis
  - Cyanosis
  - Apnea
  - Hypoglycemia
  - Catecholamine circulation
  - Peripheral vasoconstriction

Pediatric Sepsis, Shock and Multi-Organ Dysfunction Syndrome (MODS)

Pediatric Shock and Sepsis

Pediatric and neonatal sepsis generally have much lower mortality rates than adults. The American College of Critical Care Medicine (ACCM) has developed guidelines for treating sepsis in pediatrics with goal-directed algorithm (see page 70). Pediatric septic shock with low cardiac output has a higher association with mortality, when compared to adults, who generally have a higher mortality from experiencing low systemic vascular resistance in septic shock. Recommended updates in the hemodynamic support guidelines for pediatric and neonatal septic shock, include continuous $ScvO_2$ monitoring.

International Consensus Pediatric Sepsis Criteria:

- Hypotension >40 mL/Kg isotonic fluid bolus
- Vasoactive medications to maintain BP
- 2 or more of the following:
  - Base deficit >5 meq/L
  - arterial lactate > twice normal
  - oliguria (UOP* <0.5 mL/Kg/hr)
  - CRT+ >5 sec
  - core to peripheral temperature gap > 3°C

Severe Sepsis Criteria

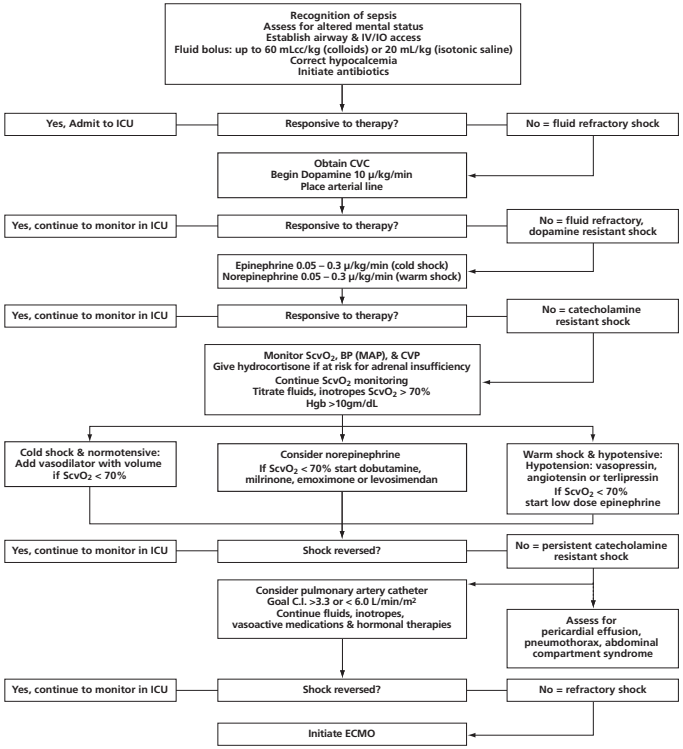
- GCS** <15 without CNS++ disease
- Lactate >1.6 mmol (arterial) or >2.2 mmol (venous)
- Measured UOP <1 mL/Kg for 2 hrs

Criteria for Septic Shock

- Hypotension after 20 mL/Kg fluid bolus
- Needs inotrope or vasopressor besides dopamine
- Any criteria from severe sepsis

[1] Refer to the reference section for the ACCM pediatric septic shock algorithm.
*UOP = urinary output
**GCS = Glasgow coma score
+CRT = capillary refill time in seconds
++CNS = central nervous system

PEDIATRIC SEPTIC SHOCK ALGORITHM



SHOCK DEFINITIONS

Cold or Warm	Fluid-refractory/ dopamine-resistant	Catecholamine-resistant	Refractory
↓Mental status, perfusion CRT* >2 sec (cold) Flash CRT* (warm) Diminished pulses (cold) Bounding pulses (warm) Mottled extremities (cold) ↓UOP⁺ (<1mL/Kg/h)	Shock despite fluid bolus >60 mL/Kg and dopamine infusion 10µ/Kg/min	Shock despite epinephrine (cold shock) or norepinephrine (warm shock)	Shock despite goal-directed therapy: inotropes, vasopressors, vasodilators, metabolic agents (glucose, calcium) and hormonal homeostasis therapy (insulin, thyroid, hydrocortisone)

*CRT = capillary refill time in seconds
⁺UOP = urinary output

TYPES OF SHOCK

Hypovolemic	Cardiogenic	Obstructive	Endocrine
• Decrease intravascular volume • Internal/external bleeding • Burns, capillary leak syndrome, protein losing enteropathy (PLE) • Vomiting, diarrhea, diuretics, endocrine	• Congenital heart disease, arrhythmias • Cardiomyopathy • Infections, toxic disorders • Metabolic disorders • Neuromuscular disorders • Connective tissue diseases • Hypoxia, trauma	• Cardiac tamponade • Hemothorax or pneumothorax • Coarctation, IAA	• Vasodilatory shock • Vasopressin deficiency • Thyroid storm • Adrenal insufficiencies
Septic		**Distributive**	
• Infections • Inflammatory response		• Anaphylaxis • Spinal cord injuries	

SHOCK STAGES

Compensated	Uncompensated	Irreversible
• Compensatory mechanisms to maintain BP • Increase HR and myocardial contractility • Systemic venous constriction • Systemic arterial constriction – Cool, pale skin – Muscle weakness – Restlessness, irritability – Decrease UOP – GI dysfunction* – BP normal	• Local tissue hypoxia • Beginning lactic acidosis • Intense sympathetic vasoconstriction – Mental deterioration – Cold, clammy skin – Pale mucus membranes and nail beds – Hypotensive – S-T changes → myocardial ischemia – Oliguria	• Profound tissue hypoxia • Profound organ damage • Severe hypoperfusion • Anaerobic metabolism • Stiff lungs • Inadequate cardiac output • Leaky capillary syndrome • Anuria • GI tract necrosis* • Death

*GI = gastrointestinal

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS) CRITERIA

Temperature	Heart Rate	Respiratory Rate	WBC
>38˚C or <36˚C (rectal) >37.8˚C or <35.8˚C (oral) >37.2˚C or <35.2˚C (axillary)	>90th percentile for age	>90th percentile for age $PaCO_2$ <32 mmHg	>12,000 <4,000

Multiple Organ Dysfunction Syndrome (MODS)

MODS often results following sepsis, but can occur in patients with other diagnosis. Critically ill pediatric patients who experience MODS have high association of mortality.

It can be defined as an acute dysfunction of two or more organ systems, as listed in the diagnostic criteria.

Primary: diagnosed within first 7 days of PICU admission

Secondary: diagnosis of MODS >7 days after admission to PICU; or within 7 days and sequential organ dysfunctions in an interval >72 H after MODS diagnosis and onset; and as a consequence of SIRS

DIAGNOSTIC MODS CRITERIA

Organ system	Criteria
Cardiovascular	SBP <40 mmHg (<1 yr) or <50 mmHg (>1 yr) HR <50 or >220 bpm (if <1yr) or <40 or 200 bpm (if >1 yr) Cardiac arrest pH <7.2 meq/L with normal PaO_2 IV inotropes other than dopamine
Pulmonary	RR >90 (if <1 yr) or > 70 (if >1yr) $PaCO_2$ >65 mmHg or <40 mmHg (non-cyanotic congenital heart disease) Mechanical ventilation PaO_2 <200 (non-cyanotic congenital heart disease)
Neurologic	Glasgow coma score <5 Fixed-dilated pupils
Hematologic	Hb <50 gm/dL WBC <3,000 Platelet <20,000 $_D$-Dimer >0.5 mg/mL with PTT >20 sec
Renal	BUN >36 mmol/L Creatinine >2.0 mg/dL without pre-existing renal disease Dialysis
Hepatic	Total bilirubin >3 mg/dL
Gastrointestinal	Gastric bleeding and one of the following: Hb drop >20gm/dL, blood transfusion, hypotension, gastric surgery or death

Traumatic Brain Injury

Traumatic Brain Injury can be primary and direct trauma to the head, intracranial malformations, CNS disorders and/or infectious process. Secondary are often resultant from cerebral hypoxia, edema and decreased cerebral perfusion. Intracranial pressure (ICP) is monitored continuously via an intraventricular catheter, which allows for CSF drainage for increased pressures and evaluating cerebral perfusion pressures (CPP).

$$CPP = MAP - ICP$$

Suggested value: CPP ≥40 mmHg (infants) or ≥50 mmHg (children)

NORMAL INTRACRANIAL PRESSURE (ICP)

Age Group	ICP (mmHg)
Newborn	0.7-1.5 mmHg
Infants	1.5-6.0 mmHg
Children	3.0-7.5 mmHg
Adolescents - adults	<10 mmHg

ALTERED STATES OF CONSCIOUSNESS

State	Presenting Symptoms
Cloudiness	Lethargy, confused, drowsy alternating with hyperexcitability
Delirium	Disorientation, fear, irritability, visual hallucinations, agitation
Obtunded	Increase periods of sleep, decrease alertness, disinterested
Stupor	Unresponsive except to noxious stimuli
Coma	No responsiveness

Jugular Bulb Monitoring (SjO_2)

See Advanced Minimally Invasive Monitoring.

Endocrine (Glucose) Monitoring

Glucose Monitoring

Glucose is formed by gluconeogenesis during the digestive process and is vital for cerebral metabolism .

Hypoglycemia (<40 mg/dL) is a potential metabolic problem for critically ill children. This can be the result of overutilization of glucose or underproduction of glucose via glycogenolysis or conversion of gluconeogenic precursors. Infants and children have less glycogen stores and larger glucose requirements compared to adults.

Glycogen reserve depletion:	Glucose requirements:
<12 hours (infant)	8-10 mg/Kg/min (infant)
<24 hours (older child/adult)	2-3 mg/Kg/min (older child/adult)

Hypoglycemia occurs when fuel sources (lactate, ketones, and free fatty acids) are depleted and counter regulatory hormones (insulin, cortisol, epinephrine, norepinephrine, growth hormone) stimulation is maximized causing:

- Glycogen store depletion
- No glycemic response to glucagon
- Exhaustion of gluconeogenic stores (low lactate level)
- Elevated free fatty acids and ketones
- Insulin undetectable

Common Causes:

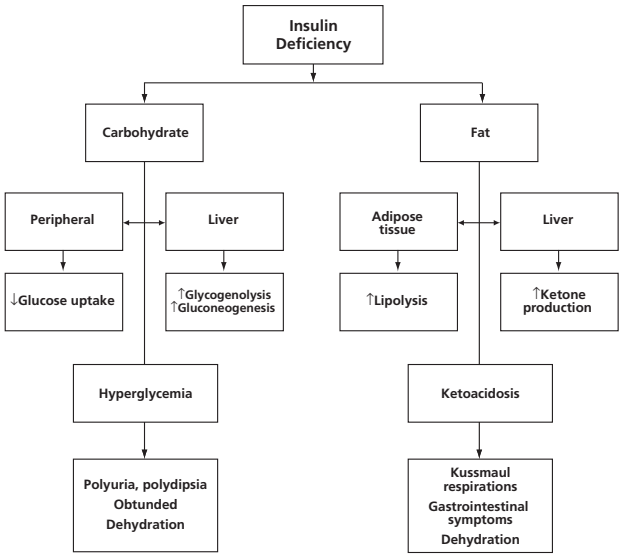
- Glycogen storage disorders/deficiencies
- Gluconeogenesis disorders/deficiencies
- Fatty acid disorders
- Hormone deficiencies
- Hyperinsulinism
- Iatrogenic/drug-induced
- Other (includes sepsis)

Hyperglycemia is the body's response to stress, usually from an overproduction of glucose rather than an impairment of glucose uptake. Hyperglycemia can be defined as a fasting blood glucose ≥110 mg/dL. Threshold values to start insulin therapy range from 110 mg/dL to >200 mg/dL. Patients with hyperglycemia have prolonged ICU stays, poorer neurodevelopmental outcomes, increased infection rates and is associated with increased mortality.

Stress Hyperglycemia Hormone/Cytokine Mediators

- Glucagon
- Epinephrine
- Norepinephrine
- Glucocorticoids
- Growth hormone
- Tumor necrosis factor

INSULIN DEFICIENCY ALGORITHM



Diabetes Mellitus (DM)

Diabetes Mellitus (DM) is a result of decreased secretion of insulin in the beta-cells of the islets of Langerhans in the pancreas. Causes can be hereditary, obesity, autoimmune antibodies. Risks during DM are osmotic pressure on extracellular fluid leads to dehydration; loss of glucose in the urine and osmotic diuresis depletes fluids and electrolytes. DM is associated with certain syndromes, infections, medications and endocrinopathies.

Diabetes Ketoacidosis (DKA)

Diabetic Ketoacidosis is a common complication of diabetes mellitus. DKA = glucose >200 mg/dL, positive ketonemia/ketonuria and pH <7.3 and HCO^{3-} <15 mEq/L. It is a life-threatening manifestation of severe insulin deficiency that can lead to cerebral edema from hyperglycemia.

Characterization:

- Inadequate insulin
- Hyperglycemia (due primarily from insulin deficiency)
- Dehydration
- Electrolyte loss
- Metabolic acidosis
- Ketosis

Insulin deficiency is the result of:

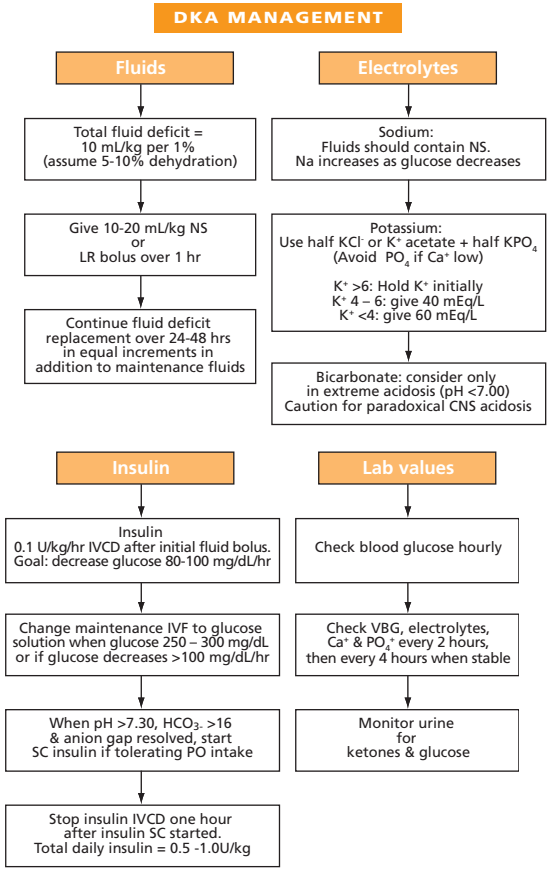
- Increase gluconeogenesis
- Accelerated glycogenolysis
- Impaired peripheral glucose utilization

Treatment:

- Fluid expansion with isotonic solution (0.9% NS or LR) 10-20 mL/Kg for first hour
- Insulin IVCD 0.05-0.1U/Kg/hr decreasing serum glucose 100 mg/dL/hr until serum glucose = 200-300 mg/dL
- Start glucose solution for hydration maintaining serum glucose 100-150 mg/dL

DKA FLUID/ELECTROLYTE REQUIREMENTS

Element	Maintenance	Losses
H_2O	1500 mL/m^2	60-100 mL/Kg
Na^-	45 mEq/m^2	5-13 mEq/Kg
K^+	35 mEq/m^2	4-6 mEq/Kg
Cl^-	30 mEq/m^2	3-9 mEq/Kg
PO_4^-	10 mEq/m^2	2-5 mEq/Kg



CNS = central nervous system
IVCD = intravenous continuous drip
IVF = intravenous fluid
LR = lactated ringers
NS = 0.9 normal saline
PO = oral
SC = subcutaneous
VBG = venous blood gas

Diabetes Insipidus (DI)

Diabetes Insipidus is a result of vasopressin deficiency or vasopressin insensitivity from genetic, anatomic or acquired causes. Patients most commonly seen in ICU with DI are post head trauma or post cranial surgical procedures.

Syndrome of Inappropriate Antidiuretic Hormone (SIADH) is a result of vasopressin or antidiuretic hormone (ADH) hyper-secretion in the posterior pituitary gland. Patients in the ICU experiencing SIADH may be from central nervous system (CNS) infection, trauma, tumors, surgery or other CNS disorders and pneumonia.

ALTERED PITUITARY SECRETION (DI AND SIADH)

Measurement	DI	SIADH
Urine output	Increased	Decreased
Specific gravity	Decreased	Increased
Serum sodium	Hypernatremia	Hyponatremia
Clinical findings	Dehydration	Fluid retention

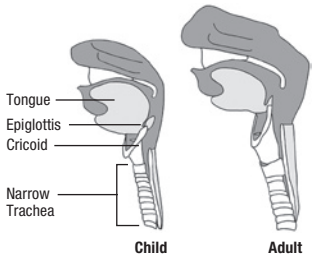
Basic Monitoring and Procedures

Airway Management

Neonatal and pediatric airways differ from adult airways until about age 8–14 years. Airways differ in size, shape and airway position within the neck. Tracheal diameter is considerably smaller than an adult, making infants and children more susceptible to edema and obstruction. The infant tongue is large in proportion to the oral cavity. Diameter and tracheal length increase with age.

Differences in pediatric/adult airway

- Airway more superior (pediatric) versus anterior (adult)
- Tongue takes greater proportion of oral cavity
- Higher tracheal opening location
  - C1 (infancy)
  - C3–4 (up to 7 years)
  - C4–5 (adult)
- Cricoid ring narrowest portion (pediatric) versus vocal cords (adult)
- Larger tonsils and adenoids
- Acute angle between epiglottis and larynx
- Small cricothyroid membrane



ETT estimation calculation

Pediatric

$$\text{Tube (mmID)} = \frac{\text{age (yr)}}{4} + 4 \quad \text{or} \quad \text{Tube size} = 16 + \frac{\text{age (yr)}}{4}$$

$$\text{Tube size} = \frac{\text{Height (cm)}}{20}$$

Newborns

$$\text{Tube size (mmID)} = \frac{\text{post-conceptual age (wks)}}{10}$$

Tube insertion length >2 yrs

$$\text{Distance (cm)} = \frac{\text{Age (yr)} + 12}{2} \quad \text{or} \quad \text{ETT (ID)} \times 3$$

**Note: internal diameter is approximate size of patient's fifth finger*

ETT GUIDELINE SIZES FOR INFANTS/CHILDREN

Age	Internal Diameter (mm)	Orotracheal (Length in cm)	Nasotracheal (Length in cm)
Premature	2.0–3.0 (uncuffed)	6–8 cm	7–9 cm
Newborn	3.0–3.5 (uncuffed)	9–10 cm	10–11 cm
3–9 months	3.5–4.0 (cuffed*/uncuffed)	11–12 cm	10–13 cm
9–18 months	4.0–4.5 (cuffed*/uncuffed)	12–13 cm	14–15 cm
18 months–3 years	4.5–5.0 (cuffed*/uncuffed)	12–14 cm	16–17 cm
4–5 years	5.0–5.5 (cuffed/uncuffed)	14–16 cm	18–19 cm
6–7 years	5.5–6.0 (cuffed/uncuffed)	16–18 cm	19–20 cm
8–10 years	6.0–6.5 (cuffed/uncuffed)	17–19 cm	21–23 cm
11–13 years	6.0–7.0 (cuffed/uncuffed)	18–21 cm	22–25 cm
14–16 years	7.0–7.5 (cuffed/uncuffed)	20–22 cm	24–25 cm

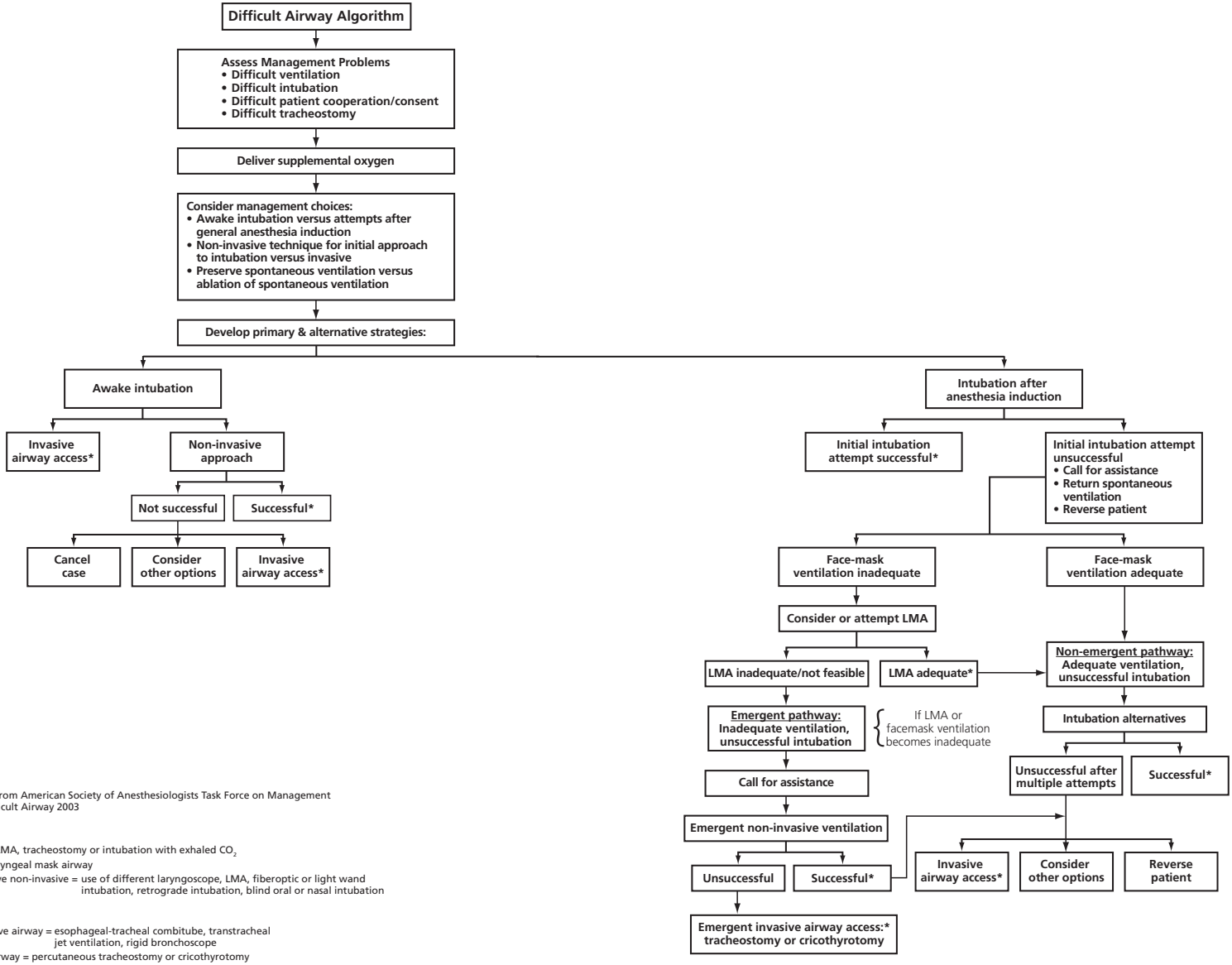
*Cuffed ETT in <2y/o children can be considered controversial but may also be indicated.

LARYNGOSCOPE BLADE SIZE

Age	Size
0–1 month (<2.5Kg)	0 (straight)
0–3 months	1.0 (straight)
3 months–3 years	1.5 (straight)
3–12 years	2.0 (straight or curved)
12–18 years	3.0 (straight or curved)

LARYNGEAL MASK AIRWAY (LMA)

LMA Size	Weight (Kg)	Cuff Volume (mL)
1	<5 Kg	2–5 mL
1.5	5-10 Kg	3–8 mL
2	10-20 Kg	5–10 mL
2.5	20-30 Kg	10–15 mL
3	30-50 Kg	15–20 mL
4	50-70 Kg	25–30 mL



Modified from American Society of Anesthesiologists Task Force on Management of the Difficult Airway 2003

*Confirm LMA, tracheostomy or intubation with exhaled CO_2

LMA = laryngeal mask airway

Alternative non-invasive = use of different laryngoscope, LMA, fiberoptic or light wand intubation, retrograde intubation, blind oral or nasal intubation

Non-invasive airway = esophageal-tracheal combitube, transtracheal jet ventilation, rigid bronchoscope

Invasive airway = percutaneous tracheostomy or cricothyrotomy

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-abdominal pressure) to an electrical signal which is amplified, filtered and displayed on a bedside physiologic monitor in both a waveform and numeric value in mmHg.

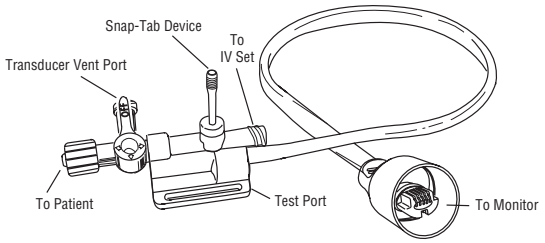
TruWave disposable pressure transducers are available in two sizes, 3 mL/hr (standard) and 30mL/hr (umbilical) flush devices. The 3 mL/hr transducer has a blue pull tab and the 30 mL/hr transducer has a yellow pull tab. The 3 mL/hr flush device is designed to be used with pressure bag inflated to 300 mmHg. The 30 mL/hr (umbilical) flush device is designed to be used only with an infusion or syringe pump with the integral flush device acting as a restrictor to prevent free flow fluid in excess of the mL/hr flush rates (30 mL/hr) in case the tubing becomes detached from the pump.

The 30 mL/hr flush device is specifically designed for **neonatal use only via the umbilical artery.** It should *only* be used in conjunction with an infusion or syringe pump instead of a pressure bag. The umbilical artery is the *only* artery that can be used for a variety of intravenous infusions beyond arterial pressure monitoring. Use of infusion or syringe pumps should be used to ensure adequate control of flow rates. Higher-flow capability flush devices (30 mL/hr) allow for higher volume (> 3 mL/hr) intravenous infusions as well as to prevent high pressure alarms on infusion or syringe pumps that might occur if a standard (3 mL/hr) flush device were used.

The umbilical artery can be used for:

- Intravenous infusions
- Medications
- Parenteral nutrition
- Arterial blood pressure monitoring
- Resuscitation
- Transfusion
- Blood sampling

TRUWAVE DISPOSABLE PRESSURE TRANSDUCER COMPONENTS



WARNING: Do not use a flush device during Intracranial pressure (ICP) monitoring. Disposable pressure transducers without flush devices should be used for ICP monitoring.

