Part VIII: Shock

Chapter 58: Hypovolaemic Shock

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Shock is usually defined as a severe pathophysiological syndrome associated with inadequate or disordered tissue perfusion and abnormal cellular metabolism. The term actually encompasses a group of cardiovascular disorders with distinctive aetiologies and pathophysiological patterns. The most important subgroups are hypovolaemic, septic, anaphylactic and cardiogenic shock, but other types such as traumatic and toxic shock are also recognized.

Aetiology

Hypovolaemic shock is due to inadequate left ventricular preload, which usually requires a relative or absolute reduction in the circulating blood volume by 15-25%, leading to a major reduction in cardiac output. Haemorrhage is the commonest cause, but loss of plasma or protein-free extracellular fluid can also result in hypovolaemic shock. Commonly associated clinical conditions include trauma, burns, peritonitis, pancreatitis, severe vomiting, diarrhoea, fistulae and excessive diuresis.

Pathophysiology

Most of the cellular changes and compensatory mechanisms seen are common to all forms of shock. Ischaemia and impaired tissue perfusion lead to generalized cellular damage, although organs may vary considerably in their ability to withstand ischaemia. Anaerobic cellular metabolism leads to depletion of adenosine triphosphate and failure of the cell membrane sodium potassium pump, resulting in cell swelling due to sodium and water influx. Anaerobic metabolism also produces a progressively worsening metabolic (lactic) acidosis, and blood lactate levels may be used as a guide to the severity, prognosis and effectiveness of therapy in shock. Mitochondrial calcium loss further impairs the efficiency of oxidation and phosphorylation, and may interfere with other organ-specific functions such as myocardial contractility. Eventually, a large number of cytotoxic, vasodilator, vasoactive and other substances are released into the circulation, resulting in progressive vasodilation, myocardial depression, increased capillary permeability and, eventually, intravascular coagulation. These substances include histamine, kinins, lysosomal enzymes, various prostaglandings, serotonin and tumour necrosis factor (cachectin).

Compensatory mechanisms include autoregulation of tissue blood flow and increased sympathetic discharge. This results in arteriolar as well as venous vasoconstriction, and an increase in heart rate and myocardial contractility, thus tending to improve cardiac output, venous return, and blood pressure. Blood is diverted away from less important areas to vital organs, which initially include the brain, heart, kidneys, liver and respiratory muscles. Because of the alteration in Starling forces surrounding capillaries, interstitial fluid is mobilized into the vascular tree. However, as shock progresses, the vasoconstrictive effects of the sympathetic nervous system is overwhelmed by the accumulation of local tissue metabolites,

and capillary permeability increases, leading to extravasation of plasma into the interstitial space. These late changes compound the effects of intravascular hypovolaemia.

Clinical Features

Cardiovascular System

The terms "hypotension" and "shock" should not be regarded as interchangeable, as either can occur without the other being present. Classical features of hypovolaemic shock include, in addition to hypotension, tachycardia, pallor, sweating, cyanosis, hyperventilation, confusion and oliguria. As the blood pressure falls, the pulse pressure narrows and measurement by non-invasive methods mays become increasingly difficult and inaccurate.

Tachycardia usually precedes the development of hypotension, but may not be present if the patient has pre-existing heart disease or has been taking beta-adrenergic blocking drugs. Myocardial contractility may be impaired due to ischaemia, infarction, pre-existing heart disease, or possibly the presence of endotoxin or a myocardial depressant factor.

Respiratory System

Hyperventilation occurs early in shock due to peripheral chemoreceptor stimulation and later, metabolic acidosis. A reduced pulmonary blood flow results in an increased physiological dead space, which is however, more than compensated by the increase in respiratory rate and minute volume. PaO_2 is often well-maintained, but advanced shock may result in respiratory failure due to progressive atelectasis, the onset of the adult respiratory distress syndrome (ARDS), respiratory muscle fatigue due to inadequate respiratory muscle perfusion or more rarely, respiratory centre depression due to inadequate cerebral perfusion. Pre-existing lung disease, chest trauma and cardiac failure may all contribute.

Kidneys

In early shock, the glomerular filtration rate (GFR) is well preserved by autoregulation, but oliguria nevertheless occurs, due to antidiuretic hormone (ADH) and aldosterone secretion. As GFR falls, oliguria persists or worsens. These changes may be modified by renal trauma, pre-existing renal disease or infection, the presence of crush injuries, or the use of nephrotoxic drugs such as aminoglycosides. If shock is severe or prolonged, acute renal failure (ARF) may result. This is initiated by a temporary disorder of glomerular blood flow regulation, and mechanisms such as acute tubular necrosis (ATN) may be of less significance than was once believed.

Liver

Both hepatic arterial and portal venous blood flow are reduced in shock. The commonest clinical consequence is the development of a benign and reversible conjugated hyperbilirubinaemia following a shock episode. In addition, the liver's reticuloendothelial function is impaired following shock, though the clinical significance of this uncertain.

Gastrointestinal Tract

Shock produces profound mucosal vasoconstriction throughout the gastrointestinal tract. The most clinically important consequence of this is an increased susceptibility of the gastric mucosa to acid damage, and subclinical gastric bleeding is probably much more common than the well-recognized association of stress ulceration (gastric erosions) with shock would suggest.

Intermediary Metabolism

Hyperglycaemia is mainluy due to insulin resistance to which catecholamines, glucagon and glucocorticoid secretion all contribute. Catecholamines also inhibit insulin release. Catabolism of both fat and carbohydrate is inhibited in shock, and there is increasing reliance on skeletal muscle amino acids, especially branched chain amino acids, as a fuel source. Lactic acid production is increased due to both mitochondrial dysfunction and hypoxia.

