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Hemodynamic Monitoring in the Acute Management of Pediatric Heart Failure

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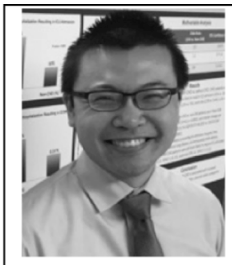

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Abstract: One of the basic tenets of cardiac critical care is to ensure adequate tissue oxygenation. As with other critical illness such as trauma and acute myocardial infarction studies have demonstrated that making the right diagnosis at the right time improves outcomes. The same is true for the management of patients at risk for or in a state of shock. In order to optimize outcomes an accurate and timely assessment of cardiac function, cardiac output and tissue oxygenation must be made. This review discusses the limitations of the standard assessment of cardiovascular function, and adjunctive monitoring modalities that may be used to enhance the accuracy and timely implementation of therapeutic strategies to improve tissue oxygenation.

Keywords: Cardiac output, hemodynamics, monitoring, near infrared spectroscopy, pediatrics, tissue oxygenation, venous oximetry.

INTRODUCTION

The driving principle of intensive care medicine is to optimize the relationship between oxygen supply and demand and in doing so ensure adequate tissue oxygenation [1]. One of the primary roles of the cardiac intensivist is to foresee deviations from the expected and desired clinical trajectory and to implement the correct strategies in a timely manner in order to optimize outcomes [2]. The notion of doing the right thing at the right time is exemplified in the "golden hour" of trauma and is no less applicable to the management of children at risk for or in a state of cardiogenic shock. The types of hemodynamic monitoring used in the cardiac intensive care unit are numerous and diverse. This review will outline standard hemodynamic monitoring, discuss their limitations, and review additional monitoring modalities including serum lactate levels, near-infrared spectroscopy (NIRS), venous oximetry, and transpulmonary thermodilution.

STANDARD HEMODYNAMIC PARAMETERS

Pulse Rate

The field of critical care medicine evolved from the treatment and resuscitation of wounded soldiers in Normandy and Korea in the 1940s and 50s. In the Mobile Army Surgical Hospital (MASH) units, physician caring for critically ill patients discovered the importance of monitoring vital signs. Perhaps the most basic and fundamental of vital signs is pulse rate. While a single solitary heart rate can offer a glimpse of the patient's clinical state, monitoring trends and changes in heart rate are essential to the cardiac intensivist in their determination of the patient's hemodynamic state and trajectory. Changes in heart rate can be indicative of but not specific for changes in ventricular preload, function and output, as well as be reflective of a change in metabolic demand. Because changes in heart rate are generally nonspecific they need to be considered within the clinical context. Heart rate trends may be useful for example in determining the optimal ventricular filling pressure, as discussed below. An assessment of cardiac rhythm is an essential part of the management of the critically ill patient where electrocardiographic monitoring is routinely used in the cardiac intensive care unit to survey for arrhythmias that may occur, particularly following cardiac surgery.

Systemic Arterial Blood Pressure

Systemic arterial blood pressure monitoring is another mainstay of standard hemodynamic monitoring. Noninvasive blood pressure monitoring is usually obtained using oscillometric devices such as the Dinamap (Device for Indirect Measurement of Blood Pressure). Studies have shown that oscillometric devices such as Dinamap give comparable blood pressure readings when measured against indwelling arterial catheters [3]. Nonetheless, they are not without their limitations. Studies have found that noninvasive oscillometric devices are less reliable for diastolic blood pressure and are also less accurate at lower blood pressure and during clinical shock [4, 5]. An additional limitation is the intermittent nature of non-invasive pressure monitoring.