Components of a Physiologic Pressure Measurement System

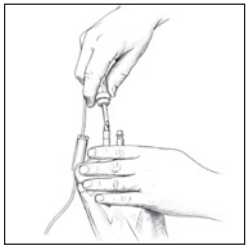
- Invasive catheter
- Edwards TruWave kit
  - Non-compliant pressure tubing
  - Stopcocks
  - Transducer housing
  - 3 mL/hr flush device*
  - Cable connection
  - Fluid administration set
- Normal saline flush solution (500 or 1000 mL) (Heparin per institutional policy)
- Pressure infusion bag (Appropriately 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.

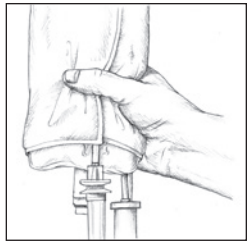
**Note: 30 mL/hr neonatal TruWave Kit is used only on the umbilical artery with infusion or syringe pumps instead of pressure bags.*

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

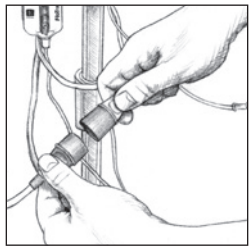
1. Wash hands.
2. Open TruWave disposable pressure transducer packaging and inspect contents. Replace all caps with non-vented caps and ensure that all connections are tight.
3. Remove the TruWave transducer from its packaging and insert into an Edwards Lifesciences mounting back-plate that is secured 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).



   - See instructions below if using infusion pump instead of a pressure bag and advance to step 8
5. Insert flush bag into pressure infuser bag (DO NOT INFLATE) and hang on IV pole at least 2 feet (60 cm).
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 catheter, and then aspirate and flush system to assure catheter is intra-vascular and remove residual bubbles.

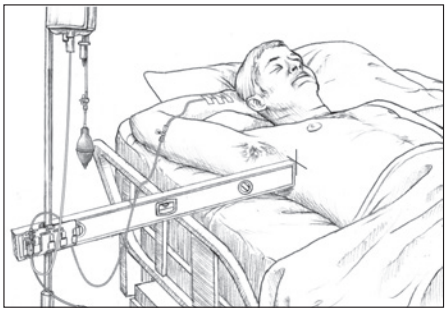


11. Level the stopcock just above the TruWave transducer to the phlebostatic axis.
12. Open the stopcock to atmospheric air. Zero pressure, per bedside monitor's instructions for use.
13. Inspect pressure trace on bedside monitoring screen to confirm appropriate pressure scale, alarm settings, pressure label, color coding and physiologic waveform are present.

**Note: When using a syringe pump, draw up the flush solution into a syringe (syringe size based upon institution policy) or prime compatible infusion pump tubing. An in-line filter may be added to tubing at the connection before the transducer to decrease risk of embolism if cyanotic cardiac lesion present (based upon institution policy). Prime the extension or IV tubing and TruWave transducer tubing. Ensure that all air bubbles are removed and eliminated.*

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.

TruWave Disposable Pressure Transducer Specifications	
Operating Pressure Range	-50 to +300 mmHG
Operating Temperature Range	15° to 40° C
Storage Temperature Range	–25° to +70° C
Sensitivity	5.0µ V / V / mmHg ± 1%
Nonlinearity and Hysteresis	± 1.5% of reading or ±1 mmHg whichever is greater
Excitation Impedance	350 ohms ± 10% with Edwards Monitor Cable attached
Signal Impedance	300 ± 5%
Zero Offset	± 25 mmHg
Zero Thermal Drift	± 0.3 mmHg / °C
Output Drift	± 1 mmHg per 8 hours after 20 second warm-up
Sensitivity Thermal Drift	± 0.1% / °C
Natural Frequency	40 Hz nominal for a standard kit (48"/12"); > 200 Hz for transducer alone
Leakage Current	<2µ amps at 120V RMS 60 Hz
Overpressure Tolerance	–500 to +5000 mmHg

Flow Rate Across Flush Device with IV Bag Pressurized to 300 mmHg	
• Blue Snap-Tab	3±1 mL/hr
• Yellow Snap-Tab	30±10 mL/hr

***At 6.00VDC and 25°C unless otherwise stated. All specifications meet or exceed the AAMI Standard for performance interchangeability of resistance bridge type blood transducers.*

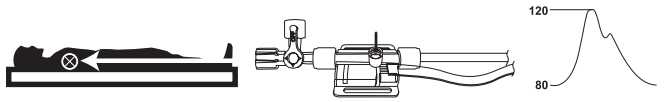
Best Practice in Maintaining Physiologic Pressure Transducer System

- **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

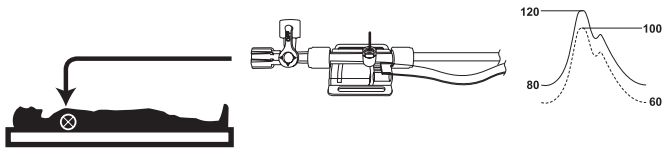
Impact of Improper Leveling on Pressure Readings

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.

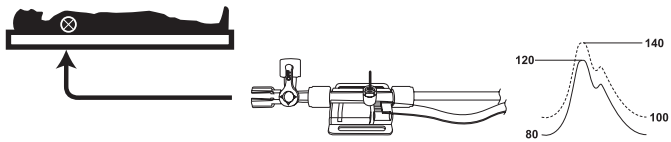
For every inch (2.5 cm) the heart is offset from the reference point of the transducer, a 2 mmHg of error will be introduced.



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 over damped physiologic pressure system will result in an underestimated systolic pressure and an overestimated diastolic pressure.

An under damped physiologic pressure system (commonly referred to as 'whip') 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.

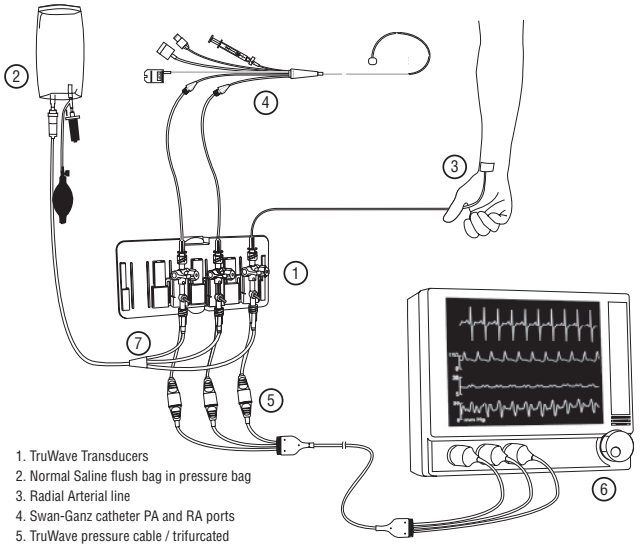
Note: See page 95 for further information and examples of square wave tests.

Pressure Monitoring Systems

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) or via constant flow using an infusion pump. An integral flush device with a restrictor limits the flow rate to approximately 3 mL/hour for children and adults. Typically, heparinized normal saline is used as the flush solution with a range of heparin from 0.25 u/1mL to 2 u/1mL ratio. Non-heparinized solution has been used with patients with sensitivity to heparin. (The use of heparin or non-heparin flush solution is per each individual institution and the assessment of risk vs. benefit to the patient.)

For infants (<6 Kg), a syringe or infusion pump may be utilized to limit amount of hourly fluid delivered to 1-2 mL/hr for pressure lines compared to the 3 mL/hr on a pressure bag system. It is recommended that a minimum of at least 1mL/hr is infused on all pressure lines.

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

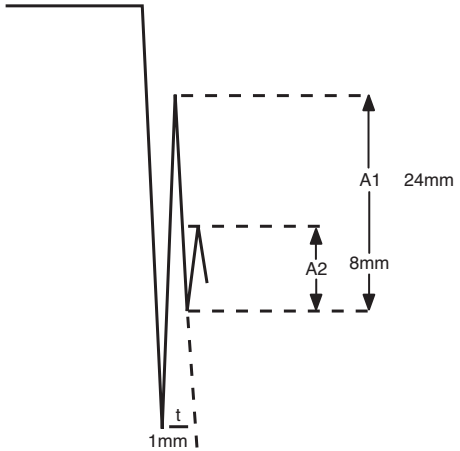
Activate the flush device by pulling the snap tab or pull tab. Observe the bedside monitor. The waveform will sharply rise and "square off" at the top. Observe the tracing as it returns to baseline.

Calculate the Natural Response (fn)

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

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

AMPLITUDE RATIOS

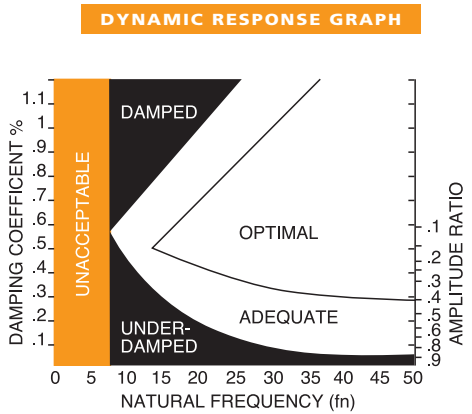


Determine the Amplitude Ratio

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.

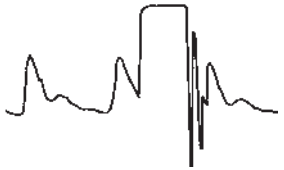


Simple Evaluation of Dynamic Response

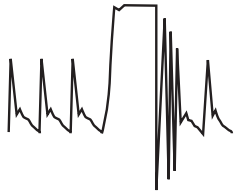
Determining the dynamic response characteristics of a pressure monitoring system by calculating the amplitude ratio and damping coefficient may not be feasible at the bedside when a rapid assessment of the waveform is required. A simple evaluation of dynamic response can be obtained by performing a square 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

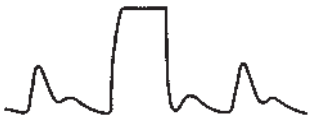
1. Activate snap or pull tab on flush device
2. Observe square wave generated on bedside monitor
3. Count oscillations after square wave
4. Observe distance between the oscillations



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

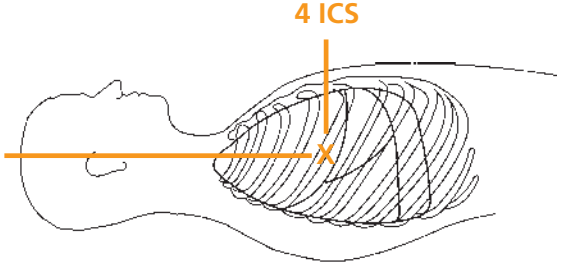
To obtain accurate pressure measurements, the level of the air-fluid interface must be aligned with the chamber or vessel being measured.

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.

PHLEBOSTATIC AXIS



Mid-Point
A-P Chest Wall

Components of 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: closure of the aortic valve, marking the end of systole and the onset of diastole

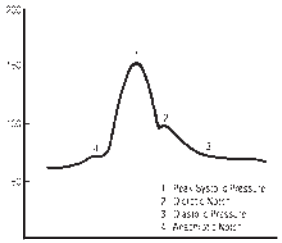
Diastolic pressure: relates to the level of vessel recoil or amount of vasoconstriction in the arterial system. May be termed the descending limb

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

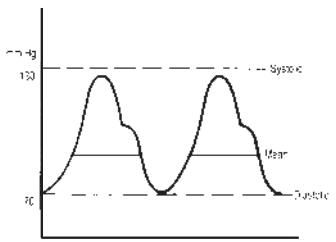
Pulse pressure: difference between systolic and diastolic pressure

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

COMPONENTS OF ARTERIAL PULSE



MEAN ARTERIAL PRESSURE



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

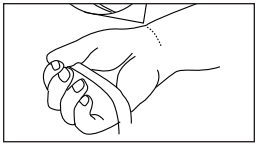
Arterial Pressure Monitoring

Intra–Arterial Monitoring

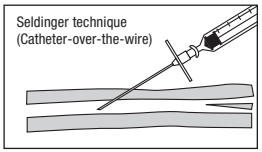
Steps for Radial Arterial Catheterization

The radial artery is the most common artery used for continuous blood pressure monitoring and arterial sampling. (For other insertion sites, refer to alternative catheterization sites on page 100.)

1. Wash hands.
2. Radial artery is the most commonly used site for arterial catheterization.
   - Left radial artery should be avoided if the left subclavian artery will be used in a coarctation of the aorta repair.
   - Right radial artery should be avoided if right subclavian artery will be used in an arterial-to-pulmonary shunt repair.
3. An Allen test may or may not be done prior to catheterization of the radial artery.
4. Dorsiflex the wrist (it may be helpful to restrain wrist onto an arm board).

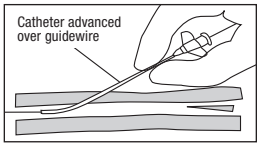


5. Apply sterile gloves and cleanse site according to institution policy.
6. Commonly an over-the-needle (OTN) catheter or introducer needle is used. Insert it percutaneously over the site of the radial artery.
   - The needle and catheter can be advanced until arterial blood flow is seen in the needle hub, advance the catheter over the needle into the artery.

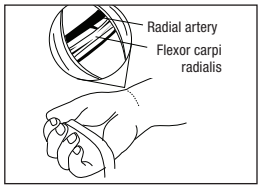


   - An alternative technique is to advance the needle and catheter until arterial flow stops, transfixing the artery. Remove the needle and gently pull back the catheter until flow returns. The catheter can then be advanced into the artery.

   - If using an introducer needle and modified Seldinger technique, then a short guidewire can be inserted through the catheter. The catheter can then be threaded over the guidewire into the artery.



   - Surgical cutdown may be attempted if previous percutaneous arterial catheterization is unsuccessful or if previous arterial catheterizations have been performed.



Arterial Cutdown Catheterization

Arterial cutdown catheterization is done in the same manner as described in the section for venous cutdown catheterization, but accessing the artery.

1. The procedure should be performed under strict sterile guidelines as a surgical procedure.
2. Make a transverse incision at the site, deep enough into the subcutaneous tissue, but not deep enough as to lacerate the artery.
3. Isolate the artery using a blunt dissection. Place two suture threads under the vessel in a sling-like fashion.
4. The distal suture is tied and used to stabilize the vessel. An arteriotomy is performed to insert the catheter, or the catheter can be inserted with an over the needle approach similar to percutaneous insertions.
5. Following successful placement (confirmed by aspirating blood), the proximal suture can be tied. The incision is closed and a dressing is placed according to institution policy.

Alternative Sites Used For Arterial Catheterizations

- *Umbilical:* easy access on newborns within first few weeks of life. There are two umbilical arteries and one umbilical vein. The umbilical arteries are small, thick walled and generally constricted
- *Femoral:* frequently used site, offers reliable measurements, may be reserved for patients requiring future cardiac catheterization. Avoid ipsilateral cannulation of femoral artery and vein (for central line access or ECMO cannulation)*
- *Dorsalis pedis or posterior tibialis:* maybe less reliable following cardiac bypass surgery*
- *Brachiocephalic:* rarely used, decrease collateral circulation*
- *Axillary:* has good collateral circulation, risk for retrograde embolism to aorta or cerebral vessels*

Arterial Catheter Size	Site	Catheter (OTN) Size	French Size
Infants <10 Kg	RA, PTA, DPA, TA	24Ga/22Ga	2.5 Fr
	Femoral, Axillary	22Ga/20Ga	2.5 Fr/3 Fr/3.5 Fr
	Umbilical		3.5 Fr
10-40 Kg	RA, PTA, DPA, TA	22Ga/20Ga	2.5 Fr/3 Fr/3.5 Fr/4 Fr
	Femoral, Axillary	20Ga/18Ga	3 Fr/3.5 Fr/4 Fr
>40 Kg	RA, PTA, DPA, TA	22Ga/ 20Ga	
	Femoral, Axillary	18Ga/16Ga	3 Fr/4 Fr

Note: Radial artery (RA), Posterior tibial artery (PTA), Dorsalis pedis artery (DPA), Temporal artery (TA)

**All extremity sites include risk for loss of limb with arterial catheterization*

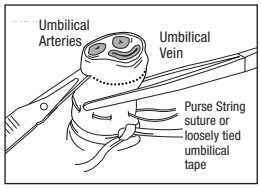
ABNORMAL ARTERIAL PRESSURE WAVEFORMS

Elevated systolic pressure	Systemic hypertension Aortic insufficiency
Decreased systolic pressure	Aortic stenosis Heart failure Hypovolemia Pericardial effusion
Widened pulse pressure	Systemic hypertension Aortic insufficiency Increased intracranial pressure
Narrowed pulse pressure	Pericardial effusion Cardiac tamponade Congestive heart failure Cardiogenic shock Aortic stenosis
Pulsus bisferiens	Aortic insufficiency Obstructive hypertrophic cardiomyopathy
Pulsus paradoxus	Pericardial effusion Cardiac tamponade Pulmonary embolism
Pulsus alternans	Congestive heart failure Cardiomyopathy

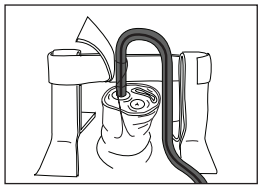
Steps for Umbilical Arterial Catheterization

1. The umbilical cord (often referred to as the umbilical stump) is secured with a loose umbilical tie at the base.
2. Grasp the cord with forceps (0.5–2 cm from base); make a transecting cut to clearly reveal the vessels.

   Catheters can be placed "high" (resides at T6–T9) or "low" (resides at L3–L5). A high cannulization is calculated as follows: 3 x (Kg) + 9 = length in cm.
3. Insert the catheter into the umbilical artery and suture using a purse-string tightly cinched suture. Then apply a square knot suture 1 cm above the umbilical stump before tying off.



4. Use a bridge dressing (indicated below) to secure in place.



5. Continuous infusion solution is necessary to keep catheter patent. A solution with heparin 1U/mL is recommended at infusion rates of 0.5 mL/hr for 3.5 Fr catheter and 1 mL/hr for a 5 Fr catheter.
6. If blanching of the lower extremities occurs secondary to vasospasm, apply heat to the contralateral leg. Remove the UAC if blanching is persistent to prevent ischemic complications of the lower extremities. Complications are generally rare with UAC lines, but can include thrombosis of the aorta or its major branches, hemorrhage, infection, aneurysmal dilation of the aorta and umbilical artery rupture.

Removal of Umbilical Arterial Catheter

Removing the umbilical arterial catheter is done by withdrawing the catheter slowly in increments of 1–2 cm over several minutes. This allows for the artery to spasm and hemostasis to occur. If bleeding persists, apply pressure to the umbilical cord by pinching the stump.

Central Venous Access

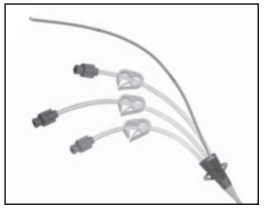
Types of Central Venous Access Devices

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 mostly 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 increase 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.

Edwards 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.



Indications for Use of Central Venous Access Devices

- Rapid fluid administration – for example, in cases of, or at high risk of, high blood loss
    - Multiple trauma
    - Sepsis
    - Burns
    - Cardiothoracic surgery
    - High-risk surgery, post transplantation
- Administration of IV fluids requiring dilution within the central circulation to avoid vascular damage (i.e. chemotherapy, total parenteral nutrition with dextrose >10-12.5%)
- 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
- Coagulopathy or predisposition to bleeding

Complications

- Carotid artery puncture or cannulation secondary to the proximity of the internal jugular vein
- Pneumothorax (air in pleural space collapsing lung), internal jugular (IJ) approach has a lower incidence of a pneumothorax than a subclavian or low anterior (IJ) approach. Patients with overinflated lungs (i.e. PEEP) may have an elevated risk of pneumothorax especially with a subclavian approach
- Hemothorax (blood in pleural space collapsing lung), secondary artery puncture or laceration
- Hemorrhage within chest (hemothorax, tamponade) or from insertion site
- Pelvic or retroperitoneal bleeding with femoral approach
- 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, dysrhythmias, cardiac perforation, catheter migration SVC to RA, or extravascular
- Bloodstream infection if full barrier precautions not practiced at time of insertion or if aseptic techniques are not followed when accessing the line
- Nerve damage causing diaphragm paralysis

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
- Mitigating inadvertent carotid puncture/cannulation, multiple sticks
- Ultrasound guided central line placement

Note: Location of a CVC tip in the right atrium should be avoided due to the risk of cardiac perforation possibly resulting in a cardiac 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
- Double, triple or quad lumen catheters have more functional ports, but are usually of a smaller lumen size (i.e. 7 Fr 16/16 gauges vs. 7 Fr 18/18/18/16 gauge)

Cross Section of 7 Fr Double Lumen

Cross Section of 7 Fr Quad Lumen

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

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.

Length

Central venous catheters come in varying lengths. 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 positioning in-and-out of the skin and decreases chance of infection.

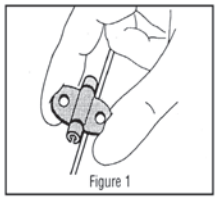


Figure 1

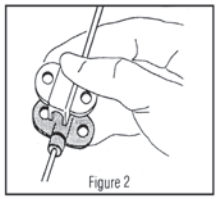


Figure 2

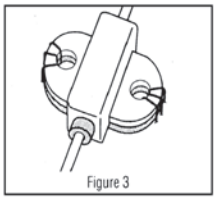


Figure 3

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		
Blood sampling		

*These are suggestions only.

CVC PORT COLOR DESIGNATION

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

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 thrombolytic 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.

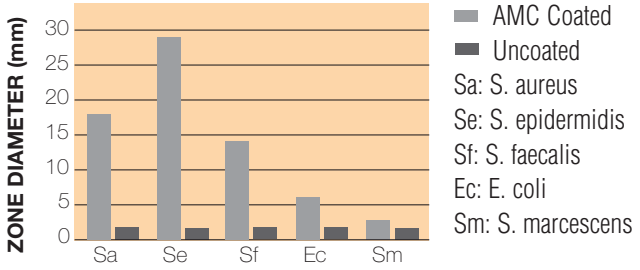
Antimicrobial Catheter Material

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.

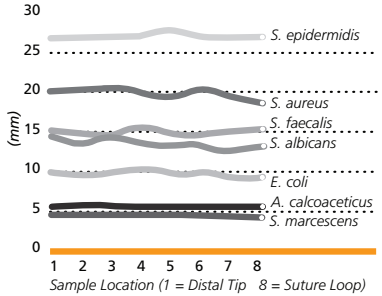
Infection Control with Double Protection

AMC Thromboshield treatment is an exclusive combination of benzalkonium chloride with heparin coating as a double-safeguard both inside and out. Benzalkonium chloride provides antimicrobial protection against commonly encountered organisms. AMC Thromboshield treatment is applied to the entire surface of the catheter, both inside and outside. It offers a significant advantage over catheters with protection only on the outside, as contamination can lead to infection in the inner lumen resulting in catheter-related sepsis.

MICROBIAL INHIBITION WITH AMC THROMBOSHIELD COATING



ZONE OF INHIBITION



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

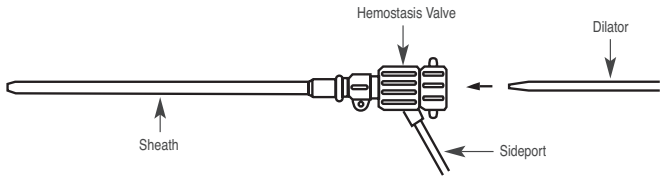
Sometimes an introducer is used for central venous access when rapid volume resuscitation is needed or is left in place following the removal of a pulmonary artery catheter. Components of the introducer system usually include:

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

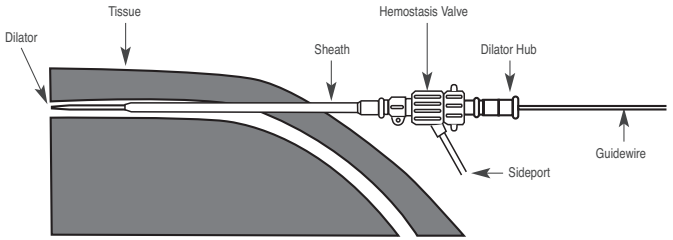
After insertion, the guidewire and dilator are removed, leaving the sheath in place. Fluids may be administered through the side port, while the hemostasis valve prevents bleed-back and/or air embolization.

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



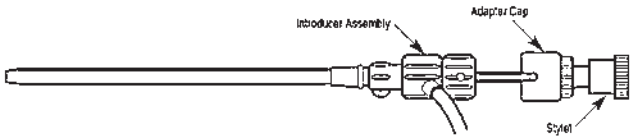
TUOHY-BORST VALVE INTRODUCER (INSERTED)



Infusion Catheter (aka Cordis CVC)

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.

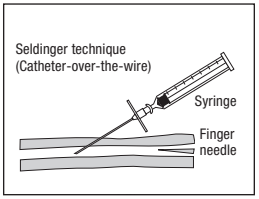
INFUSION CATHETER



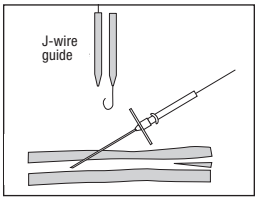
Modified Seldinger Technique for CVC Insertion

Procedure should be done using sterile technique with full body drape, mask, gown, hat and gloves. Identify site and landmarks for insertion of central line. Ultrasound-guided insertion is recommended to easily locate the vein, marking the site for needle insertion. Clean site with age appropriate antiseptic skin prep. Place fenestrated body drape over prepped site.

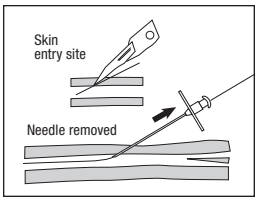
Using the introducer needle or over the needle (OTN) cannula, puncture the skin and enter the vessel lumen. Blood may freely flow back into the syringe when in vessel lumen or may begin to flow as needle is being pulled back.



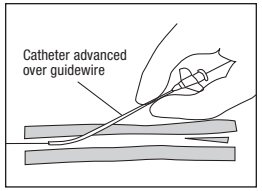
Hold the needle or OTN securely, remove the syringe and insert the guidewire. The guidewire should thread without resistance. Do not force the guidewire if resistance is met. Adjust the needle and re-attempt. Watch for ventricular ectopy during insertion of the guidewire. If ectopy is present, gently withdraw guidewire until it ceases. (Persistent ectopy usually suggests guidewire has been inserted into the heart.)