Management

It is important to remember that shock is a dynamic disorder in which haemodynamic and metabolic variables are continually changing. An urgent and well organized approach is important, and frequent or even continuous reassessment will be required. As in other areas of Intensive Care, the diagnostic and therapeutic processes must proceed in parallel rathen than in series.

1. Ventilation and Oxygenation

As with other types of resuscitation, airway and breathing should receive the most immediate attention. It is well established that the combination of shock and respiratory failure has an extremely high mortality. Hence, all shocked patients should receive high flow supplemental oxygen by face-mask, and intubation and mechanical ventilation should be instituted early if there is evidence of respiratory inadequacy. The presence of pneumothorax should be specifically sought and treated in trauma cases.

2. Circulatory Resuscitation

The use of one or more large bore peripheral intravenous cannulae is usually more appropriate than the urgent placement of a central venous catheter, unless peripheral access is unavailable. Obvious external haemorrhage should be controlled by local pressure, but procedures such as urgent thoracotomy with aortic cross-clamping are probably of little value in the vast majority of cases. The use of a PASG suit (pneumatic anti-shock garment) remains controversial, but may help tamponade intra-abdominal bleeding in some circumstances. When the response to resuscitation suggests ongoing intra-abdominal or intra-thoracic bleeding, complex investigations should be kept to a minimum, and every effort should be made to organize definitive surgical management as soon as possible.

3. Choice of Intravenous Fluids

As a general rule, the fluid chosen should most closely match the fluid lost. There has been controversy over the relative merits of crystaloids and colloids as plasma expanders. Supporters of colloids contend that resuscitation with colloids is more rapid and with less adverse effects on the lungs. On the other hand, crystalloid users consider that crystalloids are more appropriate, as they equilibrate between intravascular and interstitial fluid space, overcoming the main problem in shock, ie, shrinkage of the entire extracellular compartment. Crystalloids are also cheaper although 2-4 times the volume may be required to achieve a comparable degree of resuscitation. Colloids present a very small risk of allergic reactions. There is however, no clear evidence to favour either fluid type, especially as regards adverse influence on pulmonary function. A mixture of colloids (initially for rapid improvement) and crystalloids is commonly used in practice.

There is new interest in use of hypertonic saline solutions, ie, 3% for plasma expansion and resuscitation. The mechanism of plasma volume expansion is thought to be due to an increased osmolality pulling intracellular fluid into the extracellular spaces. Smaller volumes are required to achieve the same physiological endpoints, and improvement may be more sustained if dextran is added. Hypertonic saline solutions may play an important role but further clinical studies are required.

Blood should be transfused where significant blood loss has occurred, with the aim of maximizing tissue oxygen delivery by maintaining a haematocrit of at least 0.3 (equivalent to a haemoglobin concentration of about 10 g/dL). The use of oxygen-carrying blood substitutes such as perfluorocarbons and stroma-free haemoglobin is not established.

4. Monitoring

Heart rate, blood pressure, respiratory rate, urine output, mental state and temperature should all be measured and recorded with a frequency appropriate to the clinical state of the patient. A central venous catheter may be useful to assess the right ventricular preload, particularly if trends rather than individual readings are observed. However, the central venous pressure (CVP) may be difficult to interpret in patients with lung disease or selective right or left ventricular dysfunction. In some of these cases, a pulmonary artery catheter may be helpful, but in uncomplicated hypovolaemia, this should only uncommonly be required. Neither the CVP nor the pulmonary capillary wedge pressure (PCWP) provide a meaningful measure of the circulating blood volume.

5. Acid-Base and Electrolyte Therapy

Hypokalaemia, hyponatraemia and metabolic acidosis are characteristic of most shock states, and should be managed in the usual ways. Hyponatraemia and acidosis will almost always respond to volume repletion using appropriate fluids. Bicarbonate administration carries a considerable concurrent sodium load, and may worsen intracellular acidosis, tissue oxygen delivery, and hypokalaemia. Hence, only severe and persistent acidosis should be treated in this way.

6. Inotropic Drugs

Failure of the patient to respond to fluid replacement should prompt a careful search for some other problem, ie, tension pneumothorax, pericardial tamponade, or continuing bleeding. In a few cases, the use of an inotrope may be indicated, and the choice is best determined by haemodynamic measurements. However, differences among these agents are probably of minor clinical importance in this setting and adrenaline (1-10 microg/min), dopamine (1-20 microg/kg/min) and dobutamine (1-20 microg/kg/min) are all commonly used.

7. Diuretics

Frusemide (10-40 mg) or mannitol (10-20 g) are often given when oliguria persists despite apparently adequate fluid resuscitation. However, there is no clear evidence that this improves renal perfusion or can prevent or ameliorate the development of acute renal failure, and care must be taken to ensure that a brisk diuresis does not worsen hypovolaemia or hypokalaemia. There is some evidence to suggest that infusion of dopamine at low dosage (2-5 microg/kg/min) may be helpful.

8. Other Treatment

The use of corticosteroids in shock has been studied extensively for about 30 years with no firm conclusion. This in itself suggests they are unlikely to be of major benefit, despite theoretical and laboratory-based support for their use. They are almost certainly contraindicated if concurrent sepsis is present.

Naloxone has been used in shock, and anecdotal reports suggest it may temporarily reverse hypotension, possibly by antagonizing central delta receptors. Currently there is no good evidence that outcome from shock can be significantly improved by the use of naloxone.

A number of other agents have potential value in the therapy of shock. These include glucagon, fructose diphosphate, amrinone, thyrotropin-releasing hormone (TRH), tri-iodothyronine (T3), prostaglandin E1 and prostacyclin. All require considerably more study before they can be recommended for clinical use.