

Hemodynamic Monitoring in the Neonatal and Pediatric Cardiac Patient: What the Direct Care Nurse Needs to know

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Introduction:

Due to the rapidly changing hemodynamic states of children, the developmental changes that occur with age, and the wide range of size and weight, no single mode of hemodynamic and oxygen transport monitoring can consistently meet the needs of the heterogeneous intensive care unit (ICU) population. Some non-invasive modes such as echocardiography, may provide excellent information on the anatomic effect on alterations in cardiac output, but provide only qualitative estimates of cardiac function. More comprehensive monitoring of cardiac output in the pediatric cardiac intensive care setting may be obtained by one or more of the following modes. These guidelines provide indications for the use and interpretation of the data provided by these modes.

Critical Thinking Points:

- There is no substitute for sequential physical examination to evaluate the effectiveness (or lack thereof) of our interventions and therapeutic decisions
- Data received from all monitoring modes must be correlated with physical assessment and results from all monitoring modes
- Data should be evaluated using multiple, consecutive points
- Changes in therapy should be evaluated with the same monitoring modalities

Invasive Monitoring Modes:

- **Venous oximetry**
 - Mixed venous oxygen saturation (SVO₂) reflects the hemoglobin-bound oxygen remaining following systemic oxygen extraction (VO₂)
 - A surrogate measure of adequate oxygen delivery (DO₂)
 - Site of measurement
 - True mixed SVO₂ is measured from pulmonary artery
 - May be approximated by measuring SVO₂ using central catheter positioned at the right atrium (RA), superior vena cava (SVC), or IVC-RA junction
 - Normal SVO₂ is 25-30 points less arterial saturation (SaO₂)
 - Causes of Low SVO₂
 - Low cardiac output state

- Hypoxia
 - Anemia
 - Increased metabolic rate
 - Causes of Elevated SVO2
 - High cardiac output state
 - Supra normal oxygen delivery
 - Poor oxygen extraction
 - Decreased metabolic rate
 - Advantages
 - More timely/sensitive at detecting impaired oxygen transport than lactate
 - May allow for intervention prior to tissue hypoxia & increased lactate
 - Disadvantages
 - True mixed SVO2 measurement requires pulmonary arterial catheter placement
 - Central SVO2 measurements are impacted by catheter location
 - RA placement may have decreased SVO2 measurement due to lower oxygen content of blood returning from the coronary sinus or may have increased SVO2 measurement due to higher oxygen content of blood returning from the renal arteries
 - SVC vs. IVC measurement variation related to differences between cerebral and somatic perfusion
 - Central SVO2 measurements may be impacted by intracardiac shunts
- **Lactate**
 - Weak anion that is a byproduct of anaerobic metabolism, which can result in an elevated anion gap metabolic acidosis
 - Causes of elevated lactate
 - Shock, regional tissue hypoxia
 - Seizures
 - Diabetic ketoacidosis
 - Toxins: alcohol, cocaine, carbon monoxide, cyanide
 - Medications: epinephrine, linezolid, metformin, propofol, acetaminophen, theophylline
 - Miscellaneous: thiamine deficiency, malignancy, liver dysfunction
 - Measurement and Levels
 - Arterial and venous samples correlate well, normal levels vary by assay used
 - Levels should be measured within 15 minutes of obtaining sample
 - Treatment and Prognosis

- Elevated lactate levels and the inability to clear lactate is associated with increased mortality in children after congenital heart surgery
 - Treatment should be targeted at underlying cause, typically optimizing oxygen delivery
 - If cardiac output and oxygen delivery have been maximized and lactic acidosis remains, other underlying causes should be investigated
- **Pulse Index Continuous Cardiac Output Monitor (PiCCO)**
 - Continuous monitoring of hemodynamic variables via arterial and central venous catheters
 - Catheter placement
 - Requires PiCCO specific arterial catheter with attached thermistor
 - Most commonly placed in femoral artery
 - Radial, brachial or axillary artery may also be used
 - Infusion port attaches to standard central venous catheter
 - Catheter should be in central position, within or adjacent to right atrium
 - Femoral vein placement overestimates intrathoracic volume measurements
 - If femoral vein is used, avoid ipsilateral femoral artery catheter, as injection too close may impact thermistor measurements
 - Trans-cardiopulmonary thermodilution
 - Known quantity of cold injectate is infused through central venous catheter
 - Thermistor in arterial line measures change in temperature over time
 - Calculates area under the curve for cardiac output measurement using Stewart-Hamilton formula: $CO = [V1(TB - T1)K1K2] / [S(\Delta TBdt)]$
 - V1 = injectate volume
 - TB = blood temperature
 - T1 = injectate temperature
 - K1 = empiric factor to correct for warming in catheter
 - K2 = computation constant
 - Change in temperature over time (AUC)
 - Anatomical sources of error: regurgitant valve lesions, intracardiac shunts
 - Continuous measurements by pulse contour analysis
 - Heart rate, systolic, diastolic and mean arterial blood pressures, central venous pressure (CVP), SVO2
 - Stroke volume
 - Area under systolic portion of the arterial waveform