After guidewire has been easily threaded into the vessel, remove the needle while holding the guidewire secure in place. A small nick can be made in the skin for easier insertion of the central line catheter. If using a dilator, thread over guidewire to adequately dilate the vessel and remove.



Thread the central line catheter over the guidewire. Once catheter has been inserted to point where guidewire is visible exiting the distal lumen, secure the end of the guidewire while advancing catheter to desired position or location.

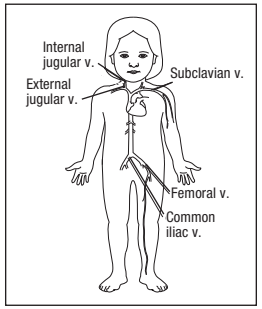


Secure catheter in place and each lumen. Blood should be easily aspirated from all catheter lumens. Confirm tip placement via radiologic study.

Insertion Sites

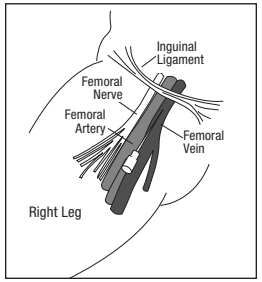
Central venous catheters are inserted via the internal jugular (IJ), subclavian or the femoral veins.

The femoral vein is superficial in the anterior thigh, passing beneath the inguinal ligament eventually becoming the external iliac vein. The femoral vein lies medial to the femoral artery which is medial to the femoral nerve. The mnemonic NAVL (nerve, artery, vein, lymphatic) is traditionally used, (lateral to medial) to remember order of structures when accessing the femoral vein. The patient should be flat and supine. The thigh is slightly abducted and externally rotated. The introducer needle should be inserted 2–3 cm distal to the inguinal ligament and 0.5–1 cm medial to the femoral pulse.

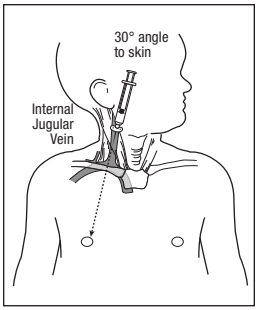


Note: Ipsilateral artery and vein cannulation is not recommended.

The internal jugular exits the jugular foramina to join the subclavian vein, which forms the innominate vein (or brachiocephalic) and continues to the superior vena cava. The right internal jugular is preferred over the left which has a sharp angle where the innominate joins the superior vena cava.

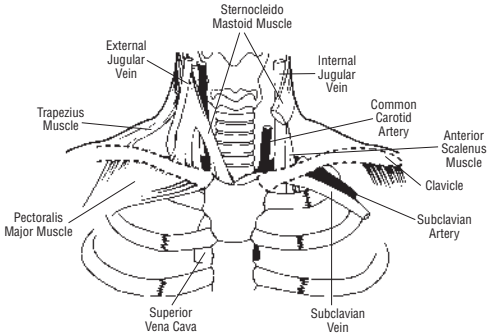


Approaches to the internal jugular can be anterior, middle or posterior depending on angle of insertion. Patient is placed supine in a slight trendelenburg position, a roll under the shoulders to extend the neck and the head turned away from side being approached.



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). The patient should be in a supine and slightly trendelenburg position with a roll along the spine between the shoulder blades. The introducer needle is inserted at the lateral and middle third of the clavicle, while directing it towards the sternal notch.

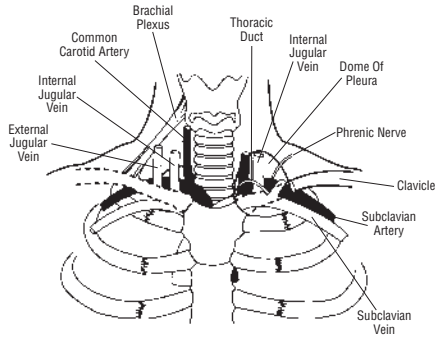
RELATIONSHIP OF CLAVICULAR LANDMARKS TO VASCULAR ANATOMY



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 pnemo-thorax. 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.

COMMONLY USED GUIDEWIRE, SHEATH & DILATOR SETS

Dilator/Sheath (Fr)	Needle (Gauge)	Guidewire Diameter (in.)
4 or 5	21	0.015/0.018
5 or 6	19	0.021, 0.025
7 or 8	18	0.035

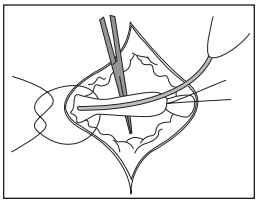
CENTRAL VENOUS CATHETER GUIDELINES

Age	Size (Fr)	Vein Location
<6 months	4 Fr	jugular/subclavian/femoral
6 months–2 years	4 Fr/5 Fr	jugular/subclavian/femoral
2–5 years	5 Fr/6 Fr	jugular/subclavian/femoral
>5 years	5 Fr/6 Fr/7 Fr/8 Fr	jugular/subclavian/femoral

Venous Cutdown Catheterization

Venous cutdown may be useful when rapid volume resuscitation or treatment for severe metabolic disturbances in the critically ill or injured child is required. And when percutaneous cannulation has been unsuccessful.

1. The procedure should be performed under strict sterile guidelines as a surgical procedure.
2. Make a transverse incision at the site, deep enough into the subcutaneous tissue, but not deep enough as to lacerate the vein.
3. Isolate the vein using a blunt dissection. Place two suture threads under the vessel in a sling-like fashion.
4. The distal suture is tied and used to stabilize the vessel. A venotomy is performed to insert the catheter, or the catheter can be inserted with an over the needle approach similar to percutaneous insertions.
5. Following successful placement (confirmed by aspirating blood and infusing fluids), the proximal suture can be tied. The incision is closed and a dressing is placed according to institution policy.

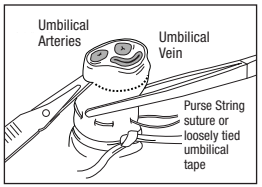


Umbilical Venous Catheterization

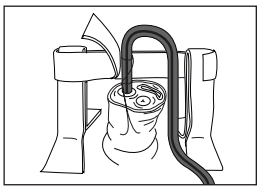
Umbilical venous catheterization can be used in newborns up to a few weeks old if there is an adequate umbilical cord (stump) remaining. The umbilical cord consists of two arteries and one vein. The umbilical vein can be identified as the largest vessel and is thin walled. Catheterization is performed in the same manner as catheterization of the umbilical artery. The tip of the catheter should reside at the junction of the IVC and right atrium. Using the following formula can help determine the length necessary for umbilical venous placement:

$$\frac{[(3(Kg) + 9]}{2} + 1 = \text{length in cm}$$

UVC generally should not be left in place greater than 10–14 days. Removal is similar to that of the UAC. Complications associated with umbilical venous placement include infection if catheter remains in place for a prolonged period, thrombosis formation can lead to portal hypertension if occurs in the portal venous system, hepatic necrosis with hypertonic solutions via the umbilical vein and necrotizing enterocolitis. Air embolus can be a deadly complication with umbilical catheterization.



Secure with a bridge dressing as described under umbilical artery catheterization section.



UMBILICAL VENOUS CATHETER SIZES

Catheter French Size	Infant Weight (Kg)
3.5 Fr	<1 Kg
5 Fr	1–3.5 Kg

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 to 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, post cardiopulmonary bypass, sepsis and emergency conditions associated with blood volume deficits.

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

However, there are many factors that influence CVP values, for example, cardiac performance, blood volume, vascular tone, intrinsic venous tone, increased inta-abdominal or intrathoracic pressures and vasopressor therapy. Therefore, using CVP to assess either preload or volume status of the patient may be unreliable as well as CVC location (i.e. femoral placement).

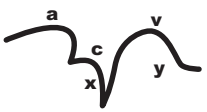
CVP INTERPRETATION (CVP RANGE 2–8 MMHG)

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 vaso-dilation (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 pressure. The *v wave* represents the atrial events during ventricular contraction — passive atrial filling — and follows the T wave on the ECG. When the atrial pressure is sufficient, the tricuspid valve opens, and the *y descent* occurs. Then the cycle repeats.

RIGHT ATRIUM



"a" = Atrial Contraction

"c" = Closure of Tricuspid Valve

"v" = Passive Atrial Filling

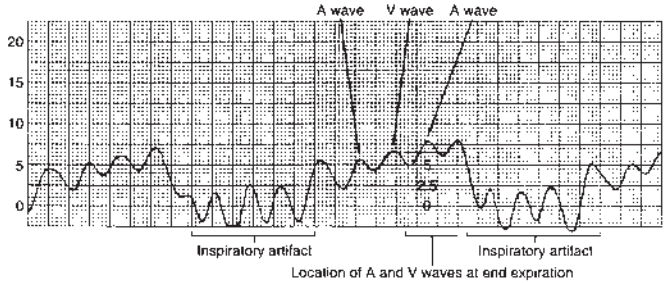
"x" = Atrial Diastole

"y" = Atrial Emptying

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

WAVEFORM 6-7

Reading CVP waveforms with spontaneous inspiratory artifact



Note: For more information www.PACEP.org

Intraosseous Access

Intraosseous

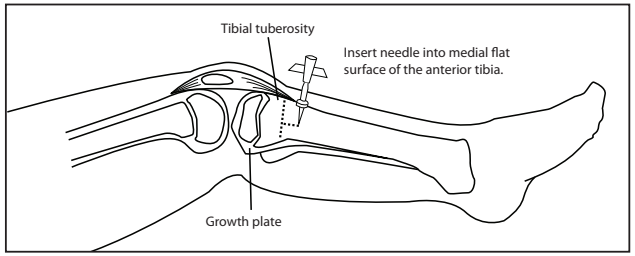
Intraosseous (IO) access can be performed rapidly for any critically ill child requiring emergent venous access. IOs offer quick access for rapid fluid resuscitation, medications and lab sampling (such as chemistries or type and crossmatch), especially in cardiac arrest or shock patients. It is instituted as a temporary form of access (12 to 24 hours) until other venous access can be obtained. A trocar needle is used to prevent bone marrow from occluding the needle. The tibia or distal femur is the preferred site. Identifying proper landmarks is important to avoid inserting needle into the growth plate of the bone.

IO Site	Landmarks
Proximal tibia	Halfway between anterior and posterior border of the tibia, 1–2 cm distal to the tibial tuberosity
Distal tibia	Proximal to medial malleolus, halfway between superior and anterior borders of the bone
Distal femur	1 cm proximal to femoral plateau
Iliac crest	
Sternum (adults only)	

Complications from IO lines are mostly related to length of time the IO is left in place. Osteomyelitis has a higher incidence with IO lines that have been left in >24 hours. Cellulitis or compartment syndrome can occur with extravasation of fluid into the surrounding tissues at the insertion site. Bone fracture or damage to the growth plate is associated with improper insertion or needle stabilization.

Intraosseous Insertion

1. Identify landmarks for insertion. Site is cleaned using a topical aseptic solution.
2. Angle the needle slightly away from the joint space or perpendicular to the bone.
3. Using the non-dominant hand, stabilize the bone that will be used. Do not use opposite hand under extremity for stabilization in case trocar needle perforates through extremity.
4. Hold the trocar needle or intraosseous device so that the hub rests in the palm with the thumb and index finger 1–2cm from the tip.
5. Use a trocar to insert the device needle into the bone. (Do not use rocking motion when inserting, as this can cause an enlargement of the opening and result in fluid extravasation.)
6. Gradually increase applied pressure with the back-and-forth "screwing" motion until a sudden decrease in resistance ("trap-door effect") is felt. Note: Do not advance the needle further to prevent puncture of both cortices which can lead to extravasation of fluid into surrounding tissues.
7. Remove the trocar. Attempt to aspirate or infuse. With proper placement, fluid should infuse without any resistance.
8. Secure the line as in the umbilical applications.



Note: If intraosseous drill device is used, consult manufacturer's directions for proper insertion.

Fluid Management

CALCULATION OF MAINTENANCE FLUID FOR PEDIATRICS [BASED ON BODY WEIGHT OR BSA]

Body Weight Method (Kg)	Fluid Requirements/ 24 hrs	Fluid Requirements/hr
<10 Kg	100 mL/Kg/24°	4 mL/Kg/hr
10–20 Kg	1000 mL + 50mL/Kg for every Kg >10Kg	2 mL/Kg/hr for every Kg >10Kg
>20 Kg	1500 mL + 20 mL/Kg for every Kg >20 Kg	1 mL/Kg/hr for every Kg >20 Kg

Note: This method may not be appropriate for neonates <14 days old.

Body Surface Area Method

(use if >10 Kg) = 1500 – 2000 mL/m^2/day

Electrolyte requirements per BSA

H_2O 1500 mL/m^2/24h

Na^+ 30–50 mEq/m^2/24h

K^+ 20–40 mEq/m^2/24h

Dehydration Calculation

Fluid deficit (L) = pre-illness wt (Kg) – illness wt (Kg)

% Dehydration = (pre-illness wt – illness wt) / pre-illness wt x 100

DEHYDRATION SEVERITY PHYSICAL SIGNS

Sign	Mild	Moderate	Severe
Pre-illness wt	5% loss	10% loss	15% loss
Skin turgor	↓	Tenting	Tenting
Mucus membranes	Dry	Very dry	Parched
Skin color	Pale	Grey	Mottled
Urinary Output	↓	↓↓	Azotemic
BP	Normal	Normal, ↓	↓↓
Heart rate	Normal, ↑	↑	↑↑
Fontanelle	Flat	Soft	Sunken
Central Nervous System	Consolable	Irritable	Lethargic, comatose

DEHYDRATION SEVERITY INDEX

Severity	Signs/Symptoms	Fluid Deficit mL/Kg	% Fluid Deficit
Minimal	Thirsty	30 mL/Kg	3%
Mild	Dry mucus membrane	30–50 mL/Kg	3–5%
Moderate	Sunken eyes, fontanel, soft globe	50–70 mL/Kg	5–7%
Moderate-severe	Skin tenting	70–120 mL/Kg	7–12%
Severe	Hypovolemic shock	>120 mL/Kg	>12%

ESTIMATED WATER & ELECTROLYTE DEFICITS IN DEHYDRATION

Type	H_2O (mL/Kg)	Na^+ (mEq/Kg)	K^+ (mEq/Kg)	Cl^- & HCO_3^- (mEq/Kg)
Isotonic (serum Na^+ 130–150 mEq/L)	100–150 mL/Kg	8–10 mEq/Kg	8–10 mEq/Kg	16–20 mEq/Kg
Hypotonic (serum Na^+ <130 mEq/L)	50–100 mL/Kg	10–14 mEq/Kg	10–14 mEq/Kg	20–28 mEq/Kg
Hypertonic (serum Na^+ >130 mEq/L)	120–180 mL/Kg	2–5 mEq/Kg	2–5 mEq/Kg	4–10 mEq/Kg

CALORIC DAILY REQUIREMENTS

Age	Daily Requirements
High risk neonate	120–125 cal/Kg
Neonate	100–120 cal/Kg
1–2 years	90–100 cal/Kg
2–6 years	80–90 cal/Kg
7–9 years	70–80 cal/Kg
10–12 years	50–60 cal/Kg

Intra-Abdominal Pressure Monitoring

Intra-Abdominal Pressure (IAP) Monitoring (Bladder Pressure Monitoring)

Intra-abdominal hypertension (IAH) can be seen in trauma patients or following abdominal surgery. IAH occurs when IAP exceeds 20 mmHg. Intra-abdominal compartment syndrome (IACS) is present when IAH exists along with associated organ dysfunction. Complications from abdominal hypertension include: ascites, capillary leak syndrome, ischemia-reperfusion injury, shock, and can be life-threatening.

SYMPTOMS AND RISK FACTORS OF INTRA-ABDOMINAL COMPARTMENT SYNDROME [IACS]

Symptoms	Risk Factors
Respiratory insufficiency (results from V/Q mismatch) Decrease preload leading to hemodynamic compromise Decrease renal function, renal failure Decrease cardiac output Splanchnic hypoperfusion	Severe penetrating or blunt abdominal trauma Ruptured aortic abdominal aneurysm Retroperitoneum hemorrage, pneumoperitoneum Neoplasm Pancreatitis Liver transplantation Massive Ascites Abdominal wall eschar Renal failure

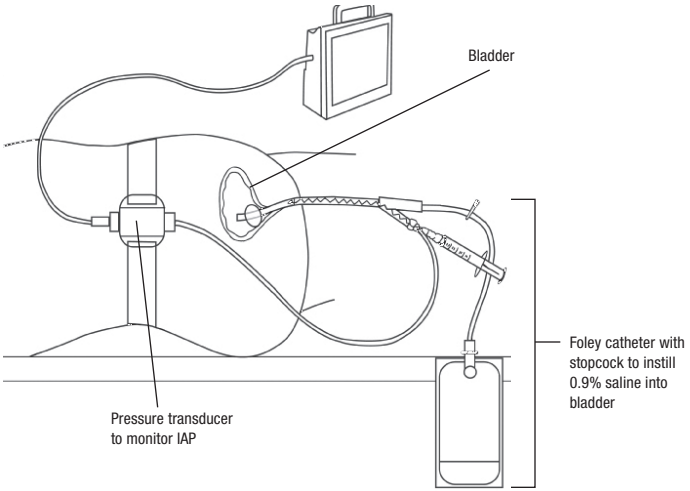
Intra-Abdominal Pressure (IAP) Monitoring Set-Up

IAP can be measured via bladder pressures with a Foley catheter and transducer.

- Connect pressure transducer to Foley catheter via 3-way stopcock
- Instill 1–2 mL/Kg 0.9% saline (maximum volume = 50–60 mL)
- Zero the transducer at the level of the symphis pubis
- Pressures are measured as mean
- Values >15 mmHg may require abdominal decompression

Abdominal decompression should be considered once IAP exceeds >20–25 mmHg and in the presence of any deterioration in pulmonary, cardiovascular, renal dysfunction and bowel ischemia.

IAP MONITORING SET-UP



Closed Sampling Systems

VAMP, VAMP Jr., and VAMP Plus (Venous Arterial Blood Management Protection System)

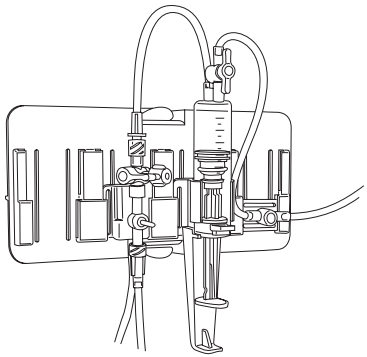
The VAMP products are closed sampling systems which provide protection against nosocomial infections, reduces blood loss, provides consistent clearing volumes, undiluted accurate blood sampling and safer reinfusion of clearing volume. The Z-site sampling port replaces stopcocks, is self-sealing, prevents residual blood build-up and is designed for needleless access.

The VAMP Jr. system is designed specifically for neonatal and pediatric patients. It is designed to require decreased volume with a smaller reservoir (3 mL) for more accurate sampling and flushing in pediatric and neonatal patients. The VAMP Jr. reservoir is designed with "cc" markings to help determine the appropriate clearing volumes and the samples required. (1 cc = mL). It is designed for connection onto umbilical, arterial and central line catheters and is also available preassembled with the TruWave disposable pressure transducers (DPTs).

The VAMP Plus system provides a large-volume reservoir with two sample sites, allowing for convenient and flexible blood sampling in both surgery and intensive care. The 12 mL reservoir design can be bracket mounted on an IV pole or used as a syringe.

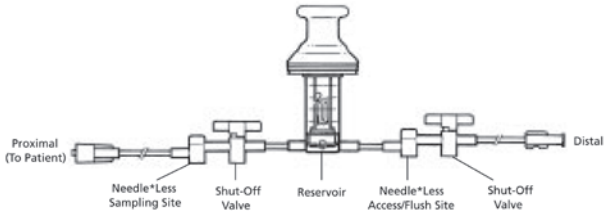
VAMP PLUS SYSTEM

12 mL reservoir



VAMP JR. SYSTEM

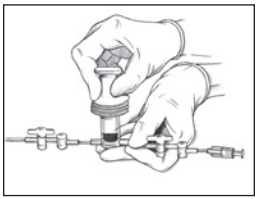
3 mL reservoir



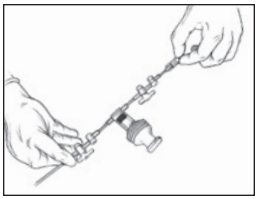
VAMP Jr. Directions for Use

Priming

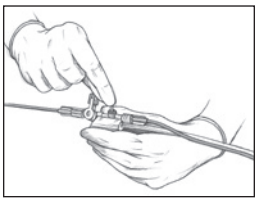
1. Open reservoir plunger to approximately 0.5 mL to facilitate flow of priming solution.



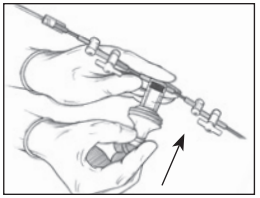
2. With the shut-off valve in the open position (parallel to the tubing), hold sampling site above the Edwards VAMP Jr. reservoir at 45° angle.



3. Provide flow by pulling Snap-Tab of the Edwards TruWave disposable pressure transducer. Slowly deliver priming solution to remove air.

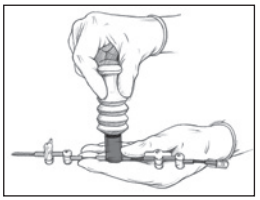


4. Close plunger and remove all air from the system. Ensure that VAMP Jr. and catheter connection is a fluid-to-fluid connection.



Drawing the Clearing Volume

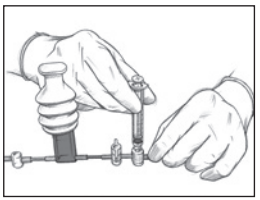
5. Close the distal shut off valve by turning the handle perpendicular to the tubing. Smoothly and evenly pull up on the reservoir plunger to draw the required amount of clearing volume consistent with the patient's clinical condition, patient's size or institution protocol. (Recommended pull rate is 1 mL every 10–15 seconds.)



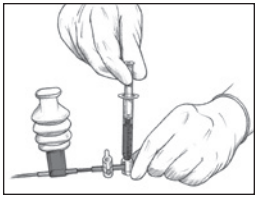
6. Close the proximal shut-off valve by turning handle perpendicular to tubing.

Drawing Blood Samples from the VAMP Jr. Needleless Sampling Site

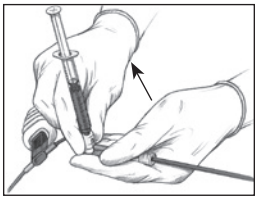
7. Swab proximal sample site. Ensure plunger is depressed to the bottom of the syringe. Attach cannula. Push cannula into proximal sample site.



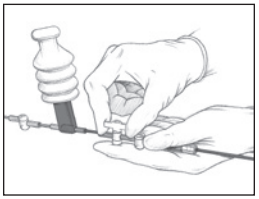
8. Slowly draw the blood sample.



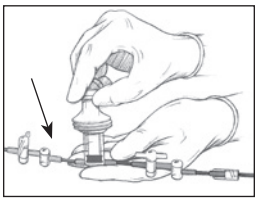
9. While holding the cannula, remove the syringe/cannula from the sampling site by pulling straight out.



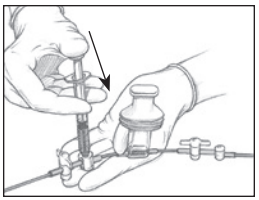
10. Open the proximal shut-off valve by turning handle parallel to tubing.



11. Slowly, smoothly, and evenly, push down on the plunger until it is fully closed. (Recommended rate is 1mL every 10–15 seconds.)



12. Fill syringe with flush solution. Attach Interlink Needleless cannula to the syringe. Ensure syringe and cannula are free of air bubbles. Swab the distal access port. Insert syringe and cannula into the distal access port. Slowly flush line per institution policy. Open the distal one-way valve.



VAMP JR. SYSTEM

VAMP Jr.	VAMP Jr. / DPT Combo Kits
VMP306 2″ proximal, 4″ distal for neonatal application	VMP306PX 30cc TruWave DPT with 2″ proximal, 4″ distal VAMP Jr. for neonatal application
VMP406 3″ proximal, 3″ distal for pediatric application	VMP406PX 3cc TruWave DPT with 3″ proximal, 3″ distal VAMP Jr. for pediatric application
VMP426 10″ proximal, 16″ distal for pediatric application	VMP426PX 3cc TruWave DPT with 10″ proximal, 16″ distal VAMP Jr. for pediatric application
VMP448 19″ proximal, 29″ distal for pediatric application	VMP448PX 3cc TruWave DPT with 19″ proximal, 29″ distal VAMP Jr. for pediatric application

VAMP SYSTEM VENOUS ARTERIAL BLOOD MANAGEMENT PROTECTION

TruWave/VAMP Combo Kits	Case Qty.	Model No.
20″ VAMP System with Arm-mount Reservoir	10	PXVMP120
60″ VAMP System with Arm-mount Reservoir	10	PXVMP160
72″ VAMP System with Arm-mount Reservoir	10	PXVMP172
84″ VAMP System with Arm-mount Reservoir	10	PXVMP184
60″ VAMP System with Pole-mount Reservoir	10	PXVMP260
72″ VAMP System with Pole-mount Reservoir	10	PXVMP272
84″ VAMP System with Pole-mount Reservoir	10	PXVMP284
60″ VAMP System with Arm-mount Reservoir on the arterial line with: • Two TruWave Disposable Pressure Transducers with two 3 mL/hr flush devices • Bifurcated IV set (macrodrip) • One set of 48″ and 12″ pressure tubing • Three 3-way stopcocks	10	PXVMP2X21
60″ VAMP System with Pole-mount Reservoir on the arterial line with: • Two TruWave Disposable Pressure Transducers with two 3 mL/hr flush devices • Bifurcated IV set (macrodrip) • One set of 48″ and 12″ pressure tubing • Three 3-way stopcocks	10	PXVMP2X22

Advanced Minimally Invasive Monitoring

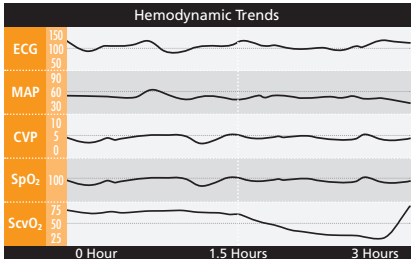
ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972

Advanced Minimally Invasive Monitoring: Venous Oximetry

Venous Oximetry Physiology and Clinical Applications

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

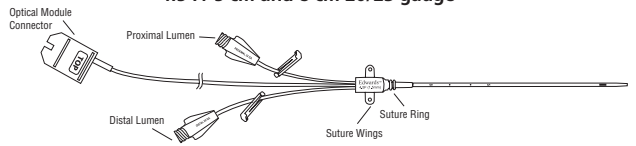
Continuous fiberoptic venous oximetry is a valuable tool for monitoring the balance between oxygen delivery and consumption at the bedside. Continuous venous oximetry is a sensitive real-time indicator of this balance, which can be applied as a global or regional indicator – with mixed venous oxygen 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.

