Cardiovascular support during neonatal intensive care

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Abstract
Effective cardiovascular support for neonates requires an understanding of cardiovascular physiology, the developmental stages of the neonate and knowledge of the available treatment options. This review aims to provide physiologically-based recommendations for treatment, referring only to aspects of physiology that can be generally measured in neonates on neonatal units. This review is intended to give an insight into how physiology and pharmacology can be balanced when tailoring care to individual babies.

Keywords cardiac output; hypotension; neonatal; vasopressor

Introduction

Effective cardiovascular support for neonates requires an understanding of cardiovascular physiology that takes account of the developmental stages and knowledge of the available treatments. This review aims to provide physiologically-based recommendations for treatment, referring only to aspects of physiology that can be generally measured in neonates on neonatal units. Different aspects of physiology may be assessed on paediatric intensive care units.

Cardiovascular physiology

An adequately functioning circulation maintains oxygen and nutrient delivery to tissues and removal of waste products. This in turn is determined by the need for oxygen and nutrition by specific organs. An increased blood supply from the heart is possible when demanded by tissues under stress. In such circumstance the heart pumps more blood per minute through the cardiac cycle. This is accompanied by a constriction of arterioles in the peripheral tissues which increases peripheral resistance and maintains the arterial blood pressure. Normal circulatory function depends on three factors: cardiac function, vascular tone and blood volume. The arteriolar constriction during increased demand is selective. This leads to blood flow diverting from skin, gastrointestinal tract, kidneys and to heart and brain to conserve oxygenation to the most vital organs.

The goal of cardiovascular support is to maintain adequate tissue oxygen delivery. The concepts that underlie management strategies can be summarized in a set of formulae see Box 1.

A large number of neural, chemical and humoural factors are involved in the complex regulation of the cardiovascular system. Since these cannot be described in detail for each patient, clinical assessment and treatment need to be individualised using biomarkers and clinical judgement. Multiple factors within the cardiovascular system can fail during cardiovascular compromise. These factors provide ways to assess the adequacy of tissue oxygen delivery. For example, as tissue oxygen delivery falls aerobic metabolism becomes anaerobic. This anaerobic metabolism is highly inefficient consuming a large amount of glucose and produces lactic acid. This leads to changes on a blood gas that includes a drop in pH and bicarbonate, a rise in base deficit and lactate. It is important to be aware that the cardiovascular system is also affected by respiratory activity: there is a clear relationship between intrathoracic pressure fluctuations due to respiration. This can be particularly important with high levels of ventilatory support.

Tissue perfusion is difficult to measure directly and clinicians resort to biomarkers which are considered to represent tissue perfusion: changes in heart rate, blood pressure, skin perfusion, urine output and blood acid base balance and lactate concentration. These are not sensitive as clinically relevant cellular hypoxia and poor tissue perfusion may be present before there are clinically apparent changes.

Developmental factors

The neonatal myocardium lacks sarcoplasmic reticulum and has an increased ratio of fibrous non-contractile tissue and possess diminished sympathetic intervention. The structural differences present when compared to the heart of a term neonate means that the preterm heart has a high output at rest and lack of contractile reserve. The addition of an immature autonomic system present in the preterm neonate leaves it vulnerable to cardiovascular compromise.

Blood pressure

Blood pressure (BP) is the determinant of tissue perfusion which is the easiest to measure and so is the most commonly used biomarker of tissue perfusion. Blood pressure is determined by factors such as myocardial muscle strength, elasticity of blood vessels and blood volume. Blood pressure determinants are also important to consider when evaluating hypotension. Blood pressure is not a perfect indicator of cardiac output. Blood pressure may in fact increase as CO is falling due to the compensatory vasoconstriction and can therefore be misleading unless careful clinical assessment is conducted. Blood pressure can increase, decrease or remain unchanged depending on the degree of compensation in HR, stroke volume and SVR. As CO falls HR increases so that stroke volume decreases due to reduced filling. Accurate measurements of CO and SVR cannot be done easily. In

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Formulae that summarise key concepts in cardiovascular physiology