Indwelling arterial catheters are considered the standard of care for the management of hemodynamically unstable

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patients, providing a continuous display of the arterial waveform and additional information not available with the non-invasive modality. The upstroke of the arterial waveform provides information about cardiac function. If the rate of rise of the pressure wave is rapid, contractile function is good. A slow upstroke can be indicative of poor cardiac function but is also seen in aortic stenosis and with elevations in systemic vascular resistance. The area under the systolic portion of the waveform is proportional to the stroke volume. A low pulse pressure may reflect a low stroke volume. A widened pulse pressure is seen in states characterized by diminished vascular tone, in patients with an aorta to pulmonary arterial runoff and in patients with aortic insufficiency. Indwelling catheters also facilitate the titration of vasoactive medications, particularly afterload reducing agents; allow for an assessment of the response to volume expansion; and they permit frequent arterial blood gas sampling to assess the adequacy of gas exchange. An important limitation of blood pressure is that it is the product of cardiac output and systemic vascular resistance, thus it may not be an accurate indicator of cardiac output.

Central Venous Pressure

Central venous pressure monitoring allows for the continuous assessment of ventricular filling pressures and pressure waveforms and is commonly used in the cardiac intensive care unit. Because ventricular compliance is affected by a number of conditions including positive pressure ventilation and cardiac disease, a determination of the optimal ventricular filling pressure is indicated. If volume administration increases stroke volume then the ventricle is operating on the ascending portion of its pressure stroke volume curve and preload reserve is present. The presence of preload reserve is reflected in a prompt decrease in heart rate, increase in the systolic blood pressure or improvement in venous oximetry following volume administration. If there is no improvement in these parameters, preload reserve is exhausted and additional fluid will only increase pulmonary congestion without improving output. Its worth noting that the determination of the optimal ventricular filling pressure is an on-going exercise, as ventricular compliance may vary over time due to changes in respiration (see cardiopulmonary interactions) and ventricular function [6, 7]. Further confounding the determination of the optimal ventricular filling pressure is the fact that there is no correlation between the central venous or right atrial pressure and left atrial pressure in patients with cardiac and or pulmonary disease [8]. Finally, it is worth noting that studies have shown that there is a very good correlation between femoral venous and central venous pressures so long as there is a continuous column of fluid between the two sites of measurement [9].

End-tidal CO_2 Monitoring

The concentration of CO_2 in expired gas is predicated on tissue CO_2 production, pulmonary blood flow and alveolar ventilation. In capnography, infrared devices measure the concentration of CO_2 in inhaled and exhaled gases over time. The normal baseline on the capnogram should have a CO_2 concentration of zero, reflecting inspiratory as well as early expiratory gas; this is followed by a sharp upstroke reflecting mid-exhalation and increasing alveolar gas; this is followed by the plateau phase, which represents a leveling off of alveolar gas; the capnogram then abruptly falls to zero, as the expiratory phase is terminated and inspiratory gas dilutes out the remaining CO_2. There is a minimal difference between measured arterial CO_2 ($PaCO_2$) and the end tidal CO_2 level ($P_{ET}CO_2$) in normal subjects due to minor abnormalities in the relationship between alveolar ventilation to perfusion. However, when pulmonary perfusion is limited lung units with large ventilation to perfusion ratios may be created, particularly in the setting of positive pressure ventilation. This leads to an inefficiency in CO_2 removal or wasted ventilation and the creation of a large arterial to end tidal CO_2 gradient. Increasing ventilator support will be of limited benefit in improving CO_2 removal and may very well worsen the extent of wasted ventilation. Strategies that decrease airway pressure and improve pulmonary perfusion will decrease the extent of wasted ventilation, improve CO_2 removal and improve cardiac output (see cardiopulmonary interactions elsewhere is this issue).

Limitations of the Standard Assessment of Cardiac Function and Cardiac Output

While the physical examination and the interpretation of standard hemodynamic parameters (heart rate, central venous pressure, blood pressure) is essential to the management of the critically ill patient, estimations of cardiac function, cardiac output and the adequacy of tissue oxygenation based on an analysis of these standard clinical parameters are often discordant from measured values. This has been shown to be the case in numerous studies and is present across disciplines and levels of experience with the most senior care providers fairing no better than more junior care providers [10-12]. These findings provide the rationale for enlisting additional tools for monitoring cardiovascular function.