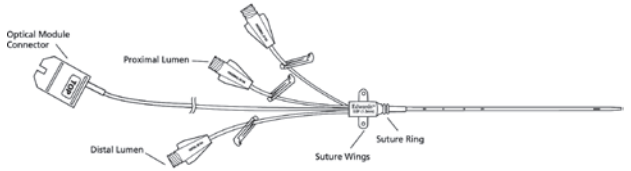
$ScvO_2$ became available in 2003 for adults on an 8.5 Fr central venous catheter platform (Edwards PreSep catheter). This capability is also available via 4.5 Fr and 5.5 Fr central venous oximetry catheters (Edwards PediaSat catheter) for pediatric use since 2007. With the tip of the PediaSat central venous catheter placed in the SVC, $ScvO_2$ can be measured and displayed on any Edwards oximetry monitor.

PEDIASAT OXIMETRY CATHETER

4.5 Fr 5 cm and 8 cm 20/23 gauge

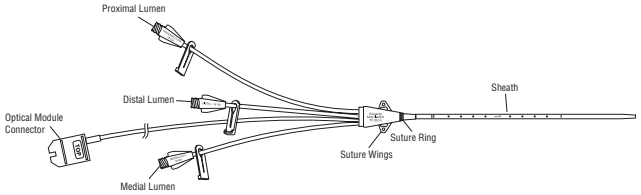


5.5 Fr 8 cm and 15 cm 18/23/23 gauge



PRESEP OXIMETRY CATHETER

8.5 Fr 16 cm and 20 cm 16/18/18 gauge



Difference Between SvO_2 and $ScvO_2$

Since SvO_2 and $ScvO_2$ are affected by the same four factors (cardiac output, hemoglobin, oxygenation/ventilation, 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 coronary sinus (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

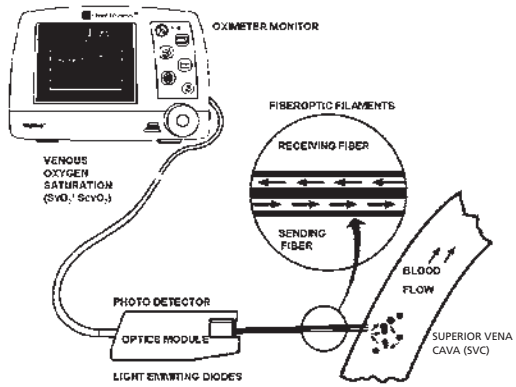
$ShvO_2$ – hepatic venous oximetry

$ScsO_2$ – coronary sinus oximetry

Continuous $ScvO_2$ Monitoring Technology

All venous oximetry is measured through reflection spectrophotometry. Light is emitted from an LED optics module 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.

Interference with $ScvO_2$ Readings

Technical issues and therapeutic interventions may affect fiberoptics. Both the distal lumen and the sending/receiving optics reside at the tip of the catheter as separate lumens. Therefore, tip position may influence signal quality index (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). However, patients with cyanotic mixing heart defects may have extremely low $ScvO_2$ levels. Therefore, it is important to establish a baseline for each patient. 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
- Over delivery of oxygen

When Change is Significant

$ScvO_2$ and SvO_2 values are not static and will fluctuate approximately ± 5%. These values may show significant changes with activities or interventions such as suctioning; however, 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 $ScvO_2$:

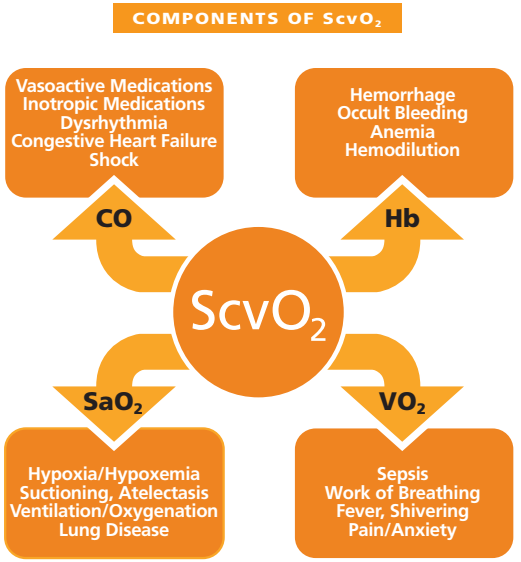
- Cardiac output
- Hemoglobin
- Arterial oxygen saturation (SaO_2)
- Oxygen consumption

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

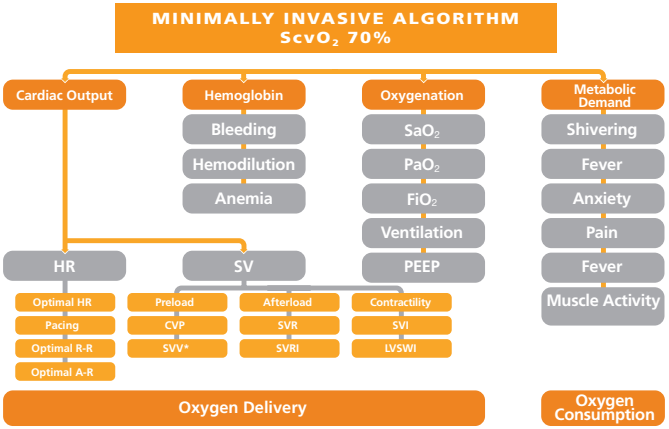
Clinical Applications of $ScvO_2$

$ScvO_2$ 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.



Using $ScvO_2$ monitoring allows the clinician to differentiate between issues of oxygen delivery versus oxygen consumption.



**SVV is an indicator of preload responsiveness.*

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 or lactate levels 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% (or above 50% in uni-ventricular post-op cardiac patients), has been proven to lead to better patient outcomes.

Insertion Recommendations

1. Right internal jugular vein is the ideal recommended insertion site.
2. Adequate dilation of the fascia and vessel is recommended for ease of insertion.
3. Rotating the catheter as it enters through the skin and fascia may also ease insertion, since distal exit port is not concentric at catheter tip.
4. If performing *In-Vivo* calibration as the initial calibration, complete calibration after catheter has been sutured in place and any rolls under neck or shoulders have been removed.

To Perform *In Vitro* Calibration:

1. Connect optical module to Vigileo monitor and allow 20 minutes for optical module to warm up.
2. Connect catheter to optical module.
3. Rotate navigation knob to oximetry frame, press to display oximetry menu.
4. Select parameters **$ScvO_2$** or **SvO_2**.
5. Select ***IN VITRO* CALIBRATION**.
6. Select **HGB** (hemoglobin) OR **HCT** (hematocrit).

a. Use default value or enter lab value if available.

7. Select **CALIBRATE**.
8. Upon completion of a successful calibration, the monitor will display the message: **"*In Vitro* Calibration OK. Insert Catheter then select Start."**
9. Remove catheter from tray.
10. Flush catheter (never flush lumens before *In Vitro* calibration).
11. Insert catheter into appropriate position.
12. Select **START**.

To Perform *In Vivo* Calibration:

1. Rotate navigation knob to highlight oximetry frame, press to display oximetry menu.
2. Confirm catheter position and SQI before performing – Press ***IN VIVO* CALIBRATION**.
3. Select **CONTINUE**.
4. Select **DRAW**, after checking for SQI of 1 or 2.
5. Slowly draw waste sample and discard. Slowly draw lab sample and send for analysis by co-oximeter.
6. Use the navigation knob to enter oximetry value and HGB or HCT.
7. Select **CALIBRATE**.

To Transport:

1. With the catheter connected to optical module, unplug optical module from Vigileo monitor and transport patient.
2. After transport, reconnect optical module to the Vigileo monitor, **WAIT 20 SECONDS**.
3. Rotate navigation knob to oximetry frame and press to display oximetry menu.
4. Select **RECALL OM DATA**.
   a. If Calibration data is greater than 24 hours old, then a new calibration must be performed.
   b. Make sure time and date match if using different monitors.

To Update Hemoglobin:

1. Rotate navigation knob to highlight oximetry frame and press to display oximetry menu.
2. Select **HGB UPDATE**.
3. Enter **HGB** or **HCT** level.
4. Select **CALIBRATE** and wait 25 seconds.
5. Press **RETURN**.

Compatibility with Philips Monitors

The Philips IntelliVue SO_2 modules are compatible with Edwards Lifesciences' Oximetry and Swans-Ganz catheters. Consult Philips for additional information and directions for use with Philips' bedside monitors. Philips' VueLink cables can also be used to slave $ScvO_2$ / SvO_2 values from Vigileo and Vigilance monitors to bedside Philips monitors.

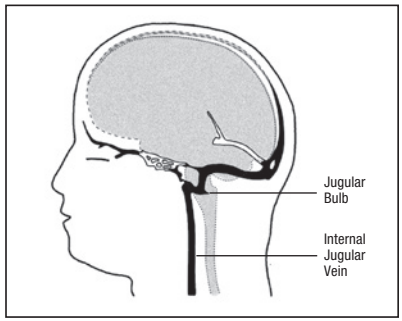
Jugular Bulb

Venous Oximetry Monitoring In the Jugular Bulb (SjO_2)

Jugular venous oximetry does not directly measure cerebral blood flow (CBF), but can provide information about the adequacy of cerebral blood flow in relation to cerebral metabolic demands. Arterio-jugular oxygen content difference ($AjDO_2$) is also reflective of the adequacy of cerebral blood as it relates to cerebral metabolic demands and can be calculated as the difference of oxygen content between arterial and jugular venous samples. Although continuous venous oxygen saturation is measured in one jugular bulb, it can be assumed that it reflects the global cerebral blood flow over hemispherical blood flow.

Jugular bulb oximetry can be used to monitor cerebral oxygenation and tissue perfusion in patients who have experienced traumatic brain injury as a result of trauma, neurological disease processes, post-operative neurosurgical procedures or following an anoxic episode such as cardiac arrest. Normal values are 65-75%. It has been documented that with intermittent sampling of two or more occurrences with values <50% has been associated with poor neurological outcomes.

The catheter is inserted percutaneously using the modified Seldinger technique. The needle enters the vein lateral to the carotid artery at the level of the cricoid cartilage. It is then directed upward towards the external meatus. Catheter tip should reside at the level of the first or second vertebrae and tip placement verified with lateral cervical spine x-ray. A continuous infusion of ~2 mL/hr or less is recommended to prevent thrombus formation. Rapid aspiration during sampling can result in inaccurate values from extracranial blood.



Oximetry Messages and Troubleshooting

OXIMETRY FAULTS AND ALERTS

Oximetry Fault Messages	Possible Cause(s)	Suggested Action(s)
Light Range	• Poor optical module/ catheter connection • Debris or film obstructing optical module/catheter connection lens • Optical module malfunction • Catheter kinked or damaged	• Verify secure optical module/catheter connection • Clean optical module/catheter connectors with 70% isopropyl alcohol and swab, let air dry and recalibrate • Replace catheter if damage is suspected and recalibrate
OM Disconnected	• Optimal module connection at monitor not detected • Bent or missing optical module connector pins	• Verify secure optical module/catheter connection • Check optical module cable connector for bent/missing pins
OM Memory	• Optimal module memory malfunction	• Change optical module and recalibrate
Value Out of Range	• Incorrectly entered oximetry, HGB or Hct values • Incorrect HGB units of measure • Calculated oximetry value if outside of the 0–99% range	• Verify correctly entered oximetry, HGB and Hct values • Verify correct HGB unit of measure • Obtain updated oximetry lab values and recalibrate
Red/IR Transmit	• Debris or film obstructing optical module/catheter connection lens • Optical module malfunction	• Clean module/catheter connections with 70% isopropyl alcohol and swab, let air dry and recalibrate • Change optical module and recalibrate
OM Temperature	• Optical module malfunction	• Change optical module and recalibrate
Oximetry Not Available	• Internal system malfunction	• Power monitor off and on to restore system • If problem persists, contact Edwards Technical Support
Oximetry Alert Messages	**Possible Cause(s)**	**Suggested Action(s)**
SQI = 4	• Low blood flow at catheter tip or catheter tip against vessel wall • Significant changes in HGB/Hct values • Catheter tip clotted • Catheter kinked or damaged	• Verify proper catheter position. For SvO_2, verify proper catheter position in the pulmonary artery ▪ Confirm wedge pressure balloon inflation volume of 1.5 mL (for SvO_2 only) ▪ Confirm appropriate catheter placement for patient's height, weight and insertion site ▪ Consider chest x-ray for evaluation of proper placement • Aspirate then flush distal lumen per hospital protocol • Update HGB/Hct values using Update function • Check catheter for kinking and recalibrate • Replace catheter if damage is suspected and recalibrate

OXIMETRY WARNINGS

Oximetry Warning Messages	Possible Cause(s)	Suggested Action(s)
In Vitro Calibration Error	• Poor optical module and catheter connection • Calibration cup wet • Catheter kinked or damaged • Optical module malfunction • Catheter tip is not in catheter package calibration cup	• Verify secure optical module/catheter connection • Straighten any visible kinks; replace catheter if damage is suspected • Change optical module and recalibrate • Verify catheter tip is securely seated in calibration cup • Perform *in vivo* calibration
Unstable Signal	• Changing oximetry, HGB/Hct, or unusual hemodynamic values	• Stabilize patient per hospital protocol and perform *in vivo* calibration
Wall Artifact or Wedge Detected	• Low blood flow at catheter tip • Catheter tip clotted • Catheter tip wedged in vessel or against vessel wall	• Aspirate then flush distal lumen per hospital protocol • Verify proper catheter position. For SvO_2, verify proper catheter position in the pulmonary artery ▪ Confirm wedge pressure balloon inflation volume of 1.5 mL (for SvO_2 only) ▪ Confirm appropriate catheter placement for patient's height, weight and insertion site ▪ Consider chest x-ray for evaluation of proper placement • Perform *in vivo* calibration

OXIMETRY GENERAL TROUBLESHOOTING

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

VIGILEO MONITOR PRODUCT SPECIFICATIONS

Color Display	5.2 in. (132.5 mm) x 3.9 in. (99.4 mm) TFT | 640 x 480 pixels
Power/Electrical	**AC Mains:** 100 – 240 VAC, 50/60 Hz | 1A maximum consumption
Trend Range	0.1 – 72 hours
Size	**H:** 7.3 in. (185.4 mm) | **W:** 10.7 in. (271.8 mm) | **D:** 8.4 in. (213.4 mm)
Weight	6 pounds (2.73 kg) | IV pole-mount capability
Bi-directional Patient Monitor Communications	**Analog input/output (selectable voltage)** **Input:** 0 to 1V, 0 to 5V, 0 to 10V | **Output:** 0 to 1V, 0 to 10V **Digital input/output, serial communication interface (RS232)** Maximum data rate — 57.6 kilobaud
Printer Communications Medial	**USB Port:** V1.1-compatible type A connector

PEDIASAT OXIMETRY CATHETER* SPECIFICATIONS

Model Number	Lumens	Length (cm)	Size Fr (mm)	Continuous $ScvO_2$	Lumen Size Gauge (mm)			Minimum Guidewire Size inch (mm)	AMC Thromboshield**
					Distal	Proximal	Medial		
XT245HS	2	5	4.5 (1.50)	Yes	20 (0.89)	23 (0.64)	-	0.018 (0.46)	Yes
XT248HS	2	8	4.5 (1.50)	Yes	20 (0.89)	23 (0.64)	-	0.018 (0.46)	Yes
XT358HS	3	8	5.5 (1.83)	Yes	18 (1.24)	23 (0.64)	23 (0.64)	0.025 (0.64)	Yes
XT3515HS	3	15	5.5 (1.83)	Yes	18 (1.24)	23 (0.64)	23 (0.64)	0.025 (0.64)	Yes
The following kits are available in the U.S. only.									
XT245HK	2	5	4.5 (1.50)	Yes	20 (0.89)	23 (0.64)	-	0.018 (0.46)	Yes
XT245K	2	5	4.5 (1.50)	Yes	20 (0.89)	23 (0.64)	-	0.018 (0.46)	
XT248HK	2	8	4.5 (1.50)	Yes	20 (0.89)	23 (0.64)	-	0.018 (0.46)	Yes
XT248K	2	8	4.5 (1.50)	Yes	20 (0.89)	23 (0.64)	-	0.018 (0.46)	
XT358HK	3	8	5.5 (1.83)	Yes	18 (1.24)	23 (0.64)	23 (0.64)	0.025 (0.64)	Yes
XT358K	3	8	5.5 (1.83)	Yes	18 (1.24)	23 (0.64)	23 (0.64)	0.025 (0.64)	
XT3515HK	3	15	5.5 (1.83)	Yes	18 (1.24)	23 (0.64)	23 (0.64)	0.025 (0.64)	Yes
XT3515K	3	15	5.5 (1.83)	Yes	18 (1.24)	23 (0.64)	23 (0.64)	0.025 (0.64)	

*PediaSat catheters are designed for use with Edwards oximetry monitors and optical cables. **All model numbers with an "H" contain AMC Thromboshield, an antibacterial heparin coating which decreases viable microbe count on the surface of product during handling and placement. Many catheters are available with or without heparin coating.

PRESEP CENTRAL VENOUS OXIMETRY CATHETER* SPECIFICATIONS

Model Number	Lumens	Length (cm)	Size Fr (mm)	Continuous $ScvO_2$	Lumen Size Gauge (mm)			Recommended Dilator Fr (mm)	Minimum Guidewire Size inch (mm)	AMC Thromboshield**
					Distal	Proximal	Medial			
X3820HK	3	20	8.5 (2.83)	Yes	15 (1.77)	18 (1.33)	18 (1.33)	10.5 (3.5)	0.32 (0.8)	Yes
X3820K	3	20	8.5 (2.83)	Yes	15 (1.77)	18 (1.33)	18 (1.33)	10.5 (3.5)	0.32 (0.8)	
X3820HS***	3	20	8.5 (2.83)	Yes	15 (1.77)	18 (1.33)	18 (1.33)	10.5 (3.5)	0.32 (0.8)	Yes

*PreSep catheters are designed for use with Edwards oximetry monitors and optical cables. **All model numbers with an "H" contain AMC Thromboshield, an antibacterial heparin coating which decreases viable microbe count on the surface of product during handling and placement. Many catheters are available with or without heparin coating.

PEDIASAT INFUSION SPECIFICATIONS

Model	PediaSat 4.5 Fr, 5 cm	PediaSat 4.5 Fr, 8 cm	PediaSat 5.5 Fr, 8 cm	PediaSat 5.5 Fr, 15 cm
Usable length (cm)				
Port exit distance from tip				
Proximal	1 cm	1 cm	2 cm	2 cm
Medial	N/A	N/A	1 cm	1 cm
Lumen volume (mL)				
Distal	0.2 mL	0.2 mL	0.2 mL	0.2 mL
Medial	N/A	N/A	0.2 mL	0.2 mL
Proximal	0.1 mL	0.1 mL	0.2 mL	0.2 mL
Infusion rates (mL/hr max)				
Distal	950 mL/hr	635 mL/hr	1780 mL/hr	1280mL/hr
Medial	N/A	N/A	730 mL/hr	440 mL/hr
Proximal	830 mL/hr	460 mL/hr	780 mL/hr	440 mL/hr

Swan-Ganz Catheters

Advanced and Standard Technology

ADVANCING CRITICAL CARE
THROUGH SCIENCE-BASED EDUCATION
SINCE 1972

The Swan-Ganz Pulmonary Artery Catheter

Standard Swan-Ganz Catheter

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

Placing Swan-Ganz pulmonary artery catheters in infants and pediatrics is done less frequently than in adults, in part due to higher difficulty gaining access, patient size, variable anatomy and physician experience. Pulmonary artery catheters may be placed under direct visualization at the end of cardiopulmonary bypass surgery for post-operative monitoring.

PULMONARY ARTERY CATHETER SIZES

Weight (Kg)	Suggested size (Fr)
<10 Kg	2/3 Fr
10–18 Kg	4/5 Fr
>18 Kg	6/7 Fr

Pulmonary artery catheter sizes commonly used in pediatrics are 4 Fr and 5 Fr for children between 10-18 Kg and 6 Fr and 7 Fr for children weighing more than 18 Kg. For children who are less than 10 Kg, there are also 2 Fr and 3 Fr that are double lumen, non-balloon tipped with a distal port and thermistor. Complications with intracardiac placed lines are that they can be mistakenly sutured to the cardiac muscle.

Complications with removal of an intracardiac placed pulmonary artery catheter can be risk of hemorrhage. Having blood on hand and a thoracic surgeon readily available may be necessary in case there is a need for an emergent thoracotomy.

The standard Swan-Ganz catheter measures:

- Right Heart Pressures:
  - Right atrial pressure (RAP)
  - Pulmonary artery pressures (PAP)
    - Pulmonary artery systolic (PAS)
    - Pulmonary artery diastolic (PAD)
    - Pulmonary artery mean (PAM)
    - Pulmonary artery occlusion pressure (PAOP)
- Thermodilution Cardiac Output:
  - Edwards CO-Set iced, closed bolus injectate system*
  - CO-Set room temperature, closed bolus injectate system*
- Pulmonary Artery Blood Sampling For Laboratory Analysis:
  - Mixed venous blood oxygen saturation (SvO_2)**
  - Serial measurements of right heart chamber oxygen saturations**
- Additional available features:
  - Venous infusion port (VIP)
  - Paceport catheter – temporary right atrial and/or ventricular trans-venous pacing**
  - Angiographic catheters – designed for high pressure dye injections used in radiographic examinations**

Applications of standard Swan-Ganz catheters

- Right heart catheterization for right heart pressure measurements (PAS, PAD, PAOP) for diagnostic purposes
- Single point-in-time calculations of cardiac output using bolus thermodilution for diagnosing cardiac function
- Single mixed venous laboratory blood draws via the catheter to assess SvO_2 and the balance between oxygen delivery and consumption
- Serial right heart chamber venous blood draws to measure oxygen saturations indicating left to right intra-cardiac shunts
- Pulmonary artery angiography (available in 5 Fr, 6 Fr, 7 Fr and 8 Fr sizes)
- Temporary transvenous ventricular or atrial-ventricular pacing (5 Fr size only)

**Recommended only for adult Swan-Ganz catheters. 10 mL syringe is used for injectate for thermodilution cardiac output.*
***Note only available in adult sized catheters (7.5 Fr and 8.5 Fr)*

Advanced Technology Swan-Ganz Catheter

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

The advanced technology Swan-Ganz catheter measures:

- Right heart pressures:
  - Right atrial pressure (RAP)
  - Pulmonary artery pressures (PAP)
    - Pulmonary artery systolic (PAS)
    - Pulmonary artery diastolic (PAD)
    - Pulmonary artery mean (PAM)
    - Pulmonary artery occlusion pressure (PAOP)
- Thermodilution Cardiac Output:
  - CO-Set iced, closed bolus injectate system
  - CO-Set room temperature, closed bolus injectate system
- Pulmonary Artery Blood Sampling For Laboratory Analysis:
  - Mixed venous blood oxygen saturation (SvO_2)
- SvO_2 – mixed venous oxygen saturation is continuously measured through fiberoptic reflectance technology and is a global indicator of the balance between oxygen delivery and consumption
- CCO – continuous cardiac output, measured through advanced thermodilution technology, is a key component of oxygen delivery
- RVEF – right ventricular ejection fraction is also continuously measured through advanced thermodilution technology and algorithm analysis indicates right ventricular function and filling which can be used to help assess right heart contractility
- RVEDV – right ventricular end diastolic volume is continuously calculated by dividing stroke volume (mL/beat) by RVEF (%) giving a key indicator of preload
- SVR and SVRI – continuous systemic vascular resistance can be calculated when the Vigilance monitor obtains continuous MAP and CVP from the bedside physiologic monitor

Applications of advanced technology Swan-Ganz catheters

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

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

- Maximum amount of diagnostic information with same invasive procedure
- Continuous assessment of DO_2 /VO_2 balance with SvO_2 monitoring
- Continuous assessment of adequacy of CO by assessing DO_2 /VO_2 balance with SvO_2 monitoring
- Continuous assessment of components of stroke volume (preload, afterload, and contractility) (RVEDV, SVR, RVEF and SVI)
- Mitigation of user error in association with wedge procedure/calculation through automated alternative preload parameter (RVEDV)
- Mitigation of pulmonary artery rupture possibility associated with wedge procedure by providing automated preload parameter (RVEDV)
- Mitigation of inappropriate therapy due to miscalculation of PAOP by using automated preload parameter (RVEDV)
- Mitigation of inappropriate preload assessment secondary to changes in ventricular compliance affecting PAD or PAOP
- Mitigation of iatrogenic infection risk from bolus injections
- Mitigation of cardiac output error with CCO automation through elimination of bolus cardiac output user error
- Increased accuracy of cardiac output calculations, elimination of ventilator cycle and thermal noise effect

Applications and Contraindications

Clinical applications for Swan-Ganz pulmonary artery catheters:

- Acute Respiratory Distress Syndrome (ARDS)
- Extensive burns
- Cardiac surgery
- Significant cardiac tamponade
- Significant cardiomyopathy
- Significant constrictive pericarditis
- Drug intoxication
- Significant intra- or extra-vascular fluid shifts
- At risk for hemorrhage
- Intra- and post-operative high-risk surgery management
- Pulmonary edema
- Pulmonary embolism
- Pulmonary hypertension
- Severe sepsis
- Presence of or at risk for cardiogenic shock
- Presence of or at risk for distributive shock
- Presence of or at risk for hemorrhagic shock
- Presence of or at risk for obstructive shock
- Shock of unknown etiology
- Shock unresponsive to attempts at resuscitation
- Severe trauma

Relative contraindications for Swan-Ganz pulmonary artery catheterization:

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

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

Vigilance Monitors

The Edwards Vigilance and Vigilance II monitors are used with the advanced technology Swan-Ganz catheters to graphically and numerically display key flow parameters as well as the components of stroke volume. The Vigilance monitor houses two distinct technologies:

(1) continuous fiberoptic venous oximetry (SvO_2), and

(2) continuous thermodilution cardiac output. CCO and RVEF are measured values while RVEDV, SVR, SVRI, and stroke volume are calculated when the Vigilance monitor obtains heart rate (HR), mean arterial pressure (MAP), and central venous pressure (CVP) from the bedside physiologic monitor.