- Accuracy impacted by aortic and arterial compliance, which varies significantly among patients/disease states
- Cardiac output (from heart rate [HR] and stroke volume)
- Systemic vascular resistance (from mean arterial pressure (MAP) and cardiac output [CO])
- Pulse pressure variation
 - Variation between changes in systolic and diastolic BP over time
 - >10-13% variability predicts increased CO in response to fluid challenge
- Stroke volume variation
 - Variation of area under systolic portion of arterial waveform over time
 - >10-13% variability predicts increased CO in response to fluid challenge
- Intermittently measured parameters by thermodilution
 - Cardiac Output (CO)
 - Mean transit time (MTT): time for half of the indicator to pass the thermistor
 - Exponential decay time (EDT): derived from slope of time-concentration as it returns to baseline
 - Intrathoracic thermal volume (ITTV) = CO x MTT
 - Pulmonary Thermal Volume (PTV) = CO x EDT
 - Global end-diastolic volume (GEDV)= ITTV-PTV
 - Measurement of total volume in all four chambers of the heart at the end of diastole, a measure of preload
 - May be better than CVP in predicting fluid responsiveness
 - Intrathoracic Blood Volume (ITBV) = GEDV x 1.25
 - Extravascular lung volume= ITTV-ITBV
 - Proposed to measure pulmonary edema better than CXR/exam
 - Cardiac function index (CFI)= CO / GEDV
- Advantages
 - Avoids invasiveness of pulmonary artery catheter placement
 - Measures and derives multiple hemodynamic variables, some continuously
- Disadvantages
 - Requires intra-arterial and central venous catheters
 - Unreliable data in patients with intracardiac shunts or valvar lesions
 - Many parameters are derived, not all parameters are validated

Non-invasive Monitoring Modes:

- **Near Infra-Red Spectroscopy (NIRS)**
 - Continuous measurement of regional venous oxygen saturation (rSO₂)
 - Measures the amount of oxygenated hemoglobin using infra-red spectrum (700-900 nm) light absorption and scatter, based on Beer-Lambert Law
 - Oxygenated hemoglobin absorbs more at higher wavelengths than deoxygenated
 - Measurement from continuous absorption approximates venous concentrations
 - NIRS probe contains a light source and shallow and deep absorption detectors
 - Shallow values are subtracted from deep to eliminate contribution of superficial tissue
 - Single site placement may be used to assess specific regional oxygenation
 - Cerebral
 - Correlates with jugular bulb saturations in infants and children
 - Normal values
 - Healthy or acyanotic heart disease 60-80%
 - Cyanotic heart disease: 45-60%
 - Somatic
 - Typically dorsal lateral flank or anterior abdomen device placement
 - Baseline values vary between tissue beds and individuals
 - Normally > 10% above cerebral rSO₂
 - Multisite placement allows for regional determination of oxygenation and determination of somatic-cerebral rSO₂ difference
 - Interference : increased superficial tissue, melanin, bilirubin, body wall edema
 - Causes of low rSO₂
 - Low cardiac output state
 - Decreased regional perfusion
 - Hypoxia
 - Increased metabolic rate, global or regional
 - Anemia
 - Specific treatment thresholds have not been well validated
 - Cerebral rSO₂ <50% for prolonged periods associated with worse neurological outcomes

- Somatic rSO₂-cerebral rSO₂ difference <10 may indicate decreased somatic perfusion
- Somatic rSO₂ – cerebral rSO₂ difference approaching zero is predictive of biochemical shock and longer ICU stay
- NIRS trends may be more useful than specific values
- Advantages
 - Continuous monitoring, may serve as an early indicator of alterations in cardiac output (prior to SVO₂ or lactate)
 - Non-invasive
- Disadvantages
 - Lack of consensus on specific critical thresholds
 - Correlation to outcome not well validated