Haemodynamics and cardiovascular shock

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Abstract

Haemodynamics is the measurement of the circulation within the body. Shock exists when the perfusion of vital organs is inadequate. The use of haemodynamic monitoring aims to identify hypoperfusion early, and allow for its correction in a timely manner, before irreversible organ dysfunction occurs. This article outlines the basic physiology of the circulation and the mechanisms of different forms of shock. Current invasive and non-invasive methods of haemodynamic monitoring are described, such as may be encountered in theatre or in the Critical Unit.

Keywords Afterload; haemodynamic monitoring; preload; pulmonary artery catheter; pulse countour analysis; shock; stroke volume variation; systolic pressure variation

Blood flow around the body is dependent on the pump (i.e. the contractile force of the heart), the volume of circulating blood and the resistance to the flow of blood in the circulatory system. Resistance to flow is mainly determined by the calibre of the small arteries and arterioles. The mean arterial blood pressure (MAP) is determined by the volume of blood ejected by the ventricles over time, cardiac output (CO) and also by the tone of the small arteries and arterioles, total peripheral resistance (TPR).

This gives us following equation for calculating the MAP.

$$\mathrm{MAP} = \mathrm{CO} \times \mathrm{TPR}$$

Control of stroke volume: the volume of blood (in ml) ejected from the left ventricle with each contraction is determined by the preload, afterload, cardiac contractility and ventricular function.

Preload – the preload is the stretch produced in the ventricles at the end of diastole, when filling is maximal. The degree of stretch has a direct effect on the force of ventricular contraction in systole and the volume of blood ejected. This relationship is illustrated by Starling's law (Figure 1). End diastolic ventricular wall stretch is estimated by measuring the volume in the ventricles at the end of diastole LVEDV.

In an overfilled or failing heart, the ventricles are unable to increase contractile force and stroke volume. This is demonstrated by the flat portion of the Frank Starling curve, which shows no increase in stroke volume despite increased end diastolic volume.

Afterload – this is the 'load' that the ventricle must overcome before it can eject a stroke volume. The main determinant of afterload is increased arterial vascular tone (TPR). Increased afterload can be fixed, for example as in aortic stenosis, or dynamic for example in the presence of varying vascular tone.

Contractility – is a measure of the intrinsic ability of cardiac muscle to contract.

Shock

Shock is a reduction in the effective delivery of oxygen and other nutrients to the tissues. This initially causes transient organ dysfunction. If the shock state persists, tissue and organ dysfunction becomes irreversible. It is therefore important that shock is identified and treated promptly.

On a cellular level, normal function depends on oxygen delivery. This can be quantified by the following equation.

$$\text{Oxygen delivery } (\mathrm{DO_2}) = \text{cardiac output} \times \text{arterial oxygen content}$$

Arterial oxygen content is primarily determined by the amount of haemoglobin and its oxygen binding capacity. Dissolved oxygen usually contributes very little. This is summarized in the equation below:

$$\text{Oxygen content (ml/dL blood)} = (\mathrm{Hb} \times \mathrm{SaO_2} \times 1.34) + (\mathrm{PaO_2} \times 0.0225)$$

($\mathrm{SaO_2}$ = oxygen saturation of arterial blood, $\mathrm{PaO_2}$ = partial pressure of oxygen in arterial blood, in kPa)

Oxygen delivery to the tissues can therefore be increased by increasing the cardiac output and the oxygen content of the blood. The oxygen content can be increased by increasing the oxygen saturation and the amount of haemoglobin, for example by increasing the inspired oxygen concentration or by transfusing the patient.

Classification of shock

The shock state can be broadly defined by four different entities; cardiogenic, extracardiac obstructive, hypovolaemic and distributive shock. In all but distributive shock the major contributor to the shock state is diminished cardiac output. In distributive shock (e.g. septic shock), the initial mechanism for shock is profound vasodilation. Although cardiac output can be depressed in septic shock, cardiac activity is often hyperdynamic (low stroke volume and poor contractility being counterbalanced by a high heart rate).

Cardiogenic shock: cardiac contractility is depressed due to an abnormality in intrinsic cardiac function. This could be due to myocardial ischaemia, arrhythmias or valvular heart disease.