Vigileo Monitor

The Edwards Vigileo monitor can be used with the continuous venous oximetry on the Swan-Ganz pulmonary artery catheters. It houses the technology for continuous fiberoptic venous oximetry (SvO_2).

Selected Swan-Ganz Catheter Specifications

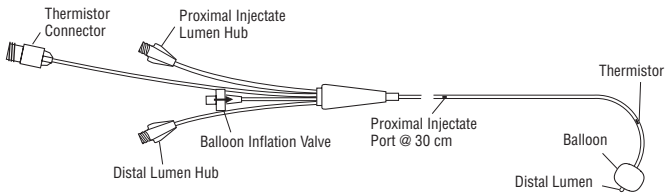
Model Numbers	131	132	177	831/834	931/991	139	744/756	744/777
Distance From Tip Port Exits (cm)								
Proximal Injectate	30	15	26	30	30	26	26	26
Proximal Infusion			30	31	NA/27	30	NA/30	NA/30
RV Infusion				NA/19	19			
Thermal Filament			14-25			14-25	14-25	14-25
Lumen Volume (mL)								
PA/Distal	1.02	0.64	0.96	0.86/0.89	0.88/0.93	0.96	0.96/0.90	0.96/0.90
Proximal Injectate	0.81	0.57	0.8	0.86/0.75	0.89/0.70	0.80	0.95/0.85	0.95/0.85
Proximal Infusion			0.95	0.87/0.97	NA/1.07	0.95	NA/1.10	NA/1.10
RV Infusion/Pacing (without probe)				-/0.93	NA/1.13			
Infusion Rates (mL/hr)								
PA/Distal			320	750/456	289/324	320	320/325	320/325
Proximal Injectate			400		724/459	400	898/562	898/562
RA Infusion/Pacing			898	NA/910	NA/66 with probe NA/811 without probe	898	NA/988	NA/988
RV Infusion/Pacing					37/56 with probe 641/757 without probe			
Natural Frequency Response/Amplitude Ratio (hz/ar)								
PA/Distal	37/2.9:1	34.0/2.1:1	25/2.1:1	34/2.6:1 33/2.6:1	33.2/2.8:1 31/2.4:1	25/2.1:1	25/2.1:1 26/2.1:1	25/2.1:1 26/2.1:1
Proximal Injectate	48/3.3:1	41.3/2.1:1	33/2.5:1	47/3.1:1 37/2.4:1	43.093.2:1 44/2.7:1	33/2.5:1	45/2.7:1 40/2.6:1	45/2.7:1 40/2.6:1
Proximal Infusion			45/2.7:1	47/3.2:1 41/2.7:1	41.3/3.4:1 46/3.2:1	45/2.7:1	NA 40/2.5:1	NA 40/2.5:1
RV Infusion/Pacing				NA 28/2.3:1	NA 49/3.4:1			

Standard Swan-Ganz Catheters

Model 131

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

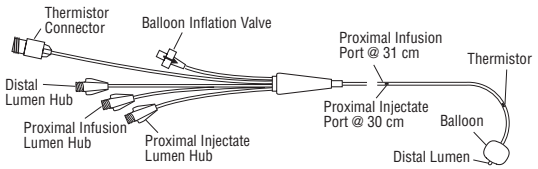
MODEL 131



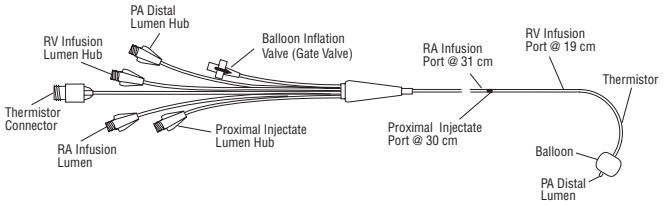
Swan-Ganz Thermodilution Catheter with Venous Infusion Port Models 831 and 834

These models have the same capability as model 131, but these venous infusion catheters provide additional lumens that exit either in the RA or both RA and RV, depending on the type of catheter. Clinical indications include those when central circulation access is needed for multiple volume and solution infusions. Intra-atrial or intra-ventricular pressure monitoring can also be obtained with these additional lumens.

MODEL 831



MODEL 834



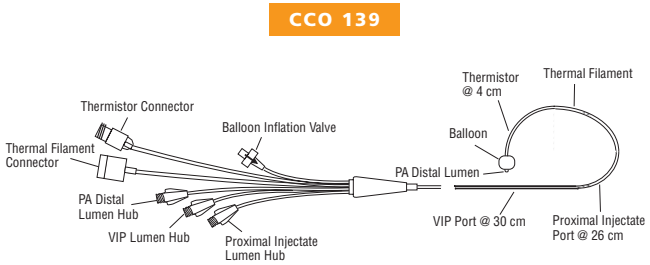
Additional RA lumen and RV lumen exits at 19 cm from tip to assure precise RV pressure monitoring.

Advanced Swan-Ganz Catheters

Advanced technology Swan-Ganz catheters combine the same basic features of the original Swan-Ganz thermodilution catheter as well as advanced continuous monitoring parameters. The balance between oxygen delivery and consumption can be continuously assessed through fiberoptic measurements of mixed venous oxygen saturation (SvO_2).*

Swan-Ganz Continuous Cardiac Output (CCO) Model 139

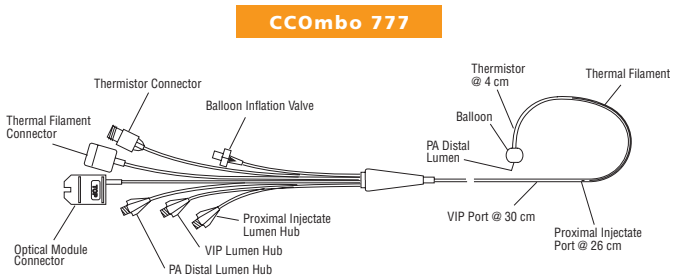
This advanced technology Swan-Ganz catheter combines the same basic features of the original Swan-Ganz thermodilution catheter as well as continuous thermodilution cardiac output measurements (CCO), a primary determinate of oxygen delivery (DO_2). Advanced technology Swan-Ganz catheters must be used in conjunction with a Vigilance series monitor. Systemic vascular resistance (SVR) can be continuously measured and displayed when the Vigilance monitor is interfaced with the bedside monitor to obtain mean arterial pressure (MAP) and central venous pressure (CVP).



For complete descriptions and indications for use of the Edwards' Advanced Technology Swan-Ganz Catheters, please consult the Quick Guide to Cardiopulmonary Care at www.Edwards.com.

Swan-Ganz CCOmbo – Volumetric (SvO_2, CCO, RVEF, RVEDV) Models 774, 777

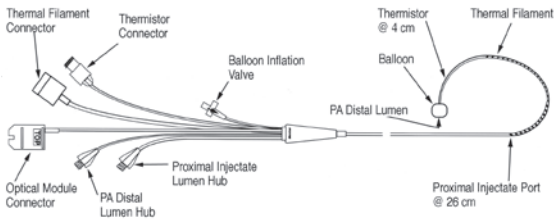
These advanced technology Swan-Ganz catheters combine the same basic features of the original Swan-Ganz thermodilution catheter as well as advanced continuous monitoring parameters. The balance between oxygen delivery and consumption can be continuously assessed through fiberoptic measurements of mixed venous oxygen saturation (SvO_2) monitoring, as well as continuous thermodilution cardiac output measurements (CCO), a primary determinate of oxygen delivery (DO_2). It also allows the further evaluation of the components of stroke volume (SV) through continuous monitoring of right ventricular end diastolic volume (RVEDV) and continuous monitoring of right ventricular ejection fraction (RVEF). Advanced technology Swan-Ganz catheters must be used in conjunction with a Vigilance series monitor. Systemic vascular resistance (SVR) can be continuously measured and displayed when the Vigilance monitor is interfaced with the bedside monitor to obtain mean arterial pressure (MAP) and central venous pressure (CVP). The heart rate from the bedside monitor must be continuously sent to the Vigilance monitors for volumetric measurements of RVEDV and RVEF.



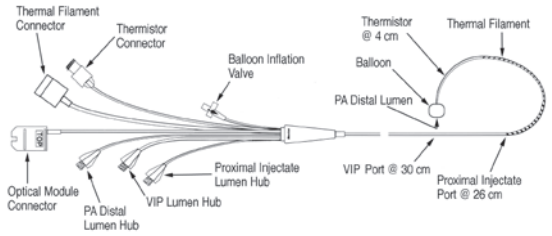
Swan-Ganz CCOmbo and CCOmbo/VIP (SvO_2 and CCO) Models 744 and 746

These advanced technology Swan-Ganz catheters combine the same basic features of the original Swan-Ganz thermodilution catheter as well as advanced continuous monitoring parameters. The balance between oxygen delivery and consumption can be continuously assessed through fiberoptic measurements of mixed venous oxygen saturation (SvO_2) monitoring, as well as continuous thermodilution cardiac output measurements (CCO), a primary determinate of oxygen delivery (DO_2). Advanced technology Swan-Ganz catheters must be used in conjunction with a Vigilance series monitor. Systemic vascular resistance (SVR) can be continuously measured and displayed when the Vigilance monitor is interfaced with the bedside monitor to obtain mean arterial pressure (MAP) and central venous pressure (CVP). A venous infusion port (VIP) is also available for intravenous medication delivery.

CCOmbo 744



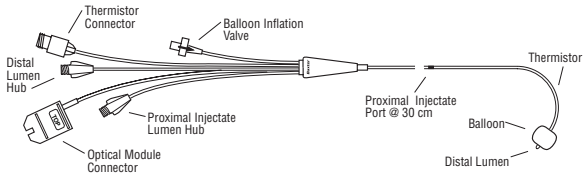
CCOmbo 746



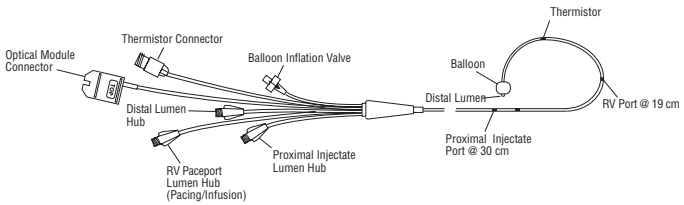
Swan-Ganz Mixed Venous Oximetry (SvO_2) Models 741 and 780

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

SvO_2 741



SvO_2 780



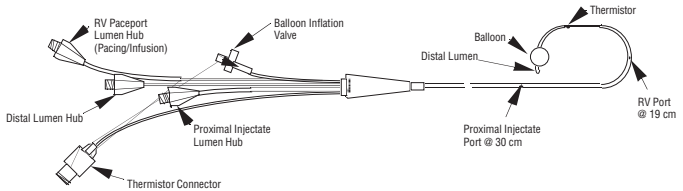
Swan-Ganz Paceport TD Catheters
Models 931 and 991

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

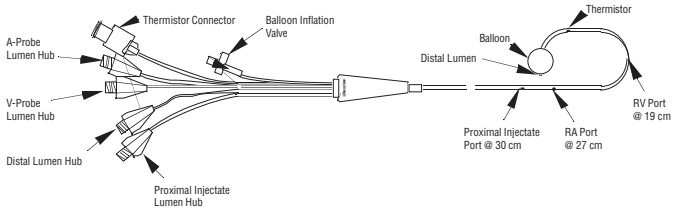
Temporary atrial, ventricular, or atrioventricular pacing can be instituted with the use of the Chandler Transluminal V-Pacing probe and atrial J pacing probe.

The additional lumens (RV lumen exits at 19 cm from the tip, RA exits at 27 cm) can also be used for pressure monitoring of their respective chambers or for additional fluid infusions.

931 PACEPORT



991 A-V PACEPORT



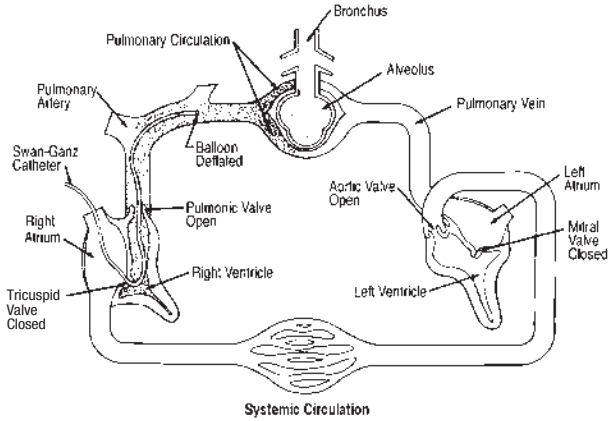
Physiological Basis for Pulmonary Artery Pressure Monitoring

Ventricles in Systole

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

VENTRICULAR SYSTOLE

RVSP = PASP



Ventricles in Diastole

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

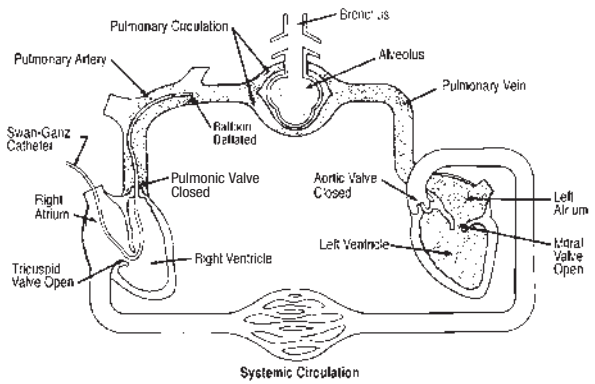
With the balloon still deflated, pulmonary artery diastolic pressure (PADP) is recorded. After the closure of the pulmonic valve, the right ventricle continues to relax. This causes a lower diastolic pressure in the right ventricle than in the pulmonary artery. RVEDP is less than PADP.

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

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

VENTRICULAR DIASTOLE

RAP = RVEDP
RVEDP < PADP
PADP ≈ LAP ≈ LVEDP



Ventricles in Diastole: Catheter Wedged

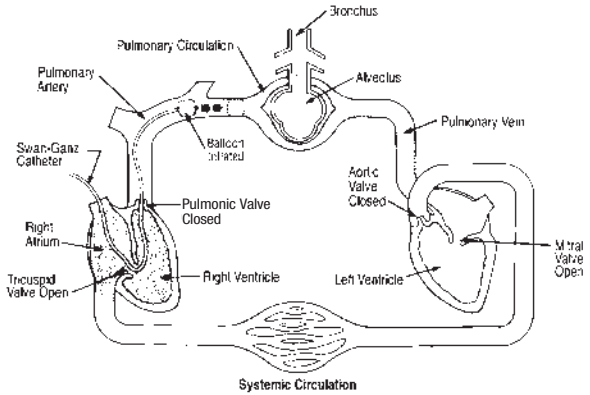
By inflating the balloon, the catheter floats downstream into a smaller branch of the pulmonary artery. Once the balloon lodges, the catheter is considered "wedged". It is in this wedge position that right sided and PA diastolic pressures are effectively occluded.

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

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

VENTRICULAR DIASTOLE

PAOP ≈ LAP ≈ LVEDP

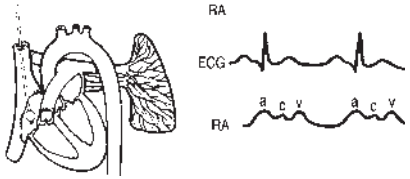


Normal Insertion Pressures and Waveform Tracings

Right Atrial/Central Venous Pressure (RA/CVP)

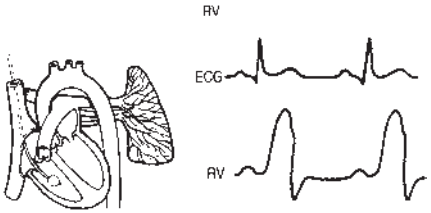
2–8 mmHg
Mean 4 mmHg

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



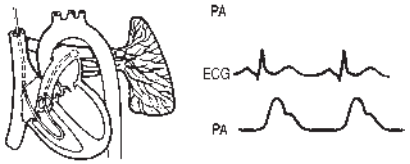
Right Ventricular

Systolic Pressure (RVSP)
20–30 mmHg
Diastolic Pressure (RVDP)
2–8 mmHg



Pulmonary Artery

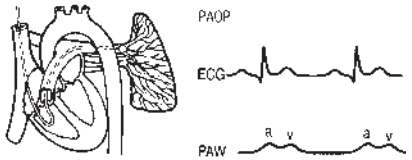
Systolic Pressure (PASP)
20–30 mmHg
Diastolic Pressure (PADP)
4–12 mmHg
Mean Pressure (MPAP)
7–18 mmHg



Pulmonary Artery Occlusion Pressure (PAOP)

Mean 4–12 mmHg

a = atrial systole
v = atrial filling, ventricle systole



Abnormal Waveform Chart

PULMONARY ARTERY WAVEFORMS

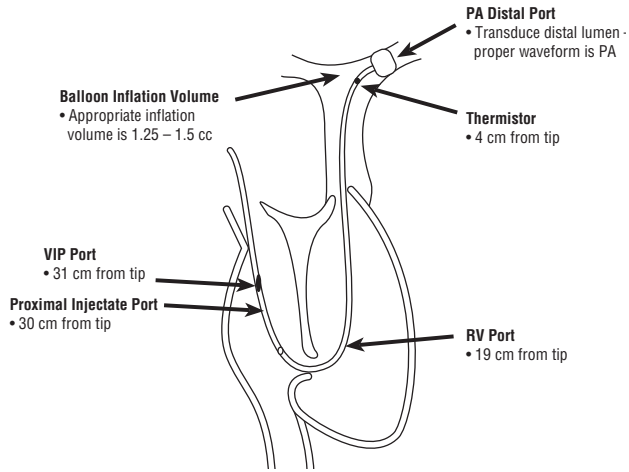
Elevated systolic pressure	Pulmonary disease Increased blood flow, left to right shunt Increased pulmonary vascular resistance
Elevated diastolic pressure	Left heart failure Intravascular volume overload Mitral stenosis or regurgitation
Reduced systolic and diastolic pressure	Hypovolemia Pulmonic stenosis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

PULMONARY ARTERY WEDGE/LEFT ATRIAL WAVEFORM

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

Swan-Ganz Catheter Port Locations and Functions

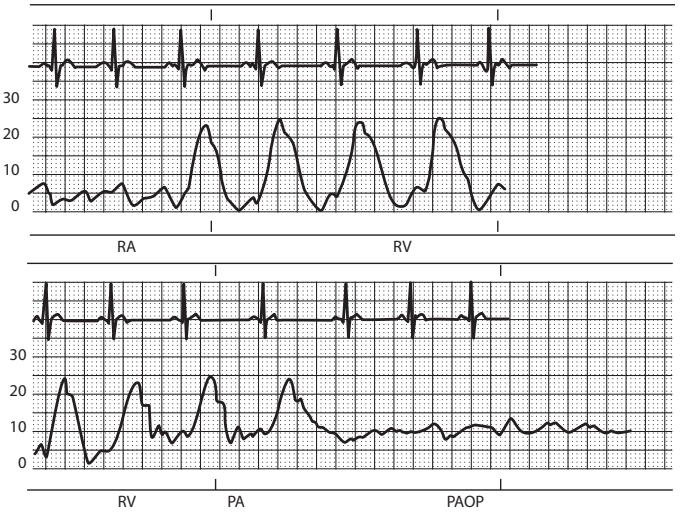
Location	Color	Function
Distal	Yellow	Monitors PA pressures
Venous Infusion Port (VIP)	White	Additional RA lumen for fluid infusion
Proximal	Blue	Monitors RA pressures, used for cardiac output injectate fluid
Balloon Gate Valve	Red	Syringe used to inflate balloon for placement and obtaining wedge values
Thermistor Connector	Yellow	Measures blood temperature 4 cm from distal tip



Insertion Techniques for the Swan-Ganz Catheter

1. Before insertion of the Swan-Ganz catheter, prepare the pressure monitoring system for use according to the institution's policies and procedures.
2. Insert the catheter following recommended guidelines and advance the catheter towards the thorax.
3. Once the catheter tip has exited the introducer sheath (approximately 15 cm) and reached the junction of the superior or inferior vena cava and right atrium, the balloon is inflated with CO_2 or room air to the full volume indicated on the catheter shaft and gate valve is locked. This position can be noted when respiratory oscillations are seen on the monitor screen.
4. Catheter advancement to the PA should be rapid, since prolonged manipulation can result in loss of catheter stiffness. The Swan-Ganz catheter is made of polyvinyl chloride (PVC) material designed to soften *in vivo*. With prolonged insertion times, a "softer" catheter may cause coiling in the RV or difficulties in catheter advancement.
5. Once the wedge position has been identified, the balloon is deflated by unlocking the gate valve, removing the syringe and allowing the back pressure in the PA to deflate the balloon. After balloon deflation, reattach the syringe to the gate valve. The gate valve is typically only placed in the locked position during catheter insertion.
6. To reduce or remove any redundant length or loop in the right atrium or ventricle, slowly pull the catheter back 2–3 cm. Then reinflate the balloon to determine the minimum inflation volume necessary to obtain a wedge pressure tracing. The catheter tip should be in a position where the full or near-full inflation volume (0.5–1.5 mL) produces a wedge pressure tracing.

Swan-Ganz Catheter Insertion Waveforms



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

Catheter Insertion Distance Markings*

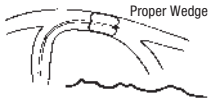
Location	Distance to VC/RA Junction	Distance to PA
Internal Jugular	15 to 20	40 to 55
Subclavian Vein	10 to 15	35 to 50
Femoral Vein	30	60
Right Antecubital Fossa	40	75
Left Antecubital Fossa	50	80

**measured in cm in adults*

Note: Catheter markings occur every 10 cms and are denoted by a thin black ring. 50 cm markings are denoted by a thick black ring. Catheter must exit introducer sheath before inflating balloon.

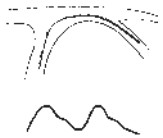
Continuous Pulmonary Artery Pressure Monitoring

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



Full inflation with 0.5–1.5 mL inflation volume. Appropriate "a" and "v" waves noted.

Overinflation of balloon. Note waveform rise on screen.



Catheter too distal. Overdamping of tracing.

Catheter spontaneous wedging. Wedge type tracing with balloon deflated.

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

1. Keep catheter tip centrally located in a main branch of the pulmonary artery

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

2. Anticipate spontaneous catheter tip migration toward the periphery of the pulmonary bed

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

post-bypass period. After termination of bypass, the catheter may require repositioning. Check the distal pulmonary artery tracing before inflating the balloon.

3. Exercise caution when inflating the balloon

- If "wedge" is obtained at volumes less than previous volumes required, pull the catheter back to a position where the full volume (0.5–1.5 mL) produces a wedge pressure tracing.
- Check the distal pressure waveform before inflating the balloon. If the waveform appears dampened or distorted, do not inflate the balloon. The catheter may be wedged with the balloon deflated. Check catheter position.
- When the balloon is reinflated to record wedge pressure, add the inflation medium (CO_2 or air) slowly under continuous monitoring of the pulmonary artery pressure waveform. Stop inflating immediately when the pulmonary artery tracing is seen to change to pulmonary artery wedge pressure. Remove the syringe to allow rapid balloon deflation, and then reattach the syringe to the balloon lumen. **Air should never be used for balloon inflation in any situation where air may enter the arterial circulation.**
- Never over-inflate the balloon beyond the maximum volume printed on the catheter shaft. Use the volume limited syringe provided with the catheter.
- Do not use liquids for balloon inflation; they may be irretrievable and may prevent balloon deflation.
- Keep the syringe attached to the balloon lumen of the catheter to prevent accidental injection of liquids into the balloon.

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

- If the pulmonary artery diastolic (PAD) and the wedge (PAOP) pressures are nearly identical, wedging the balloon may not be necessary: measure PAD pressure instead of PAOP as long as the patient's heart rate, blood pressure, cardiac output and clinical state remain stable. However, in states of changing pulmonary arterial and pulmonary venous tone (i.e. sepsis, acute respiratory failure, and shock), the relationship between PAD and "wedge" may change with the patient's clinical condition. PAOP measurement may be necessary.
- Keep "wedge" time to a minimum (two respiratory cycles or 10–15 seconds), especially in patients with pulmonary hypertension.
- Avoid prolonged maneuvers to obtain wedge pressure. If difficulties are encountered, give up the "wedge."
- Never flush the catheter when the balloon is wedged in the pulmonary artery.

5. Patients at highest risk of pulmonary artery rupture or perforation

- Patients who have pulmonary hypertension and hypothermia are at greater risk for pulmonary artery rupture or perforation.

6. Bedside physiologic monitor settings initiated and maintained

- Pulmonary artery pressure systolic/diastolic/mean alarm settings must be initiated to alert clinicians to a spontaneous wedge or changes in the patient status.
- Appropriate scaling should be used in order to visualize the pulmonary artery pressure waveform. Scales set too low (0–20 mmHg) may result in "clipping" of all or part of the waveform. Scales set too high (0-150 mmHg) may result in a "damped" appearance due to waveform compression, leading to inappropriate troubleshooting or non-recognition catheter migration into a wedge position or into the right ventricle.
- Color coding (if available) for appropriate pressure channel identification. Pulmonary artery pressures = Yellow, right atrial pressures = Blue or per institutional policy.

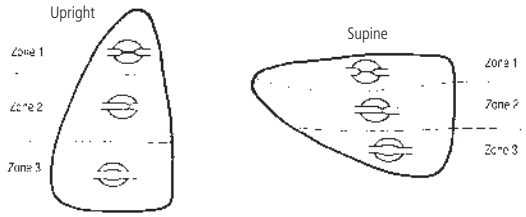
COMPUTATION CONSTANTS

Model		741HF75, 741F75 780HF75, 780F75 782HF75 and 782F75	631HF55 and 631F55
Injectate Temp (C°)	Injectate Volume (ml)	Computation Constants (CC)*	
0–5	10	0.564	–
	5	0.257	0.265
	3	0.143	0.148
	1	–	0.032
19–22	10	0.582	–
	5	0.277	0.294
	3	0.156	0.172
	1	–	0.049
23–25	10	0.607	–
	5	0.294	0.308
	3	0.170	0.183
	1	–	0.057

Lung Zone Placement

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

LUNG ZONES



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

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

Zone 3: $P_a > P_v > P_A$. Capillaries are open resulting in blood flow. Catheter tip is usually below the level of the left atrium and can be verified by a lateral chest x-ray. PAOP readings will be accurate.