In these formulae, the variables that can be modified clinically are indicated in italics. The interrelationships between these clinically modifiable variables are indicated in Figure 1.
Oxygen delivery (DO_2) is the total amount of oxygen delivered to the tissue per minute and can be calculated from the formula:

$$DO_2 = \text{Cardiac output (CO)} \times \text{arterial oxygen content}$$
$$= CO \times (\text{oxygen bound to haemoglobin} + \text{dissolved oxygen})$$
$$= CO \times [(\text{cHb (g/dl)} \times SaO_2 \text{ x } 1.39) + (\text{dissolved oxygen})],$$ where 1.39 is the oxygen carrying capacity of haemoglobin.

As dissolved oxygen is negligible:

$$\text{Oxygen delivery to the entire body} = CO \times (\text{cHb (g/dl)} \times saO_2 \times 1.39)$$

$$\text{Oxygen delivery to the peripheral tissue} = \text{Peripheral blood flow} \times (\text{cHb (g/dl)} \times saO_2 \times 1.39).$$

The variables in italics can be treated to optimise tissue oxygenation. These include the maintenance of haemoglobin levels and an adequate CO.

$$\text{Cardiac output} = \text{Stroke volume} \times \text{HR}$$

$$\text{Stroke volume} \propto \textit{preload}$$

$$\text{Stroke volume} \propto \textit{contractility}$$

$$CO \propto \textit{Blood pressure}/\textit{systemic vascular resistance}$$

Box 1

summary, blood pressure can be an important determinant of tissue oxygen perfusion but must not be viewed in isolation.

Normal neonatal blood pressure values: blood pressure can be monitored during the neonatal period non-invasively by neonatal blood pressure cuff measurement, or invasively using an indwelling arterial catheter. The gold standard is intra-arterial measurement as small blood pressure cuffs can overestimate true blood pressure in the neonate. The commonly cited 'rule of thumb' definition of hypotension is mean blood pressure below an infants' gestational age in weeks. However it must be stressed that in clinical practice a normal blood pressure of a neonate is a blood pressure that achieves appropriate tissue perfusion.

Why is it important to treat cardiovascular insufficiency in neonates?

We know that cerebral perfusion depends on blood pressure in sick preterm neonates. Low blood pressure correlates with low

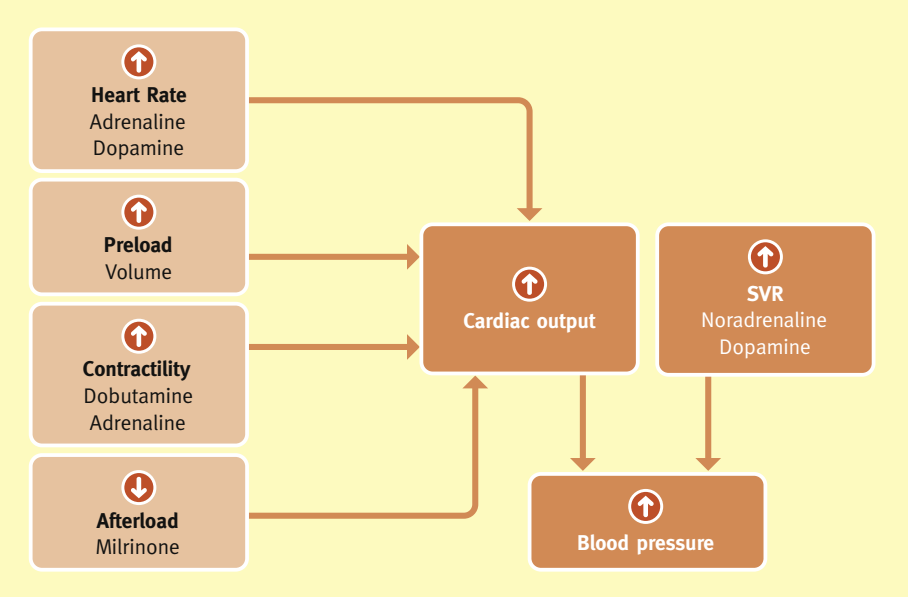

Figure 1 Interrelationships between key cardiovascular variables.

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