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Extracardiac obstructive shock: in this state the cardiac output is reduced not due to an intrinsic cardiac abnormality but because of obstruction to the ejection of a normal stroke volume. Examples include cardiac tamponade, where cardiac output is reduced by an effusion compromising both ejection and filling, initially of the right, thin walled ventricle, and subsequently biventricular impairment. Other causes are pulmonary embolism, where right ventricular ejection is prevented by an obstruction in the pulmonary arteries, and pneumothorax, when an increased intrathoracic pressure and mediastinal torsion restricts right ventricular filling, so causing a reduction in cardiac output.

Hypovolaemic shock: the acute loss of large volumes of fluid from the body (e.g. haemorrhage or burns) causes a reduction in circulating blood volume. Initial hormonal and neurogenic compensatory mechanisms act to increase vascular tone and fluid retention in order to maintain blood pressure. Shock will ensue when the loss of volume overcomes these compensatory mechanisms.

Distributive shock: the unifying problem in this category is a precipitous fall in vascular tone, which reduces blood pressure and compromises preload, reducing cardiac output. Examples include sepsis, neurogenic shock and anaphylaxis.

Septic shock – infective agents, most commonly bacteria, trigger the release of pro-inflammatory cytokines and nitric oxide. The consequences of this include vasodilation and leakage of plasma from blood vessels into surrounding tissues. The signs of shock include warm peripheries, tachycardia, hypotension, manifestations of poor organ perfusion and signs of the infective source.

Neurogenic shock – this can occur following spinal cord injury. Loss of sympathetic innervation results in unopposed parasympathetic effects on blood vessels, resulting in peripheral vasodilation and reduced venous return. Treatment follows generic ABC principles, together with spinal stabilization. Vasopressors may be needed initially to maintain adequate increasing venous return and blood pressure.

Anaphylactic shock – this is similar to septic shock, but caused by mast cell degranulation releasing substances including histamine which increase capillary permeability and cause vasodilation. This results in relative hypovolaemia and peripheral oedema. As well as supportive measures, specific treatment includes removal of the allergenic source and administration of adrenaline, chlorpheniramine and hydrocortisone.

Haemodynamic monitoring

Haemodynamic monitoring aims to optimize cardiac output and tissue oxygen delivery. Patients who receive large amounts of fluid without a specific haemodynamic target are more inclined to develop pulmonary oedema, pneumonia and respiratory compromise.

The **pulmonary artery flow directed catheter (PAFC)** (Figure 2) generally uses the thermodilution technique to monitor cardiac output. A specific volume of cold fluid is injected into the central venous port of the catheter and the resulting temperature change is detected by a thermistor at the distal end of the catheter, in the pulmonary artery. The variation in temperature over time is plotted as a curve, with the area under the

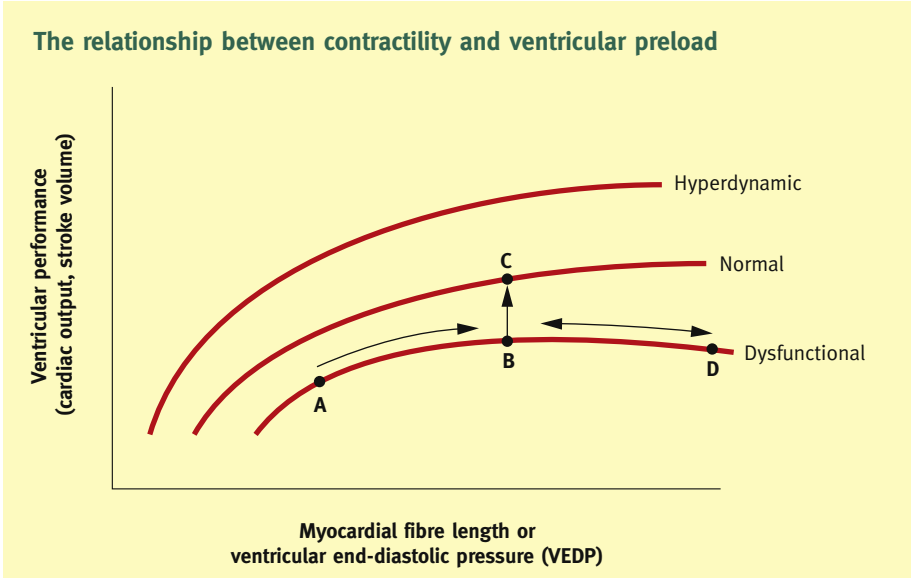

Figure 1