GUIDELINES FOR OPTIMAL LUNG ZONE CATHETER PLACEMENT

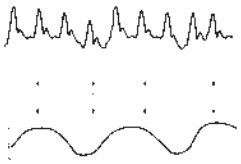
Criterion	Optimal Zone 3	Sub-Optimal Zone 1 or 2
Catheter Tip Location	Below level of LA	Above level of LA
Respiratory Variations	Minimal	Marked
PAOP Contour	"a" and "v" waves clearly present	"a" and "v" waves unclear
PAD Versus PAOP	PAD > PAOP (normal physiology)	PAOP > PAD (no abnormal "a" and "v" waves present)
PEEP Trial	Change in PAOP < ½ change in PEEP	Change in PAOP > ½ change in PEEP
Hydration Status	Normovolemic	Hypovolemic

Ventilatory Effects on Pulmonary Artery Tracings

Spontaneous Breathing

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

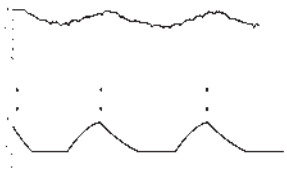
SPONTANEOUS BREATHING



Controlled Mechanical Ventilation

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

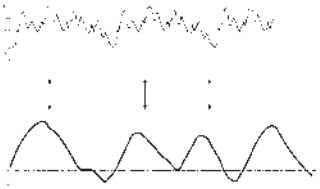
CONTROLLED MECHANICAL VENTILATION



Intermittent Mandatory Ventilation

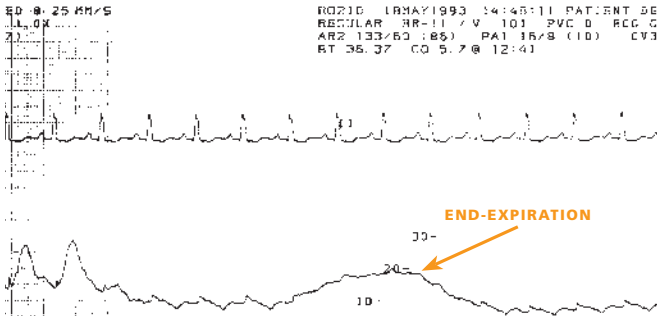
When a form of intermittent mandatory ventilation is being applied, some breaths are controlled while others are spontaneous. The impact on the tracings is that during the controlled breaths, inspiration will produce elevated waves such as those during controlled mechanical ventilation. During a spontaneous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

INTERMITTENT MANDATORY VENTILATION



This is a tracing of a patient who is spontaneously breathing. Identification of PAP pressures and PAOP pressures are influenced by the respiratory variations noted. Pressure valves should be obtained at end-expiration. Possible causes for the respiratory variation includes hypovolemia or catheter tip in a non-zone 3 placement.

PAP TO PAOP TRACING



Cardiac Output Determinations

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

Fick Method

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

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

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

(volume % = 1 mL oxygen/100 mL)

Cardiac Index = Cardiac Output / Body Surface Area (CI = CO / BSA)

- Normal (CaO_2) arterial oxygen content: 20 volume %
- Normal (CvO_2) mixed venous oxygen content: 15 volume %
- Normal (VO_2I) oxygen consumption index: 120–200 mL/min/m^2

Inserting these values into the equation:

CI = 120 to 200 / (20 – 15) x 100
= 120 to 200 / 5 x 100
= 2400 to 4000 mL/min/m^2 or 2.4 to 4 L/min/m^2

Calculating cardiac output with the Fick equation requires accurate measurement of the oxygenation variables. Slight errors in the content values may produce large errors in the oxygen consumption result. Indexed normal VO_2 values range 120–200 mL/min/m^2. Critically ill patients may not have normal oxygen consumption values. Therefore, insertion of normal values into the above Fick equation may produce erroneous cardiac output values.

Dye Indicator Dilution Method

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

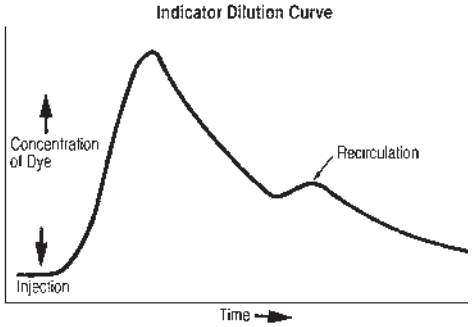
The basis of the dye indicator technique is that a known concentration of an indicator is added to a body of fluid. After allowing adequate mixing time, the dilution of that indicator will produce the amount of fluid it was added to. A densimeter records the dye or indicator concentration in the blood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

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

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

WHERE:

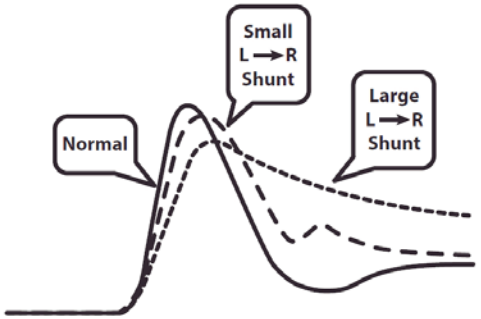
CO = cardiac output (1/min)
I = amount of dye injected (mg)
60 = 60 sec/min
Cm = mean indicator concentration (mg/L)
t = total curve duration (sec)
k = calibration factor (mg/mL/mm deflection)



Dye Dilution curves with Intra-Cardiac Shunts

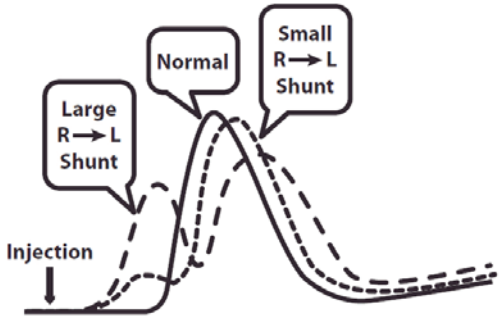
Left-to-Right Shunt Dye Dilution Curve

In patients with a small left-to-right shunt as compared to a normal pattern, there is a decreased peak height with an early "re-circulation" at the end of the curve. A large left-to-right shunt has a slower peak progression height and no peak or "re-circulation" at the end of the curve. In these patients there is also a slow return to baseline.



Right-to-Left Shunt Dye Dilution Curve

A small right-to-left shunt has an early peak because contrast rapidly enters the systemic circulation, bypassing the pulmonary vasculature. In a large right-to-left shunt there exists a more pronounced early peak.



Thermodilution Method

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

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

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

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

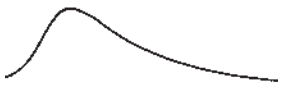
WHERE :

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

Thermodilution Curves

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

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


Normal Cardiac Output


Artifact Due to Noise Interference

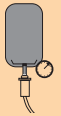
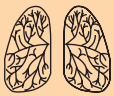
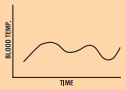

High Cardiac Output


Low Cardiac Output


Improper Injection Technique

Troubleshooting Key Factors in Optimizing Bolus CO Determinations

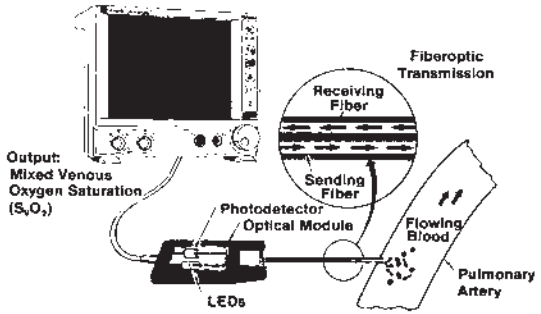
The chart below describes factors that can influence the accuracy and reproducibility of bolus thermodilution cardiac output values.

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

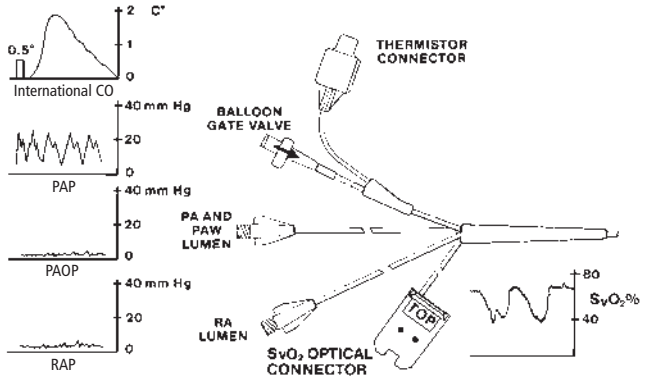
Vigilance II Monitor and Advanced Technology Swan-Ganz System

Continuous Mixed Venous Oxygen Saturation Monitoring

REFLECTION SPECTROPHOTOMETRY



SWAN-GANZ OXIMETRY TD CATHETER



Vigilance II Monitor Abbreviated Instructions for Use and Mixed Venous Oxygen Saturation (SvO_2)

For *In Vitro* Calibration

1. Connect catheter to optics module.
2. Select SvO_2 (Swan-Ganz catheter) or $ScvO_2$ (PreSep, PediaSat catheter) in the Large Parameter frame.
3. Select *In Vitro* Calibration.
4. Select Calibrate and press the knob. Wait for calibration to complete.
5. Flush catheter; check balloon. Insert catheter in PA.
6. Select START, press knob and wait for Optical Module to update.
7. SvO_2 or $ScvO_2$ value will appear in the Large Parameter frame.

For *In Vivo* SvO_2 Calibration:

1. Turn Navigation Knob to select SvO_2 or $ScvO_2$. Press knob.
2. Select *In Vivo* Calibration. Press knob.
3. Select Draw, press knob, and slowly draw waste and laboratory blood sample for co-oximeter analysis.
4. Upon receipt of lab values from drawn sample, enter venous oximetry value and either HGB or HCT.
5. Select CALIBRATE and press knob. Wait for calibration to complete.
6. Confirm that SvO_2 or $ScvO_2$ is displayed in the Large Parameter frame and that the values are correct.

To Transport the Optical Module:

1. After reconnecting patient cable and optical module, turn knob to select **SvO_2 or $ScvO_2$ in the Large Parameter frame.** Press knob.
2. Select **RECALL OM DATA** and press knob.
3. If the data in the Optical Module is <24 hours old and appears correct, select YES and press knob.

To Begin Continuous Cardiac Output (CCO) Monitoring:

1. Connect thermal filament and thermistor connections on catheter to the patient cable.
2. Press the **START/STOP CCO BUTTON**  to begin Continuous Cardiac Output (CCO) monitoring. A message will appear confirming the monitor is Collecting CCO Data.
3. The average CCO value will appear in the Large Parameter frame in from 1 to 8 minutes.

To Configure the Computer Display Screen:

1. To change screen display:
   - Turn Navigation knob to select the SET UP icon  to change display format (temperature units, international units, time format, alarm volume, and display language).
   - Select the desired action, press knob.
   - Rotate knob to select the desired change. Press knob.
   - Select RETURN and press knob to return to the display screen.
2. To change alarm settings:
   - Select desired Large Parameter frame with Navigation knob, and press the knob.
   - Select the Alarm limit value on the lower right side of the drop-down window. Press the knob, then rotate knob to select the upper value. Press knob to set the value. Repeat this process for the lower value.
   - Rotate knob to select RETURN. Press knob to exit the drop down menu.
3. To activate the Split Screen to see STAT DISPLAY:
   - Rotate Navigation Knob to select the SPLIT SCREEN icon  at the bottom of the display.
   - Only CCO(I), RVEF and EDV(I) values can be shown here. To add one of these parameters to the STAT SCREEN, select that parameter in one of the Large Parameter Frames. See the Operators Manual for a description of the STAT Screen.
   - To remove the SPLIT SCREEN, rotate the knob to select the SPLIT SCREEN icon and press the knob.

To Display the Cardiac/Oxygen Profile:

1. To display the Cardiac or Oxygen Patient Profile:
   - Press the Patient Data button  found to the right of the Display Screen.
   - Either the Oxygen Profile or the Cardiac Profile will appear.
   - Rotate the knob to select the alternate profile at the bottom of the drop down window and press the knob to change the profile.
2. To manually enter values in the Patient Profile screens:
   - Press the Patient Data button to activate the drop down window.
   - Select the appropriate Patient Profile.
   - Rotate the knob to select the desired parameter. Press the knob.
   - Enter the desired value. An asterisk will appear by the value name to designate a manually inserted value.
   - Rotate knob to select exit. Press Patient Profile button to exit the Patient Profile window.
   - **Note: once an asterisk appears the value must be "cleared" to be auto – updated.*

To Perform Bolus Cardiac Output (ICO):

1. Press the CCO/ICO button  found to the right of the Display screen. The ICO screen will appear. To exit ICO mode, press the button again.
   - Rotate the Navigation Knob to select CO or CI in the Large Parameter frames. Press the knob.
   - Select any of the options shown to make adjustments to the ICO process.
   - For automatic ICO Bolus operation, select Automatic.

- When the monitor has established a stable baseline temperature an INJECT message appears on the screen. Inject the solution at this time. Repeat this process up to 6 times. The monitor will display the cardiac output in the BOLUS frame for each injection in the series.
- After completing the desired number of injections, rotate the knob to select the BOLUS frame (3rd Large Parameter frame showing the values for each injection). Press the knob. The average of the injections will be shown in the CO/CI Large Parameter frame and the Bolus Edit drop down screen will appear.

2. To delete individual CO/CI values from the average:
   - Rotate the Navigation Knob to select the 3rd Large Parameter BOLUS frame.
   - Press knob to open the BOLUS EDIT window.
   - Rotate and press the knob to select one or more values to delete.
   - Rotate and press the knob to select REDO SERIES. Values selected for deletion will be removed and the CCO/CCI average will be displayed.
3. To Exit BOLUS CO MODE
   - From the BOLUS EDIT screen, rotate the knob and select EXIT. Press the knob.
   - Press the CCO/ICO button found at the right of the Display screen.
   - Answer the prompt to restart Continuous Cardiac Output (CCO) by rotating the knob, selecting the answer, and pressing the knob.

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

1. To start operational pause:
   - Press and hold the Alarm Silence button  for at least 3 seconds.
   - The yellow Operational Pause banner appears. Data collection and display in Large Parameter frames are paused and time stamped.
   - Alarms associated with these parameters are silenced since monitoring is interrupted.
   - Blood Temperature and Small Parameter frame parameters are monitored and displayed.
2. To discontinue operational pause:
   - Push Navigation Knob to Exit Operational Pause
   - Select Yes or No with Navigation Knob when asked if you want to restart CCO. If yes is selected, CCO will start and a new average value will appear in the Large Parameter frame within approximately 1–8 minutes.
   - With the Navigation Knob, select Yes or No when asked if you want to recalibrate SvO_2 or $ScvO_2$. If YES, the Calibration screen will appear. If NO, SvO_2 monitoring will begin using the calibration values at the time Operational Pause was begun.

Vigilance II Monitor Troubleshooting

CCO/CCI FAULTS

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

CCO/CCI FAULTS [CONT.]

CCO/CCI Faults	Possible Cause(s)	Suggested Action(s)
Check Thermistor Connection	• Catheter Thermistor connection not detected • Monitored blood temperature is <15°C or >45°C • CCO cable malfunction	• Verify that catheter- Thermistor is connected securely to CCO cable • Verify that blood temperature is between 15–45°C • Disconnect Thermistor connection and check for bent/missing pins • Perform Patient CCO Cable Test • Change CCO cable
Cardiac Output <1.0 L/min	• Measured CO <1.0 L/min	• Follow hospital protocol to increase CO • Resume CCO monitoring
Thermal Signal Loss	• Thermal signal detected by monitor is too small to process • Sequential compression device interference	• Verify proper catheter position in the pulmonary artery ▪ Confirm wedge pressure balloon inflation volume of 1.25–1.50 mL ▪ Confirm appropriate catheter placement for patient's height, weight and insertion site ▪ Consider chest x-ray for evaluation of proper placement • Temporarily turn off sequential compression device per hospital procedure • Resume CCO monitoring

CCO/CCI ALERTS

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

CCO/CCI GENERAL TROUBLESHOOTING

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

SVR/SVRI Messages and Troubleshooting

SVR/SVRI ALERTS AND GENERAL TROUBLESHOOTING

SVR/SVRI Alert Messages	Possible Cause(s)	Suggested Action(s)
SVR: Slaved-In Pressures Signal Loss	• *Vigilance II* analog input port not configured to accept MAP and CVP • Analog input interface cable connections not detected • Inaccurate input signal • External monitor malfunction	• Verify correct voltage range and low/high voltage values on the *Vigilance II* monitor for external monitor • Verify cable connection between the *Vigilance II* monitor and bedside monitor is secure • Verify correct height/weight entries and units of measure for patient's BSA • Check for signal at external monitor's analog output device • Change external device module, if used
SVR/SVRI Topic	**Possible Cause(s)**	**Suggested Action(s)**
SVR > SVRI	• Incorrect patient BSA	• Verify units of measure and values for patient's height and weight
Vigilance II MAP and CVP: ≠ External Monitor	• *Vigilance II* monitor configured incorrectly • Inaccurate input signal • External monitor malfunction	• Verify correct voltage range and low/high voltage values on the *Vigilance II* monitor for external monitor • Confirm correct units of measure for analog input port voltage values (mmHg or kPa) • Verify correct height/weight entries and units of measure for patient's BSA • Check for signal at external monitor's analog output device • Change analog input interface cable • Change external device module, if used • Clear asterisk (*) from Cardiac Profile screen for MAP and CVP if slaving from an external device

Oximetry Messages and Troubleshooting

OXIMETRY FAULTS AND ALERTS

Oximetry Fault Messages	Possible Cause(s)	Suggested Action(s)
Light Range	• Poor optical module/catheter connection • Debris or film obstructing optical module/catheter connection lens • Optical module malfunction • Catheter kinked or damaged	• Verify secure optical module/catheter connection • Clean optical module/catheter connectors with 70% isopropyl alcohol and swab, let air dry and recalibrate • Replace catheter if damage is suspected and recalibrate
OM Disconnected	• Optimal module connection at monitor not detected • Bent or missing optical module connector pins	• Verify secure optical module/catheter connection • Check optical module cable connector for bent/missing pins
OM Memory	• Optimal module memory malfunction	• Change optical module and recalibrate
Value Out of Range	• Incorrectly entered oximetry, HGB or Hct values • Incorrect HGB units of measure • Calculated oximetry value if outside of the 0–99% range	• Verify correctly entered oximetry, HGB and Hct values • Verify correct HGB unit of measure • Obtain updated oximetry lab values and recalibrate
Red/IR Transmit	• Debris or film obstructing optical module/catheter connection lens • Optical module malfunction	• Clean module/catheter connections with 70% isopropyl alcohol and swab, let air dry and recalibrate • Change optical module and recalibrate
OM Temperature	• Optical module malfunction	• Change optical module and recalibrate
Oximetry Not Available	• Internal system malfunction	• Power monitor off and on to restore system • If problem persists, contact Edwards Technical Support
Oximetry Alert Messages	**Possible Cause(s)**	**Suggested Action(s)**
SQI = 4	• Low blood flow at catheter tip or catheter tip against vessel wall • Significant changes in HGB/Hct values • Catheter tip clotted • Catheter kinked or damaged	• Verify proper catheter position. For SvO_2, verify proper catheter position in the pulmonary artery ▪ Confirm wedge pressure balloon inflation volume of 1.5 mL (for SvO_2 only) ▪ Confirm appropriate catheter placement for patient's height, weight and insertion site ▪ Consider chest x-ray for evaluation of proper placement • Aspirate then flush distal lumen per hospital protocol • Update HGB/Hct values using Update function • Check catheter for kinking and recalibrate • Replace catheter if damage is suspected and recalibrate

OXIMETRY WARNINGS

Oximetry Warning Messages	Possible Cause(s)	Suggested Action(s)
In Vitro Calibration Error	• Poor optical module and catheter connection • Calibration cup wet • Catheter kinked or damaged • Optical module malfunction • Catheter tip is not in catheter package calibration cup	• Verify secure optical module/catheter connection • Straighten any visible kinks; replace catheter if damage is suspected • Change optical module and recalibrate • Verify catheter tip is securely seated in calibration cup • Perform *in vivo* calibration
Unstable Signal	• Changing oximetry, HGB/Hct, or unusual hemodynamic values	• Stabilize patient per hospital protocol and perform *in vivo* calibration
Wall Artifact or Wedge Detected	• Low blood flow at catheter tip • Catheter tip clotted • Catheter tip wedged in vessel or against vessel wall	• Aspirate then flush distal lumen per hospital protocol • Verify proper catheter position. For SvO_2, verify proper catheter position in the pulmonary artery ▪ Confirm wedge pressure balloon inflation volume of 1.5 mL (for SvO_2 only) ▪ Confirm appropriate catheter placement for patient's height, weight and insertion site ▪ Consider chest x-ray for evaluation of proper placement • Perform *in vivo* calibration

OXIMETRY GENERAL TROUBLESHOOTING

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

CEDV Messages and Troubleshooting

CEDV ALERTS

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

ICO (Bolus) Messages and Troubleshooting

ICO FAULTS AND ALERTS

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

SWAN-GANZ CATHETERS – ADVANCED AND STANDARD TECHNOLOGY

ICO FAULTS AND ALERTS [CONT]

ICO Fault Messages	Possible Cause(s)	Suggested Action(s)
Check Injectate Probe Connection	• Injectate temperature probe not detected • Injectate temperature probe malfunction • CCO cable malfunction	• Verify connection between CCO cable and injectate temperature probe • Change injectate temperature probe • Change CCO cable
Injectate Volume not valid	• In-line probe injectate volume must be 5 mL or 10 mL	• Change injectate volume to 5 mL or 10 mL • Use a bath type probe for an injectate volume of 3 mL

ICO ALERTS

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

Adult RVEDV Quick Reference

1. Parameters Attained with Vigilance II Monitor

- CARDIAC OUTPUT (CO) = 4 – 8.0 L/min
- CARDIAC INDEX (CI) = 2.5 – 4.0 L/min/m^2
- STROKE VOLUME (SV): The volume of blood ejected from the ventricle in each beat.
  SV = CO / HR x 1000
  Normal SV: 60 – 100 mL
  Normal SVI: 33 – 47 mL/beat/m^2
- END-DIASTOLIC VOLUME (EDV): The volume of blood in the ventricle at the end of the diastole. EDV = SV/EF
  Normal RV EDV: 100 – 160 mL
  Normal RV EDVI: 60 – 100 mL/m^2
- END-SYSTOLIC VOLUME (ESV): The volume of blood in the ventricle at the end of systole.
  ESV = EDV – SV
  Normal RV ESV: 50 – 100 mL
  Normal RV ESVI: 30 – 60 mL/m^2
- EJECTION FRACTION (EF): The percentage of blood ejected from the ventricle each beat.

$$EF = \frac{EDV - ESV}{EDV} \quad \text{or} \quad \frac{SV}{EDV}$$

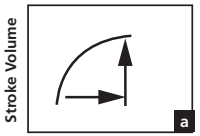
Normal RVEF: 40 – 60%

(Note: As with all measurements in hemodynamic monitoring, the absolute number is not as important as trends and changes in response to therapy.)

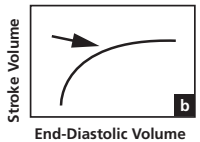
2. Goal of RV Volumetric Measurements

- Optimize RV Efficiency
- Optimize the relationship between EDV and SV

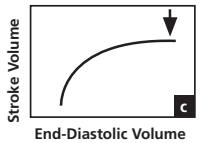
a. In an efficient state, an increase in PRELOAD (EDV) will result in an INCREASE in STROKE VOLUME (SV).



b. Prior to reaching the FLAT PART of the curve, an increase in PRELOAD (EDV) will increase SV while not causing a decrease in Ejection Fraction.



c. On the FLAT PART of the curve, a further increase in PRELOAD (EDV) will not result in an increase in SV.

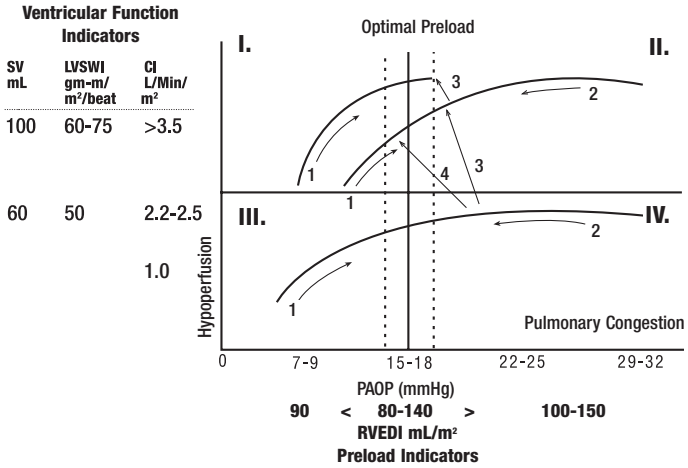


At this point, a further increase in volume may:

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

Therapy should be directed at increasing contractility or reducing afterload.

Idealized Ventricular Function Curves



I. Normal Perfusion
No Pulmonary Congestion

II. Normal Perfusion
Pulmonary Congestion

III. Hypoperfusion
No Pulmonary Congestion

IV. Hypoperfusion
Pulmonary Congestion

Possible Interventions

1 = ↑ Preload; moves along same curve, volume

2 = ↓ Preload; moves along same curve, diuretic/ venodilator

3 = ↑ Contractility; shifts to higher curve, minimal change in preload, positive inotrope

4 = ↓ Afterload; shifts to a higher curve at a lower preload, afterload reducters, vasodilators

Swan-Ganz Reference Chart

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

This chart can be used as a quick ready reference guide to choose a catheter specific to the needs of the patient.