Serum Lactate Levels

When metabolic demand exceeds oxygen delivery serum lactate will rise as lactate production outpace its clearance [1]. The constant formation of lactate during glycolysis and it continuous conversion back to pyruvate is called lactate shuttle, and it is believe to be a part of cellular signaling in addition to its role in cellular metabolism [13]. The normal serum lactate level is less than 1.5 mmol/L. An elevated or rising lactate level is often seen as an indicator of inadequate oxygen delivery and cellular hypoxia in the intensive care unit. Nonetheless, studies have found that there are nonhypoxic reasons for elevated serum lactate [14]. Hyperlactemia may be encountered after cardiopulmonary bypass in the post-operative setting and may not be associated with tissue hypoxia but rather may be reflective of a "washout" phenomenon after resumption of the normal circulation [15-17]. In any case, sustained or rising serum lactate level should not be assumed to be the result of a "washout" and should be promptly addressed. The association between hyperlactemia and morbidity and mortality following surgery is well known. In a prospective study, Charpie and colleagues monitored serum lactate levels in 46 patients who were less than 1 month old following cardiac surgery [18]. With the primary outcome defined as need for extracorporeal membrane oxy-

genation (ECMO) or death, 9 patients required ECMO or died and 37 patients did not. In those whose postoperative course resulted in ECMO or death, the average initial serum lactate level was significantly higher (9.4 ± 3.8 mmol/L vs. 5.6 ± 2.1 mmol/L; P = .03) However, the positive predictive valve for the initial postoperative lactate level was only 38%. But when changes in lactate level were trended overtime, an increase in the lactate level of 0.75 mmol/L per hour or more was strongly associated with a poor outcome (P < .0001) such as the need for ECMO and death and the positive predictive valve was 100%.

Venous Oximetry

The Fick principle states that when there is a decrease in oxygen delivery to the tissues, the arteriovenous oxygen content difference increases due to an increase in oxygen extraction. Furthermore, it is only when cardiac output and oxygen delivery drops below a critical level that an increase in oxygen extraction can no longer fully compensate, resulting in anaerobic metabolism and elevated serum lactate levels [1]. Therefore, venous oximetry provides an earlier indication of a waning cardiac output and oxygen delivery than serum lactate levels [1]. As oxygen delivery falls, the oxygen extraction ratio rises [oxygen extraction ratio: $O_2ER = SaO_2 – ScvO_2 / SaO_2$, where SaO_2 and $ScvO_2$ are arterial and central venous oxygen saturations, respectively. The normal O_2ER is 25-30%, and as it rises above 50-60% anaerobic metabolisms ensues, defining the critical O_2ER. When the critical O_2ER is exceeded serum lactate production begins to exceed its clearance and levels begin to rise. An assessment of the O_2ER is not an exercise in determining cardiac function, cardiac output or systemic oxygen delivery. The O_2ER is however an indicator of the relationship between oxygen spply and demand. Because the mixed venous saturation is rarely available outside the cardiac catheterization lab, alternative sites (central venous) are used to monitor venous oxygenation saturations and include: the jugular vein; superior vena cava; right atrium, so long as there is no left to right atrial shunting; and the inferior vena cava right atrial junction [19, 20] The utility of venous oximetry in managing patients at risk for or in a state of shock has been demonstrated by studies in adults and children with septic shock and well as in neonates following the Norwood procedure for the hypoplastic left heart syndrome [21, 22].

Near-infrared Spectroscopy

Similar to pulse oximetry near infrared spectroscopy (NIRS) assesses the concentration of oxygenated and deoxygenated hemoglobin by analyzing the absorption and scatter of near infrared light. However, unlike pulse oximetry, which analyzes the pulsatile signal in the circulation, NIRS samples the nonpulsatile component. And because a majority of blood in the microcirculation is venous (upwards of 75-80%) the oxygen saturation derived by NIRS is used as a surrogate for venous saturations for the viscera interrogated [23]. Studies have demonstrated a very good correlation between jugular saturations and cerebral NIRS derived oxygen saturations [24, 25]. Cerebral oxygen saturations are also used as a surrogate for central venous oxygen saturations and as such are used to assess the global relationship between oxygen supply and demand [26].