	Catheter Model Number	Lumens	Length (cm)	PAP/PAOP		Distance from Tip: Proximal Injectate Port	Distance from Tip: Infusion Port	Distance from Tip: RV Infusion/ VIP Ports	SvO_2	Continuous	Recommended Introducer: French Size	Recommended Introducer: mm
Advanced Technology Catheters – Continuous Hemodynamic Monitoring												
CCOmbo/CEDV/VIP	777HF8	7	110	•		26 cm		30 cm	•	•	9	3
CCOmbo/CEDV	774HF75	6	110	•		26 cm			•	•	8.5 or 9	2.8 or 3
CCO/CEDV	177HF75	6	110	•		26 cm		30 cm	•	•	8 or 8.5	2.7 or 2.8
CCOmbo/VIP	746HF8	7	110	•		26 cm		30 cm	•	•	9	3
CCOmbo	744HF75	6	110	•		26 cm			•	•	8.5 or 9	2.8 or 3
CCO	139HF75(P)	6	110	•		26 cm		30 cm		•	8 or 8.5	2.7 or 2.8
SvO_2	741HF75	6	110	•		30 cm			•		8 or 8.5	2.7 or 2.8
Standard Thermodilution Catheters (some models available in S-Tip, T-Tip, C-Tip and various stiffness characteristics to facilitate femoral approach)												
TD Catheter	131HF7	4	110	•		30 cm					8 or 8.5	2.7 or 2.8
TD Catheter with VIP Lumen	831HF75(P)	5	110	•		30 cm	31 cm				8.5 or 9	2.8 or 3
TD Catheter with two VIP Lumens	834HF75	6	110	•		30 cm	31 cm	19 cm			8.5 or 9	2.8 or 3
TD Catheter	132F5	4	75	•		15 cm					6 or 6.5	2 or 2.2
TD Catheter	096F6P	4	110	•		30 cm					7 or 7.5	2.3 or 2.5
TD Catheter	141HF7P	4	110	•		30 cm					8 or 8.5	2.7 or 2.8
TD Catheter, S tip	151F7	4	110	•		30 cm					8 or 8.5	2.7 or 2.8
TD Catheter	143HTF7	5	110	•		30 cm					8 or 8.5	2.7 or 2.8
ControlCath C tip (non PVC) (non-latex)	K9FC146F7	4	110	•		30 cm					8.5 included in kit	2.8
ControlCath C tip (non PVC)	C144F7	4	110	•		30 cm					7	2.3
ControlCath S tip (non PVC)	S144HF7	4	110	•		30 cm					7	2.3
Pacing Catheters and Thermodilution Paceport Catheters (use with models D98100 – Chandler Transluminal V-Pacing Probe and/or D98500 – Flex-Tip Transluminal A-Pacing)												
Paceport	931HF75	5	110	•		30 cm		19 cm			8 or 8.5	2.7 or 2.8
A-V Paceport	991HF8	6	110	•		30 cm	27 cm	19 cm			8.5	2.8
Pacing TD-A, V, or A-V Pacing	D200HF7	4	110			30 cm					8	2.7
Bipolar Pacing (Femoral)	D97130F5	1	90								6	2
Bipolar Pacing	D97120F5	1	90								6	2
VIP Bipolar Pacing	D97140HF5	2	100					12 cm				
Monitoring Catheters												
Double Lumen Monitoring	110F5	2	110	•							7	2.3
	111F7	2	110	•							7	2.3
	123F6P	2	110	•							6	2
Triple Lumen Monitoring	114F7P	4	110	•		30 cm					7	2.3
Pediatric Double Lumen Monitoring	116F4	2	60	•							6	2
Small French Oximetry	040HF4	2	40						•		4.5	1.5
Pulmonary Angiography	191F7	2	110			1.5–2.5 cm					7	2.3

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

SWAN-GANZ CATHETERS – ADVANCED AND STANDARD TECHNOLOGY

Notes

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.

Hemodynamic Parameters

NORMAL VITAL SIGNS AT REST BASED ON AGE

Age	Heart Rate (beats/ min.)	Respirations (breaths/min.)	BP (mmHg)	CO (L/min)	CI (L/min//m²)	SVR (dynes/ sec/cm²)	PVR (dynes/ sec/cm²)
Newborns (1-30 days)	130-160	40-60	50-90/20-60	0.8-1.0			
Infant (30 days-12 months)	120-160	30-60	74-100/50-70	1.0-1.3	4.0-5.0	2800-4000	2000-3200
Toddler (1-4 years)	90-140	24-40	80-112/50-78	1.3-2.7		2000-2800	
Preschoolers (4-5 years)	80-110	22-34	82-110/50-78	2.3-3.0			
School-aged (5-12 years)	75-100	18-30	84-120/54-80	3.0-4.0	3.0-4.5	1200-2000	40-320
Adolescents (13-17 years)	60-90	16-25	94-130/62-88	4.0-6.0			
Adults (17 and above)	60-80	12-16	100-140/70-88	4.0-8.0	2.5-4.2	770-1500	20-120

Conversion: Fahrenheit to Celsius = (°F temp – 32) x (5/9) = °C or Celsius to Fahrenheit = ([9/5] x °C temp) + 32 = °F

TYPICAL HEMODYNAMIC PROFILES IN VARIOUS ACUTE CONDITIONS

Condition	HR	MAP	CO/CL	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 ad 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

↑ = increase; ↓ = decrease; N = no change; V = variable

CALCULATION OF BODY MASS INDEX (BMI) FOR >2 YRS:

$$BMI = \frac{\text{weight in Kg}}{\text{height (m}^2\text{)}} \quad \text{or} \quad BMI = \frac{\text{weight in lbs} \times 703}{\text{height (in}^2\text{)}}$$

BMI can be calculated and plotted on growth charts to determine index of healthy weight, and as a predictor of morbidity and mortality.

CALCULATION OF IDEAL BODY WEIGHT (IBW)

1 lb = 0.454 Kg or 1 Kg = 2.2 lbs

$$\text{1-18 yrs: } \frac{(\text{height}^2 \times 1.65)}{1000} = \text{IBW (Kg)}$$

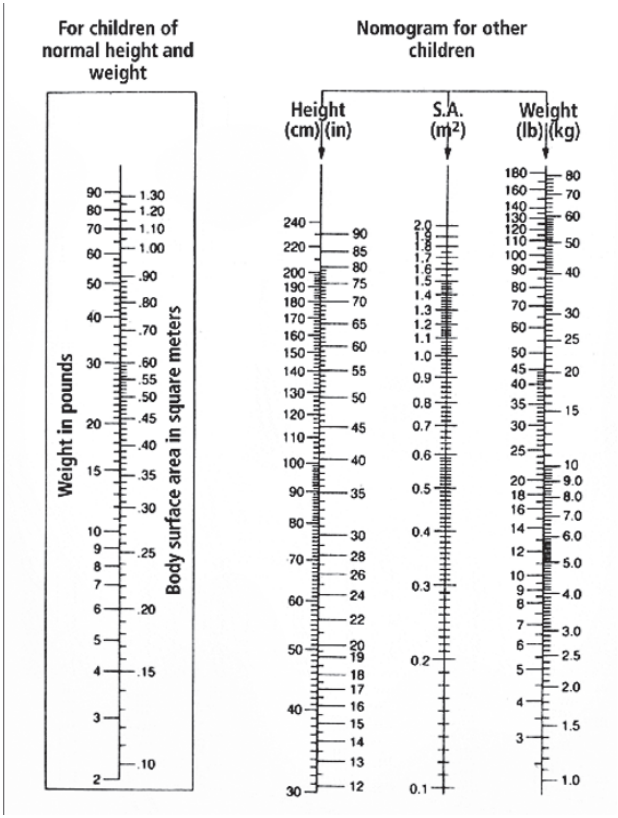
BODY SURFACE AREA CALCULATION (BSA)

1 in = 2.54 cm

$$BSA\ (m^2) = \sqrt{\frac{Ht\ (cm) \times Wt\ (Kg)}{3600}}$$

AVERAGE WEIGHTS & BSA BASED ON AGE

Age (months/years)	Ave Wt (Kg)	Approx BSA (m²)	Age (years)	Ave Wt (Kg)	Approx BSA (m²)
30-40 weeks gestation	1.3-3.0	0.12-0.20	5 years	18.0	0.76
Birth (40 weeks gestation)	3-4	0.25	6 years	20.0	0.82
3 months	5.0	0.29	7 years	23.0	0.90
6 months	7.0	0.38	8 years	25.0	0.95
9 months	8.0	0.42	9 years	28.0	1.06
12 months	10.0	0.49	10 years	33.0	1.18
2 years	12.0	0.55	11 years	35.0	1.23
3 years	15.0	0.64	12 years	40.0	1.34
4 years	17.0	0.74	Adult	70.0	1.73



Cardiac Scoring

HEART FAILURE CLASSIFICATIONS

Ross Functional Classification of Heart Failure (Pediatrics)	
Class	**Description**
I	Heart disease without limitations
II	Mild tachypnea or diaphoresis during feeds, secondary growth failure (infant); Dyspnea with exertion (older child)
III	Marked tachypnea, diaphoresis with feeds, longer to feed, failure to thrive (FTT) (infant); Marked dyspnea with exertion (older child)
IV	Tachypnea, retractions, grunting or diaphoresis at rest (infant/older child) Failure to thrive (FTT) (infant)

NEW YORK HEART CLASSIFICATION OF CARDIOVASCULR DISEASE

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

WORLD HEALTH ORGANIZATION (WHO) PULMONARY HYPERTENSION FUNCTIONAL CLASSIFICATION

Classification	Description
I	PHTN without physical activity limitation. Ordinary activity not resulting in dyspnea, fatigue, chest pain or near-syncope
II	PHTN with slight physical activity limitation. Comfortable at rest, ordinary activity may result in dyspnea, fatigue, chest pain or syncope
III	PHTN with marked physical activity limitation. Comfortable at rest, less than ordinary activity causes dyspnea, fatigue, chest pain or syncope
IV	PHTN unable to perform physical activities without symptoms. Signs of right sided heart failure. At rest may have dyspnea, fatigue and increased discomfort with physical activity

PHTN = pulmonary hypertension

HEART SOUNDS

Sounds	Location	Represents	Abnormal Association
S_1	Apex or left lower sternal border (LLSB)	• Closure mitral and tricuspid valves	• Widely split: Ebstein's anomaly, RBBB • Widely split with fixed S_2: abnormal pulmonary valve, RV overload, electrical delay in RV contraction, early aortic valve closure (mitral regurgitation)
S_2	Left upper sternal border (LUSB)	• Closure pulmonary and aortic valves • Normal physiologic split increases with inspiration	• Fixed S_2 with widely split S_1: (see above) • Narrowly split S_2: PHTN, AS, delayed LV contraction (LBBB) • Single S_2: PHTN, single semilunar valve (pulmonary or aortic atresia, truncus), severe AS • Paradoxically split S_2: Severe AS, LBBB, WPW
S_3	Apex or LLSB	• Dilated ventricles	• VSD, CHF, cardiomyopathy
S_4	Apex	• Decreased ventricular compliance	• Restrictive cardiomyopathy
Ejection click	Apex	• Stenosis of semilunar valves and great vessels	• Systemic and pulmonary HTN, persistent truncus, TOF
Midsystolic click	Apex	• Mitral valve prolapse	• Mitral valve prolapse • Hypertrophic cardiomyopathy
Diastolic snap	Apex or LLSB	• Rheumatic valvular abnormalities	• Mitral stenosis

CARDIAC MURMUR GRADES

Grade	Description
I	Faintest murmur audible on auscultation
II	Soft, easily auscultated
III	Moderately loud, not associated with palpable cardiac thrill
IV	Loud, associated with palpable cardiac thrill
V	Very loud, palpable thrill, can be heard with part of stethoscope off chest wall, palpable cardiac thrill
VI	Loudest, can be auscultated with stethoscope off chest wall, palpable thrill

TYPES OF MURMURS WITH ASSOCIATED CARDIAC DEFECTS

Systolic	Diastolic
Aortic stenosis, Ao coarctation, PDA, Pulmonary stenosis	Aortic regurgitation
ASD,VSD, AVSD	Pulmonary regurgitation
Hypertrophic cardiomyopathy	Tricuspid Stenosis
Tricuspid or mitral regurgitation, Mitral valve prolapse	Mitral Stenosis
TOF, TAPVR	
Innocent murmurs	

PULSE PRESSURE DIFFERENTIAL ASSOCIATED WITH CARDIAC DISEASE

Widened Pulse Pressure	Narrowed Pulse Pressure
Aortic insufficiency	Aortic stenosis
Arteriovenous fistula	Pericardial effusion or tamponade
Patent Ductus Arteriosus	Pericarditis
Thyrotoxicosis	Extreme tachycardia

Inotrope Score Calculations:

(Calculated for first 48 hours post-operatively in the ICU. Higher scores indicative of poorer outcomes)

Inotrope Score (mcg/Kg/min) = [(dopamine + dobutamine = amrinone) x 1] = (milrinone x 20) + [epinephrine + norepinephrine + isoproterenol) x 100]

Modified Inotrope Score (mcg/Kg/min) = (dopamine) + (milrinone x 10) + (epinephrine x 100)

Electrocardiography

INTERVALS BASED ON AGE

Age (months, years)	Heart Rate (bpm)	P-R Interval (sec)	QRS Interval (sec)	QRS Axis Range (mean)	QTc Interval (sec)
0-1 month	130-160	0.08-0.12	0.05-0.07	+30 to +180 (+110)	0.44-0.49
1-12 months	120-160	0.08-0.14	0.05-0.07	+10 to +125 (+70)	0.44-0.49
1-4 years	90-140	0.10-0.14	0.06-0.07	0 to +110 (+60)	<0.44
4-8 years	80-110	0.11-0.16	0.07-0.08	-15 to +110 (+60)	<0.44
8-12 years	75-100	0.12-0.17	0.07-0.09	-15 to +110 (+60)	<0.44
12-16 years	60-90	0.12-0.17	0.07-0.10	-15 to +110 (+60)	<0.44
>16 years	60-80	0.12-0.20	0.08-0.10	-15 to +110 (+60)	<0.44

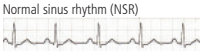
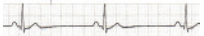
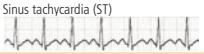
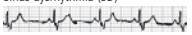
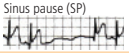
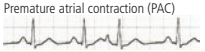
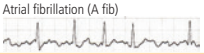
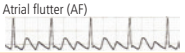
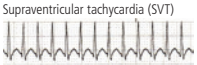
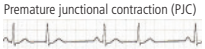
ECG COMPONENTS

	Represents	Abnormal findings
P-wave	• Atrial depolarization	• Inverted, absent or variable rate in certain dysrhythmias
PR Interval	• Atrial depolarization, beginning ventricular depolarization	• Prolonged or variable in AV blocks
QRS Complex	• Ventricular depolarization	• Widened or dysmorphic in ventricular dysrhythmias • Narrowed in accelerated or supraventricular dysrhythmias
ST Segment	• End ventricular depolarization, prior to ventricular repolarization • 2 mm elevation or depression may be normal in pediatrics	• Depressed indicative of myocardial ischemia • Elevated indicative of myocardial infarction
T-wave	• Ventricular repolarization	• In presence of hyperkalemia
QT Interval	• End of atrial depolarization to end of ventricular repolarization	• Shortened in hypercalcemia • Prolonged in hypocalcemia
U Wave	• Repolarization of purkinje fibers	• Present in hypokalemia

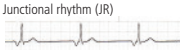
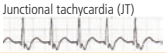
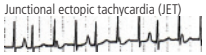
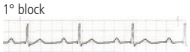
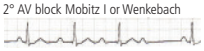
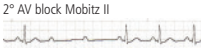
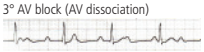
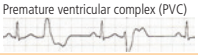
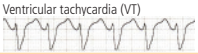
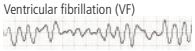
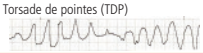
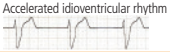
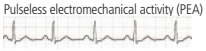
RHYTHM CATEGORIES

Sinus Rhythms	Atrial Dysrhythmias	Junctional (Nodal)	A-V Blocks	Ventricular Dysrhythmias
• Normal sinus rhythm • Sinus bradycardia • Sinus tachycardia • Sinus dysrhythmia • Sinus pause • Sick sinus syndrome	• Premature atrial contraction • Wandering atrial pacemaker • Atrial tachycardia • Atrial flutter • Atrial fibrillation • Supraventricular tachycardia	• Premature junctional beats • Junctional escape beats • Junctional rhythm • Junctional tachycardia • Junctional ectopic tachycardia	• 1° block • 2° block Mobitz I or Wenkebach Mobitz type II • 3° block, complete block	• Premature ventricular contraction • Ventricular tachycardia • Ventricular fibrillation • Torsade de pointes • Accelerated idioventricular

ECG RHYTHMS

Rhythm	Characteristics
Normal sinus rhythm (NSR) 	Regular rhythm with HR normal for age; A:V conduction 1:1
Sinus bradycardia (SB) 	Regular rhythm with HR <5th percentile for age; A:V conduction 1:1
Sinus tachycardia (ST) 	Regular rhythm with HR >95th percentile for age; A:V conduction 1:1
Sinus dysrhythmia (SD) 	Rhythm irregular with normal HR; AV conduction 1:1; common with variable respirations
Sinus pause (SP) 	Seen in digitalis toxicity or hypercalcemia
Sick-sinus-syndrome (SSS) 	Rapid atrial rhythm interspersed with sinus bradycardia; aka sinus nodal dysfunction (SND)
Premature atrial contraction (PAC) 	Early atrial contraction with abnormal p-wave, shortened P-R interval and narrow QRS
Wolf Parkinson White (WPW) 	Antegrade anomalous AV conduction via accessory pathway. Short PR interval, QRS morphology abnormal. Potential for variable dysrhythmias, most common in AV re-entry tachycardias.
Atrial ectopic tachycardia (AET) 	Variant of SVT; rapid atrial rate with narrow QRS; HR 120-300; non-responsive to cardioversion; can lead to tachycardic induced cardiomyopathy
Atrial fibrillation (A fib) 	Irregular rhythm; atrial rate 350-600 without discreet p-waves; ventricular response 110-150; normal QRS
Atrial flutter (AF) 	Atrial rate 250-350 regular with variable ventricular rate; sawtooth pattern; normal QRS
Supraventricular tachycardia (SVT) 	Re-entrant tachycardia originating other than SA node above the AV node; HR >230 bpm; abnormal p-wave with narrow QRS; responds to vagal maneuvers, adenosine and/or cardioversion
Premature junctional contraction (PJC) 	Premature beat originating near AV node; inverted or absent p-wave

ECG RHYTHMS [CONT.]

Rhythm	Characteristics
Junctional rhythm (JR) 	Slower heart rate with origin above or at AV node; absent or inverted p-waves
Junctional tachycardia (JT) 	Heart rate faster than junctional rhythm with origin above or at AV node; absent or inverted p-waves
Junctional ectopic tachycardia (JET) 	Abnormal automaticity focus near or above AV node; rapid heart rate with narrow QRS; seen post-op congenital cardiac repair, as congenital dysrhythmia or digoxin toxicity; does not respond to cardioversion or adenosine
1° block 	Prolonged P-R interval for age; 1:1 conduction; can progress into advanced blocks
2° AV block Mobitz I or Wenkebach 	P-R interval increases until QRS complex dropped; atrial conduction > ventricular; irregular rhythm
2° AV block Mobitz II 	Non-conducted p-waves seen, P-R interval constant; atrial conduction > ventricular; regular atrial rate with ventricular rate regular or irregular
3° AV block (AV dissociation) 	No relationship (conduction) between atria (p wave) and ventricle (QRS); atrial conduction > ventricular; atrial and ventricular rates independently regular
Premature ventricular complex (PVC) 	Premature ventricular depolarization; unifocal or multifocal; couplets consists of 2 pvcs combined; can occur with regular frequency i.e. bigeminal or trigeminal
Ventricular tachycardia (VT) 	3 or more PVCs with widened QRS; absent p-wave. With or without pulse
Ventricular fibrillation (VF) 	Asynchronous depolarization of ventricles with abnormal size and shape of QRS pulseless
Torsade de pointes (TDP) 	Polymorphous VT variant with axis change direction and variable ventricular rate; prolonged QTc; common in hypomagnesia
Accelerated idioventricular rhythm (AIVR) 	3 or more ectopic ventricular beats with rate slower than seen in VT; seen in digoxin toxicity, electrolyte imbalance, myocardial infarction (MI) and hypoxemia
Pulseless electromechanical activity (PEA) 	AKA electromechanical dissociation (EMD); normal ECG without pulse. Causes: hypothermia, hyperkalemia, hypoxemia, hypovolemia, hypoglycemia, acidosis, tension pneumothorax, pericardial tamponade, toxins, thrombosis (pulmonary or coronary), trauma
Asystole 	Absence of myocardial electrical activity; no ECG complexes present

PACING CODES

Chamber To Be Paced	Chamber To Be Sensed	Pacemaker Response To Intrinsic Event
A = Atrium V = Ventricle D = Dual (both atrium and ventricle)	A = Atrium V = Ventricle D = Dual (both atrium and ventricle) O = None (fixed)	I = Inhibit if senses intrinsic beat T = Trigger a beat if no intrinsic beat sensed D = Dual (inhibits and triggers) for A-V synchrony

Syndromes

Syndrome	Associated Cardiac Defects/Disorders
Alagille-Watson (cardiovertebral)	PS (peripheral)
Apert (Acrocephalosyndactyly)	PDA, ASD, VSD, PS, TOF, coarctation of Ao
Beckwith Wiedemann (exomphalos macroglossia gigantism)	Hypertrophic cardiomyopathy
Brachmann-De Lange	ASD, VSD, AV canal, PS, TOF
Brugada	Idiopathic VF, sudden cardiac death
Carpenter	PDA, ASD, VSD, PS, TOF, TGA
Cat Eye	TOF, TAPVR, persistent left-SVC, Eisenmenger complex
Cayler (Crying facies)	ASD, VSD, conotruncal cardiac defects, TOF
Charge	VSD, TOF
Cri-du-Chat (cat cry) aka Lejeune	PDA, ASD, VSD, TOF, PS
DiGeorge	Conotruncal cardiac defects, TOF with or without PV or PA, IAA, VSD, vascular rings, TA
Ehlers Danlos	Mitral valve prolapse
Eisenmenger	PDA, ASD, VSD, AV canal, A-P window
Ellis van Creveld (dwarfism)	Single atrium (incomplete form of AV septal defect)
Emery-Dreyfus Muscular Dystrophy (EDMD)	Prolongation of PR interval, sinus bradycardia, complete heart block (3° block), cardiomyopathy
Fanconi anemia (type I)	PDA, VSD
Fetal alcohol	ASD, VSD
Fragile X	MVP, Ao root dilatation, pectus excavatum
Fredreich ataxia	Hypertrophic cardiomyopathy, CHF
Goldenhaar (Oculoauricular vertebral)	PDA, ASD, VSD, coarctation of Ao, TOF
Heterotaxy (Ivemark or Isomerism)	Dextrocardia **Asplenia:** AV canal with DORV, TGA, PS or PA, TAPVR (obstructed), bilateral sinus and AV nodes **Polysplenia:** Abnormal AV valves, VSD with PS, ASD, DORV
Holt-Oram	ASD
Hurler (Mucopolysaccharidosis)	Coronary artery narrowing, thickening cardiac valves and myocardium
Kartagener	Situs solitus, ASD R-PA hypoplasia, TGA, dextrocardia, levocardia with partial abdominal situs inversus, dextrocardia with abdominal situs ambiguous
Kawasaki	Pericarditis, myocarditis, cardiac ischemia, coronary artery thrombosis or stenosis, valvular dysfunction
Leopard	PS

Syndromes [CONT.]

Syndrome	Associated Cardiac Defects/Disorders
Long QT (persistent)	Lethal ventricular dysrhythmias, QTc >0.46 sec
Marfan	MVP, dysrhythmias, Ao regurgitation, Ao dissection
Muscular dystrophy (Duchenne)	Cardiomyopathy (hypertrophic, dilated), MVP, dysrhythmias
Noonan	Dysplastic, stenotic PV, ASD, hypertrophic cardiomyopathy
Osler-Weber-Rendu (Hereditary telangiectasia)	A-V malformation throughout cardiovascular system, vascular aneurysms
Osteogenesis imperfecta	MVP, Ao root dilatation with insufficiency
Primary Pulmonary Hypertension (PPHN)	Increased PVR (systolic PAP >35 mmHg, mean PAP >25 mmHg), right heart failure
Pompe	Cardiomyopathy, prolonged PR interval
Progeria (Hutchinson-Gilford)	Myocardial infarction, progressive atherosclerosis, CHF
Rubella	PDA, PA stenosis
Scimitar	Right pulmonary veins drain to IVC, right lung with arterial supply from descending Ao
Shone complex	Parachute mitral valve, supravalvular mitral ring, sub-Ao stenosis and mitral stenosis
Sick-sinus	Bradycardia, sinus arrest, junctional escape rhythm
Shprintzen (Velo-cardio-facial)	Conotruncal defects, VSD, TA, IAA, TOF
Smith-Lemli-Opitz	ASD, VSD
TAR (thrombocytopenia absent radius)	ASD, TOF
Trisomy 13 (Patau)	ASD, VSD, cardiac positional anomalies
Trisomy 18 (Edwards)	VSD, poly-valvar thickening
Trisomy 21 (Down's)	A-V canal defect, ASD, VSD, TOF
Turner	HLHS, coarctation of Ao, bicommissural Ao valve
VACTERL	VSD, TOF
Williams	Ao stenosis (supravalvar), PS
Wolf-Parkinson-White	Dysrhythmias, SVT, AF

Medications

IVCD CALCULATIONS

Compute milligrams (mg) to add per 100 mL IVF:

$$\frac{\text{6 x pt's wt (Kg) x desired dose (µg/Kg/min)}}{\text{desired rate (mL/hr)}} = \text{mg/100 mL}$$

Compute µg/Kg/min at given rate:

$$\text{µg/Kg/min} = \left[\frac{\text{drug amt (mg)}}{\text{dilution amt (mL)}}\right] \times \left[\frac{1000}{60 \text{ min}}\right] / \text{wt (Kg) x desired rate (mL/hr)}$$

COMMONLY USED CARDIAC PHARMACOLOGIC AGENTS

Adrenergic Agonist (Inotropic Agents)	Dopamine Dobutamine	Epinephrine Norepinephrine	Isoproterenol Calcium	
Phosphodiesterase Inhibitors	Amrinone	Milrinone		
Vasodilators	Nitroprusside	Nitroglycerin	Hydralazine	Enalaprilat
Ductal Manipulators	Prostaglandin E_1	Indomethacin		
Pulmonary Hypertension	Nitric Oxide Prostacyclin	Iloprost Bosentan	Sildenafil	
Vasoconstrictors	Phenylephrine	Vasopressin		
CHF Diuretics	Furosemide Ethacrynic acid	Bumetanide Chlorothiazide	Hydrochlorothiazide Metolazone	Spironolactone
ACE Inhibitors	Captorpil	Enalapril		
Angiotensin II Receptor Blockers	Losartan	Candesartan		

EFFECTS OF INOTROPIC AGENTS ON ADRENERGIC RECEPTORS

Drug	DA (dopaminergic)	α (alpha)	$ß_1$ (beta 1)	$ß_2$ (beta 2)
Dopamine	+	–/+ (high dose)	+	+
Dobutamine	–	–	+	–
Epinephrine	–	+	+	+
Norepinephrine	–	+	+	–
Isoproterenol	–	–	+	+
Phenylephrine	–	+	–	–

+ = positive effect; – = no significant effect

ADRENERGIC AGONISTS HEMODYNAMIC EFFECTS

Drug	CO (Cardiac output)	TPR (Temperature, pulse, respirations)	BP/MAP	Renal Perfusion
Dobutamine	↑	↓	↑	↔
Dopamine	↑	↑/↓*	↔/↑*	↑*
Epinephrine	↑	↓	↑	↓
Norepinephrine	↔/↓	↑	↑	↓
Isoproterenol	↑	↓	↓	↑/↓
Phenylephrine	↓	↑	↑	↓

↑ = increase effect; ↓ = decrease effect; ↔ = no effect; * dose related

COMMONLY USED ANTI-DYSRHYTHMIA AGENTS

Class IA (Na^+ channel blocker, prolong repolarization and antivagal effects)	Class IB (Fast Na^+ channel blocker, shortens action potential and repolarization)	Class IC (Na^+ channel blocker with variable effects on repolarization)	Class II (ß- blocker)	Class III (K^+ channel blocker, prolonged repolarization	Class IV (Ca^+ channel blocker)	Non-classified
Procainamide Disopyramide	Lidocaine Mexiletine	Flecainide	Esmolol Propranolol	Amiodarone Sotalol	Verapamil	Digoxin (Cardiac glycoside) Adenosine (K^+ channel opener, hyper-polarization)

COMMONLY USED RAPID SEQUENCE INTUBATION DRUGS

Medications	Sedatives	Paralytics
Atropine Lidocaine	Thiopental Midazolam Propofol Etomidate Fentanyl Ketamine	Succinylcholine Vecuronium Pancuronium Rocuronium

COMMONLY USED AIRWAY MEDICATIONS/THERAPIES

ß-Adrenergic Agents/ Sympathomimetics	Albuterol ($ß_2$) Metaproternol Terbutaline Salmeterol ($ß_2$)	Levalbuterol ($ß_2$) Isoproterenol Fenoterol	Pirbuterol Bitolterol Epinephrine
Methylxanthines	Theophylline	Aminophylline	
Steroids (use with H_2 Blocker Prophylaxis)	Methylprednisolone Hydrocortisone Budesonide Dexamethasone	Beclomethasone Prednisone Prednisolone	Fluticasone Triamcinolone acetonide Mometasone furoate
Anticholinergics	Ipratropium bromide Oxitropium bromide	Atropine sulfate	Glycopyrrolate
Mast Cell Stabilizers	Nedocromil sodium	Cromolyn sodium	
Leukotrine Modifiers	Montelukast sodium	Zafirlukast	Zileuton
Mucolytics	Acetylcysteine		
Heliox	Helium-oxygen (80:20 or 70:30) mixture		
Inhalation/Anesthetic Agents	Sevoflurane Isofluran	Halothane	Enflurane
Other	Magnesium sulfate Ketamine Sedation	Analgesia Neuromuscular blocking agents Anti-inflammatory agents	Antibiotics Mechanical ventilation ECMO

BACTERIAL PROPHYLAXIS REGIMENS

Procedural Prophylaxis Regimen	Time Prior To Procedure	Drug	Dosage	
			Adult	Pediatric
Dental	One hour	Amoxicillin po	2 gm	50 mg/Kg
Dental–penicillin allergy	One hour	Clindamycin po ***or***	600 mg	20 mg/Kg
		Cephalexin po ***or***	2 gm	50 mg/Kg
		Azithromycin po ***or***	500 mg	15 mg/Kg
		Clarithromycin po	500 mg	15 mg/Kg
Dental	30 minutes	Ampicillin IV/IM	2 gm	50 mg/Kg
Dental–penicillin allergy	30 minutes	Clindamycin IV ***or***	600 mg	20 mg/Kg
		Cefazolin IV/IM	1 gm	25 mg/Kg
GI/GU procedure	One hour	Amoxicillin po	2 gm	50 mg/Kg
GI/GU procedure	30 minutes	Ampicillin IV/IM	2 gm	50 mg/Kg
		plus Gentamicin IV/IM (high risk pts)	1.5 mg/Kg (max dose 120 mg)	1.5 mg/Kg
GI/GU–penicillin allergy	30 minutes	Vancomycin IV (over 1 hour)	1 gm	20 mg/Kg
		plus Gentamicin IV/IM (high risk pts)	1.5 mg/Kg (max dose 120 mg)	1.5 mg/Kg

IV = intravenous; IM = intramuscular; po = oral

IMMUNIZATION GUIDELINES (www.CDC.gov/vaccines/recs/schedules)

AGE / VACCINE	Months									Years				
	Birth	1	2	4	6	12	15	18	19-23	2-3	4-6	7-10	11-12	13-18
Rotavirus			*Rota	*Rota	*Rota									
Hepatitis B (3 doses)	*Hep B	+Hep B			+Hep B (final dose >24 weeks age)								▪Hep B (catch up)	
Diphtheria, Tetanus, Pertussis			*DTaP	DTaP*	*DTaP		+DTaP (final dose)				+DTaP		▪Tdap (catch up)	▪Tdap
Haemophilus Influenzae Type b			*Hib	Hib*	*Hib	+Hib (dose can be used as booster)								
Pneumococcal			*PCV	*PCV	*PCV	+PCV				•PPV (high risk)				
Inactivated Poliovirus			*IPV	*IPV	+IPV						+IPV	▪IPV (catch up)		
Influenzae					+Influenza (yearly vaccination)									
Mumps, Measles, Rubella						+MMR					+MMR	▪MMR (catch up)		
Varicella						+Varicella					+Vari-cella	▪Varicella (catch up)		
Hepatitis A						+Hep A (2 doses, 6 months apart)				•Hep A (high risk)				
Meningococcal										•MCV 4 (2-10 years high risk)			+MCV 4	•MCV4 (high risk)
Human Papillomavirus (females only)													+HPV (3 doses)	▪HPV (catch up)

*Minimum age to receive or begin series of vaccinations

+Range for recommended age to receive and/or complete vaccinations

♦Children at high risk (i.e. immunocompromised)

■Children who did not receive vaccinations when recommended

Neurologic and Trauma Scores

GLASGOW COMA SCORE FOR NEUROLOGICAL ASSESSMENT

	Pediatrics		Infants	
	Response	**Scoring**	**Response**	**Scoring**
Eye Opening	Spontaneous	4	Spontaneous	4
	To speech	3	To speech	3
	To pain	2	To pain	2
	None	1	None	1
Verbal Response	Oriented	5	Coos, babbles	5
	Confused conversation	4	Irritable, cries	4
	Inappropriate words	3	Cries to pain	3
	Incomprehensible sounds	2	Moans to pain	2
	None	1	None	1
Motor Response	Obeys	6	Normal, spontaneous	6
	Localizes	5	Withdraws to touch	5
	Withdraws	4	Withdraws to pain	4
	Abnormal flexion	3	Abnormal flexion	3
	Extends	2	Abnormal extension	2
	None	1	None	1
	Total Possible Score	**3-15**	**Total Possible Score**	**3-15**

DEGREE OF BURNS

	First	Second	Second	Third	Fourth
Depth	**Superficial**	**Superficial Partial Thickness**	**Deep Partial Thickness**	**Full Thickness**	**Full Thickness**
Characteristics	Localized pain, erythema, dry, no blistering	Red to pale ivory color, moist, blisters, severe pain	Mottled, white, waxy areas, dry surface. May be difficult to differentiate from full thickness	Burn color white, cherry red, brown or black; dermis elasticity destroyed, dry and leathery, burn area painless. May require escharotomies	Fat, fascia, muscle, bone involved
Common causes	Sunburn	Flash burns, scalding or brief contact with heat source	Flash burns, scalding or brief contact with heat source	Flame burns, chemical or electrical burns; prolonged contact with heat source	Electrical or deep thermal burns

PEDIATRIC CEREBRAL PERFORMANCE CATEGORY (PCPC)

Score	Category	Description
1	Normal	• Normal, at age-appropriate level; school-age attends regular class
2	Mild disability	• Conscious, alert, interact age appropriately • School-age attends regular class but may not be appropriate for age • Possible mild neurological deficit
3	Moderate disability	• Conscious, sufficient cerebral function for age-appropriate independent activities of daily living (ADLs) • School-age attends special education classes, learning deficit
4	Severe disability	• Conscious, dependent on others for ADLs, impaired brain function
5	Coma, vegetative state	• Cerebral unresponsiveness, no evidence of brain cortex function • Potential for some reflexive responses
6	Brain death	• Apnea, areflexia, silent EEG

PEDIATRIC TRAUMA SCORE

Component	+2	+1	–1	Total Score
Weight (Kg)	>20 Kg	10-20 Kg	<10 Kg	9-12 = Minor trauma
Airway	Patent	Maintainable	Unmaintainable	6-8 = Possible life threatening
Systolic BP	>90 mmHg	50-90 mmHg	<50 mmHg	0-5 = Life threatening
Pulses	Palpable radial/brachial	Palpable groin	Non-palpable	<0 = usually fatal
Central nervous system	Awake	Obtunded	Comatose	
Fractures	None	Closed or suspected	Multiple closed or open	
Wounds	None	Minor	Major, penetrating or burns >10%	

INTRA-ABDOMINAL COMPARTMENT GRADING SYSTEM

Grade	Bladder Pressure (mmHg)	Recommended Therapy
I	10-15 mmHg	Maintain normovolemia
II	16-20 mmHg	Hypovolemic resuscitation
III	21-25 mmHg	Abdominal decompression
IV	>25 mmHg	Abdominal decompression and re-exploration

QUICK REFERENCE

Additional Pediatric Scoring

PEDIATRIC INDEX MORTALITY SCORE (PIM2)

The PIM2 variables are recorded from initial physical contact with the ICU physician and the child lasting up to one hour after arrival. Common pitfalls using the PIM scoring system is over diagnosis of conditions. PIM is not used in decision making for management of individual patients, but is used to predict mortality in groups of patients.

Variable	Value (yes = 1; no or unknown = 0)
Elective admission	
Recovery post procedure	
Cardiac bypass	
High risk diagnosis: (0) None (1) Cardiac arrest preceding admission to ICU (2) Severe combined immune deficiency (3) Leukemia or lymphoma (4) Spontaneous cerebral hemorrhage (do not include if not intracerebral i.e. subdural haemorrhage) (5) Cardiomyopathy or myocarditis (6) Hypoplastic left heart syndrome (7) HIV infection (8) Liver failure primary admission diagnosis (9) Neurodegenerative disorder	Record # in parentheses that corresponds to diagnosis
Low risk diagnosis: (0) None (1) Asthma primary admission diagnosis (2) Bronchiolitis primary admission diagnosis (3) Croup primary admission diagnosis (4) Obstructive sleep apnea primary admission diagnosis (5) Diabetic ketoacidosis primary admission diagnosis	Record # in parentheses that corresponds to diagnosis
No pupillary response to light (>3 mm both fixed)	
Mechanical ventilation (during first ICU hour)	
Systolic BP (mmHg): Record value as 120 if unknown Record value as 0 if in cardiac arrest Record value as 30 if shocked	
Base excess (mmHg) arterial or capillary	
FiO_2/PaO_2 (mmHg)	

PEDIATRIC LOGISTIC ORGAN DYSFUNCTION SCORE (PELOD)

Values are measured in first 24 hours in the ICU. If measured more than once in 24 hour period, the most severe score is used for calculation.

Variable		Maximum Score
Cardiovascular	Heart rate	10
	Systolic BP	
Pulmonary	PaO_2 / FiO_2	20
	$PaCO_2$	
	Mechanical ventilation	
Neurologic	Glasgow Coma Score	20
	Pupillary reaction	
Hematologic	White blood cell count	1
	Platelet count	
Renal	Creatinine	10
Hepatic	SGOT	10
	Prothrombin or INR	
	Total Score	

DISSEMINATED INTRAVASCULAR COAGULATION (DIC) SCORE

Component	0	1	2	3	Max Points Possible
Platelet count (/mm^3)	>100,000	51-100,000	≤50,000		2
Fibrinogen (mg/dL)	≥100	<100			1
*FDP titer (mg/mL)	≤5		6-40	>40	3
*d-Dimer (mg/L)	≤2		2.1->8	>8	3
Prolonged PT (sec)	<3	3-6	>6		2
Total Score					**8**

DIC score ≥5 considered evident of DIC

*If d-Dimer is used instead of FDP titer

Neonatal Scoring Tools

APGAR (APPEARANCE, PULSE, GRIMACE, ACTIVITY, RESPIRATION)

Sign	+0	+1	+2
Appearance (color)	Blue, pale, blue extremities	Pink body	Pink body and extremities
Pulse (HR)	Absent	<100 bpm	>100 bmp
Grimace (reflex to suctioning)	None	Grimace	Coughing
Activity (muscle tone)	Limp, none	Slight flexion	Strong flexion
Respiration	Absent	Weak, irregular	Strong cry

(Score at one minute, 5 minutes and every five minutes until score ≥ 7)

SCORE FOR NEONATAL ACUTE PHYSIOLOGY AND PERINATAL EXTENSION (SNAPPE-II)

Variable	Description	Score
Mean arterial pressure (MAP)	>29 mmHg	0
	20-29 mmHg	9
	<20 mmHg	19
Corporal temperature	>35.6°C	0
	35-35.6°C	8
	<35°C	15
Birth weight (gms)	<999 gms	0
	750-999 gms	10
	<750 gms	17
SGA <3rd percentile (SGA = small for gestational age)	no	0
	yes	12
PO_2/FiO_2 ratio	>2.49	0
	1-2.49	5
	0.3-0.99	16
	<0.3	28
Lowest serum pH	>7.19	0
	7.10-7.19	7
	<7.10	16
Urinary output (UOP)	>0.9 mL/Kg/hr	0
	0.1-0.9 mL/Kg/hr	5
	<0.1 mL/Kg/hr	18
Multiple seizures	none	0
	yes	19
Apgar at 5 minutes	>7	0
	<7	18

Pediatric/Neonatal Pain Scoring Tools

CRIES (NEONATAL POST-OPERATIVE PAIN MEASUREMENT SCORE)

Criteria	0	1	2
Crying	None	High pitched	Inconsolable
Requires O_2 for sats <95%	None	<30% FiO_2	>30% FiO_2
Increased vital signs	HR/BP ≤ pre-op values	HR/BP increased <20% of pre-op	HR/BP increased >20% of pre-op
Expression	None	Grimace	Grimace, grunting
Sleepless	None	Frequent wake intervals	Constantly awake

FLACC PAIN SCALE (FACES, LEGS, ACTIVITY, CRY, CONSOLABILITY)

Categories	0	1	2
Face	• No expression or smile • Withdrawn, disinterested	• Occassional grimace, frown	• Constant quivering chin, clenched jaw
Legs	• Normal position, relaxed	• Restless, uneasy, tense	• Kicking, legs drawn up
Activity	• Quiet, normal position, moves easily	• Squirming, shifting, tense	• Arched, rigid, jerking
Cry	• No cry	• Moaning, whimpers, complaints	• Steady crying, screams or sobs, frequent complaining
Consolability	• Content, relaxed	• Distractible, console with touch, holding, talking	• Difficult to console or comfort

WONG-BAKER FACES (PAIN SCALE)

COMFORT SCALE

Alertness	Calm/ Agitation	Respiratory Response	Physical Movement	BP (MAP)	HR	Muscle Tone	Facial Expression	Points
Deep sleep	Calm	No cough or spontaneous breaths	None	Below baseline	Below baseline	No tone, relaxed	Relaxed	1
Light sleep	Slightly anxious	Spontaneous respirations, little or no response to ventilation	Occasional, slight movement	Consistent at baseline	Consistent at baseline	Decreased	Normal, no facial tension	2
Drowsy	Anxious	Occasional cough, resistance to ventilation	Frequent slight movements	Infrequent elevations >15% of baseline, observed 1-3 times	Infrequent elevations >15% of baseline, observed 1-3 times	Normal	Tension evident in some facial muscles	3
Fully awake and alert	Extremely anxious	Regular coughs and/ or actively breathing against ventilator	Vigorous movements with extremities	Frequent elevation of >15% observed >3 times	Frequent elevation of >15% observed >3 times	Increased muscle tone, finger and toe flexion	Tension evident throughout facial muscles	4
Hyper-alert	Panicked	Fighting ventilator, coughing and/ or choking	Vigorous movement extremities, torso and head	Sustained elevation >15%	Sustained elevation >15%	Rigid muscles, finger and toe flexion	Grimacing, contortion of facial muscles	5

Scores 8-16 points = deep sedation 17-26 points = light sedation 27-40 points = inadequate sedation

NEONATAL DRUG WITHDRAWAL SCORING SYSTEM

Signs/Symptoms	0	1	2	3
Tremors, muscle activity	Normal	Minimally increased when disturbed or hungry	Moderate or marked increase when disturbed or hungry. Subsides when fed or bundled	Marked increase or continuous when undisturbed, seizure like movements
Irritability, excessive crying	None	Slightly increased	Moderate to severe when disturbed or hungry	Marked increase even when undisturbed
Reflexes	Normal	Increased	Markedly increased	
Stools	Normal	Explosive with normal frequency	Explosive, >8/day	
Muscle tone	Normal	Increased	Rigid	
Skin abrasions	No	Redness on knees and elbows	Breaking of the skin	
Respiratory rate/minute	<55 breaths/minute	55-75 breaths/minute	76-95 breaths/minute	
Repetitive sneezing	No	Yes		
Repetitive yawning	No	Yes		
Vomiting	No	Yes		
Fever	No	Yes		

Common Laboratory Tests

Test	Conventional Units	SI Units (International)
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 (Cl^-)	98-107 mEq/L	98-107 mmol/L
Carbon Dioxide (CO_2)	13-22 mEq/L (newborn) 20-28 mEq/L (infant/child)	13-22 mmol/L (newborn) 20-28 mmol/L (infant/child)
Bicarbonate (HCO_3)	16-24 mEq/L (<2 years) 22-26 mEq/L (>2 years)	16-24 mmol/L (<2 years) 22-26 mmol/L (>2 years)
Glucose (BS)	40-80 mg/dL (newborn) 60-100 mg/dL (child/adult)	2.2-4.5 mmol/L (newborn) 3.3-5.6 mmol/L (child/adult)
Blood Urea Nitrogen (BUN)	4-12 mg/dL (newborn) 5-18 mg/dL (infant/child)	1.4-4.3 mmol/L (newborn) 1.8-6.4 mmol/L (infant/child)
Creatinine	0.3-1.0 mg/dL (newborn) 0.2-0.7 mg/dL (infant/child) 0.6-1.2 mg/dL (adolescent/adult)	27-88 µmol/L (newborn) 18-62 µmol/L (infant/child) 44-115 µmol/L (adolescent/adult)
Ionized Calcium	4.2-5.5 mg/dL	1.05-1.38 mmol/L
Total Calcium	8.6-10.8 mg/dL	2.2-2.5 mmol/L
Magnesium	1.3-2.0 mEq/L	0.65-1.0 mmol/L
Bilirubin (total)	<12 mg/dL (newborn) <1.2 mg/dL (infant) 0.3-1.2 mg/dL (child/adult)	<205 µmol/L (newborn) <21 µmol/L (infant) 5-21 µmol/L (child/adult)
Bilirubin (conjugated)	<0.6 mg/dL (newborn) <0.2 mg/dL (infants/child)	<10 µmol/L (newborn) <3.4 µmol/L (infants/child)
Albumin	2.1-5.7 gm/dL (newborn/infant) 2.9-5.8 gm/dL (child) 3.0-5.4 gm/dL (adolescent/adult)	2.1-5.7 gm/dL (newborn/infant) 2.9-5.8 gm/dL (child) 3.0-5.4 gm/dL (adolescent/adult)
Total protein	3.6-7.5 gm/dL (newborn/infant) 4.9-8.1 gm/dL (child) 6.0-8.0 gm/dL (adolescent/adult)	3.6 -7.5 gm/dL (newborn/infant) 4.9-8.1 gm/dL (child) 6.0-8.0 gm/dL (adolescent/adult)
Amylase	5-65 U/L (newborn) 27-131 U/L (child/adult)	5-65 U/L (newborn) 27-131 U/L (child/adult)
Lipase	9-128 U/L (newborn/infant) 10-150 U/L (child) 10-220 U/L (adolescent/adult)	9-128 U/L (newborn/infant) 10-150 U/L (child) 10-220 U/L (adolescent/adult)

Common Laboratory Tests [CONT.]

Test	Conventional Units	SI Units (International)
Cholesterol	<170 mg/dL (child/adolescent) <200 mg/dL (adult)	
Lactate	5-20 mg/dL (venous) 5-14 mg/dL (arterial)	0.5-2.2 mmol/L (venous) 0.5-1.6 mmol/L (arterial)
Phosphorus (PO_4)	4.5-9.0 mg/dL (newborn/infant) 4.5-5.5 mg/dL (child) 2.7-4.5 mg/dL (adolescent/adult)	1.45-2.9 mmol/L (newborn/infant) 1.45-1.78 mmol/L (child) 0.87-1.45 mmol/L (adolescent/adult)
Alannine aminotransferase (ALT)	150-420 U/L (infant) 100-320 U/L (child) 100-390 U/L (adolescent) 30-120 U/L (adult)	150-420 U/L (infant) 100-320 U/L (child) 100-390 U/L (adolescent) 30-120 U/L (adult)
Aspartate aminotransferase (AST)	25-75 U/L (newborn) 10-60 U/L (infant/child) 15-45 U/L (adolescent/adult)	25-75 U/L (newborn) 10-60 U/L (infant/child) 15-45 U/L (adolescent/adult)
Hemotologic Studies		
Red blood cells (RBC)	3.0-6.5 million/µ L (0-6 months) 4.0-5.5 million/µ L (6 months-12 years) 4.5-6.0 million/µ L (males >12 years) 4.0-5.0 million/µ L (females >12 years)	3.0-6.5 x 10^{12}/L (0-6 months) 4.0-5.5 x 10^{12}/L (6 months-12 years) 4.5-6.0 x 10^{12}/L (males >12 years) 4.0-5.0 x 10^{12}/L (females >12 years)
White blood cells (WBC)	5,000-30,000/µ L (0-1 month) 5,000-17,000/µ L (1 month-4 years) 4,000-13,000/µ L (>4 years)	5-30 x 10^9/L (0-1 month) 5-17 x 10^9/L (1 month-4 years) 4-13 x 10^9/L (>4 years)
Hemoglobin (Hgb)	10.0-21.0 g/dL (<1 month) 9.5-14.0 g/dL (2 months-2 years) 11.5-15.0 g/dL (2 years-12 years) 13.0-16.0 g/dL (males >12 years) 12.0-15.5 g/dL (females >12 years)	100-21.0 g/L (<1 month) 95-140 g/L (2 months-2 years) 115-150 g/L (2 years-12 years) 130-160 g/L (males >12 years) 120-155 g/L (females >12 years)
Hematocrit (Hct)	31%-60% (<1 month) 30%-40% (2 months-2 years) 34%-45% (2 years-12 years) 39%-53% (males >12 years) 36%-50% (females >12 years)	0.31-0.60 (<1 month) 0.30-0.40 (2 months-2 years) 0.34-0.45 (2 years-12 years) 0.39-0.53 (males >12 years) 0.36-0.50 (females >12 years)

Common Laboratory Tests [CONT.]

Test	Conventional Units	SI Units (International)
Coagulation Studies		
Platelet count	150,000-350,000/mm^3	
Prothrombin time (PT)	<13-19 sec (0-6 months) 10-13 seconds (6 months-adult) >18 seconds (critical value 0-6 months)	
Plasma thrombin time (PTT)	60-70 seconds	
Activate partial thrombo-plastin time (APTT)	<38-52 seconds (0-6 months) 24-38 seconds (6 months-adult) >60 seconds (critical value 0-6 months)	
Fibrinogen	150-400 mg/dL (critical value <100 mg/dL)	1.5-4 g/L
PPP (platelet-poor plasma)	Negative	
Fibrinogen split product (FSP) or Fibrinogen degradation product (FDP)	<5 µg/mL	<5 mg/L
D-dimer	<0.5 µg/mL (negative)	
PT-INR	1.0 (normal) 2-3.5 (therapeutic range on anticoagulants)	
Bleeding time	<7 minutes (critical value >13 minutes)	
Activated clotting time (ACT)	107 ± 13 seconds	
Cardiac Biomarkers and Other Tests		
Creatine Kinase (CK)	10-200 U/L (newborn) 55-135 U/L (male) 30-135 U/L (female)	10-200 U/L (newborn) 55-135 U/L (male) 30-135 U/L (female)
CK-MM (muscle)	95-100%	
CK-MB (myocardial)	0-5%	
Troponin	0-0.1 µg/L	
B-type natriuretic peptide (BNP)	<100 pg/mL	
C-reactive protein	0-0.5 mg/dL	

Common Laboratory Tests [CONT.]

Therapeutic Drug Levels	Therapeutic Range	SI Units (International)
Amikacin	20-30µg/mL (peak)	
	<10µg/mL (trough)	
Cyclosporine (transplants)	BMT 130-280 ng/mL Heart, heart/lung, lung: 360-510 ng/mL (<6 months age) 280-440 ng/mL (6 months-2 years) 210-360 ng/mL (>2 years) Kidney 440-510 ng/mL (<1 month age) 360-440 ng/mL (3-6 months) 280-360 ng/mL (6-12 months) 130-210 ng/mL (>1 year)	
Cyclosporine (other)	130-280 ng/mL (nephrotic syndrome)	
Digoxin	0.8-2 ng/mL	
5-Flucytosine	25-100 µg/mL	
Gentamicin	4-10 µg/mL (peak) 0.5-2.0 µg/mL (trough)	
Phenobarbital	15-40 µg/mL (trough)	
Phenytoin	10-20 µg/mL (trough)	
Theophylline	10-20 µg/mL (trough)	
Tobramycin	4-10 µg/mL (peak)	
	0.5-2.0 µg/mL (trough)	
Trimethoprim	5-10 µg/mL (peak for 20 mg/Kg dose) 1-3 µg/mL (peak for 10 mg/Kg dose)	
Valproic acid	50-100 µg/mL (trough)	
Vancomycin	15-40 µg/mL (peak) 5-15 µg/mL (trough)	

Bibliography

ADVANCING CRITICAL CARE
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SINCE 1972

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