As with pulse oximeters, NIRS oximeters rely on a complex algorithm to convert the change in absorbance of at least two wavelength of light to an absolute saturation value. It is not a measurement of oxygen saturations. Thus, while the correlation of pulse and NIRS oximetry with measured values is very good the derived values may vary from measured values. Due to technical constraints such as these, the technology is limited to relative quantitation and is thus useful for tracking changes in a given patient. Not surprisingly, studies have found that low cerebral oxygen saturation are associated with worse long term neurological outcomes. A prospective observational trial conducted by Dent et al found that new or worsened MRI lesions were found in infants whose cerebral NIRS were below 45% for longer than 180 minutes during cardiac surgery [27]. Other studies have also demonstrated a significant correlation between depressed cerebral oxygenation intra- and postoperatively and the development of new or worsened neurologic injury and adverse neurodevelopmental outcomes [28-30].

The Pulmonary Arterial Catheter and Emerging Technologies

The pulmonary arterial catheter (PAC) provides continue measurements of right atrial and left atrial pressures, the latter obtained via a pulmonary artery occlusion pressure (PAOP). The PAOP is quite useful as studies have demonstrated a poor correlation between right and left atrial pressures in patients with underlying cardiopulmonary disease [8]. The PAOP also distinguishes between cardiogenic and permeability pulmonary edema. The PAC also allows for an assessment of pulmonary arterial pressures, which in conjunction with the PAOP enables the clinician to determine the etiology of the pulmonary hypertension (ie., left sided heart disease versus pulmonary arterial hypertension). In addition, the PAC may be used to measure cardiac output by utilizing the principles of thermodilution. A measurement of cardiac output also allows for a calculation of systemic and pulmonary vascular resistance. Controversy has arisen in recent years with regard to the use of the pulmonary arterial catheter in the adult and pediatric setting [31]. Proponents extol its ability to give hemodynamic data not available elsewhere while its opponents highlight the risks associated with its insertion and prolong use [32, 33]. In either case the information gleaned from the PAC may be essential in guiding therapy in patients who despite all efforts fail to progress and in whom additional hemodynamic data is essential to directing therapy.

Because questions swirl around the use of PACs in pediatric and adult patients, new technologies are emerging. A relatively new technology that may be used to measure cardiac output is pulse contour analysis [34]. This technology is based on the principle that the area under the curve of a central arterial waveform correlates with stroke volume. The PiCCO system (Pulsion Medical systemic, Munich, Germany) relies on a standard central venous catheter and a thermistor-tipped arterial catheter to generate a transpulmonary thermodilution-derived cardiac output. Studies have

shown an excellent correlation between cardiac output measured via the transpulmonary thermodilution method and the pulmonary artery thermodiluton method and measurements of cardiac output based on the direct Fick principle. The pulse contour analyzing technology allows for continuous monitoring of cardiac output however studies have found these measurements to be less reliable than those derived by arterial thermodiltion due to drift, necessitating frequent recalibrations.

CONCLUSION

The primary task of the cardiac intensivist is to monitor, rectify, and preserve adequate oxygen delivery relative to demand. It is always better to intervene before decompensated shock and end organ injury occurs, and therefore, the value of timely and accurate hemodynamic monitoring cannot be overstated. Although a variety of monitoring modalities are available, no single type of monitoring or measurement is without limitations. As such, they each provide a piece of the puzzle. It is incumbent on the cardiac intensivist to assimilate all of the available data provided by the various monitoring modalities and physical examination and to formulate a plan that is tailored to the individual patient's needs.

AUTHOR CONTRIBUTIONS

Authors Rocky Tsang, Paul Checchia and Ronald Bronicki contributed equally to the manuscript's researc, preparation, editing, revisions and completion.

CONFLICT OF INTEREST

Rocky Tsang, MD has no financial conflicts of interest.

Paul Checchia, MD, has no financial conflicts of interest.

Ronald Bronicki, MD, member of the speakers bureau for the Covidien Corporation.

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