Monitoring critically ill patients in accident and emergency

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Nurses play a vital role in A&E, monitoring patients’ physiological responses to stress and disease, and identifying any deterioration in their condition so that appropriate treatment can be instigated. This article describes the monitoring techniques used in A&E to assess critically ill patients.

Physiological monitoring and identification of the deteriorating patient are an essential part of the role of the A&E nurse. There are times when seriously ill patients who have been admitted to an A&E department are not recognised as such because of the staff’s busy, unpredictable workloads. The condition of these patients can then potentially become critical.

The physiological response of a critically ill patient to stress or to a disease process will largely determine that person’s outcome (Advanced Trauma Life Support (ATLS), 1997). To an extent this will depend on the degree of shock and injury sustained; this tends to be minimal for minor surgery or injury, and extensive following major accidental or surgical trauma. The physiological reserve of each individual is also an important factor.

Signals that are initiated in injured or ischaemic tissues communicate the extent of the injury systemically. These stress responses are necessary for the process of recovery. However, when trauma is severe, the resultant physiological responses are extensive and sustained, and may be detrimental and contribute to the patient’s becoming critically ill and even dying.

The stress response is initiated not only by tissue injury but also by:
- Acute blood loss;
- Shock;
- Hypoxia;
- Acidosis;
- Hypothermia;
- Altered microcirculatory blood flow;
- Altered coagulation;
- Altered immune function;
- Pain.

Haemorrhage causes the stimulation of baroreceptors in the blood vessels, which activate the central nervous system (Marieb, 1998). The response tends to be proportionate to the degree of shock. Both the degree and duration of blood volume deficit are therefore important determinants of the degree of physiological response to injury. Furthermore, since haemorrhage and hypovolaemia reduce cardiac output, tissue ischaemia may result. This is also an important activator of physiological responses to injury, not only because it may potentiate activation of the centrally mediated stress responses, but also because it leads to initiation of local responses, mediator release and cell activation.

Other initiators of the stress response are hypoxaemia, acidosis and hypercapnia, all of which act at both local and central levels.

Patient assessment

It is important to monitor a patient’s physiological responses in A&E. This not only allows assessment of the patient’s physiological reserve, but will also give a baseline against which the effectiveness of any applied treatment can be judged. Clearly, there are many physiological variables that can be assessed, and these range in complexity as well as degree of invasiveness. It is possible, however, to simplify the monitoring process for the A&E nurse.

The well-being of a patient depends on a normal supply of oxygen and nutrients reaching the tissues, particularly the vital organs such as the brain, heart and lungs. Monitoring of organ function is therefore essential, and should include respiratory function and consciousness level. The latter is measured using the Glasgow coma scale, which is a universal, but not always accurate, measurement of a patient’s level of consciousness. The parameters for assessment are shown in (Box 1).

Supply of oxygen to tissues and organs is crucial and is dependent on three important factors:
- Cardiac output (stroke volume x heart rate);
- Haemoglobin;
- Oxygen saturation (or PaO₂, which is dependent on adequate ventilatory function) (Kumar and Clark, 1999).

Monitoring helps in the early diagnosis of change in a physiological parameter and provides guidelines towards the institution of appropriate therapy. Having a basic knowledge of the principles of monitoring equipment and being able to interpret data correctly is therefore important. No amount of monitoring, however, can replace the close observation of clinical signs by the nurse in A&E.

Monitoring is not the same as treatment, nor is it a substitute for treatment. This cannot be emphasised enough. Instituting even the most invasive of monitoring techniques cannot alone alter a patient’s outcome without modification of treatment.
Physiological responses to trauma

Following major trauma, patients exhibit characteristic behaviours. These include:

- Immobility – they are fearful of moving or interacting;
- Withdrawal – they may cease to be aware of their environment and become incommunicative;
- Antagonism – they may resist interaction and are hostile to those around them.

Altered cerebral blood flow may also be a reason for altered mental state.

The simple vital signs are not normal following trauma; for example, patients are typically febrile and hypotensive, with tachycardia and tachypnoea. Fever is common in the hours and days following resuscitation from moderate to severe trauma. It may be caused by tissue inflammation and cytokine release. Following fluid replacement after trauma, a patient’s blood pressure may be low, normal, or high (ATLS, 1997). Blood pressure correlates poorly with either blood volume or flow.

Sympathetic stimulation and high levels of circulating catecholamines cause tachycardia. Following severe trauma, tachycardia typically persists even after hypovolaemia has been corrected and pain controlled. In severe trauma or shock, however, tachycardia may not occur, and the heart rate may be normal or decreased; this may seriously impair the compensatory hyper-dynamic physiological response that is necessary for recovery (ATLS, 1997).

Increased minute ventilation, due to both tachypnoea and an increase in tidal volume, is an expected response following a major operation or injury. It is driven by increased catecholamine levels, reduced haemoglobin levels and sympathetic tone, as well as by increased oxygen consumption and carbon dioxide production following trauma (Kumar and Clark, 1999).

Urine output is often diminished early after trauma or surgery because of hypovolaemia, a reduction in renal blood flow, and a hormonal milieu that leads to sodium and water reabsorption. However, resuscitation with large volumes of crystalloid solutions as well as commonly used osmotically active agents such as radiological contrast media and mannitol, increase urine output. Thus, urine output may be reduced, normal, or increased following trauma, and may not accurately reflect the intravascular volume.

That resuscitation has been adequate is best assessed by observing an improvement in the patient’s physiological parameters. These are pulse rate, blood pressure, ventilatory rate, arterial blood gases, temperature and urine output.

Monitoring techniques

Measuring tissue oxygenation

When the demand for oxygen is not being satisfied, cell injury results. Low blood flow states, microcirculatory failure and endotoxaemia are all important factors in the pathogenesis of cell injury, which may lead to organ failure in patients who are critically ill. These three factors are all interlinked.

Virtually all acute responses of cells to injury involve alterations to the cell membrane, and ischaemia and hypoxia cause cessation of normal mitochondrial activity, thus interfering with normal adenosine triphosphate (ATP) synthesis (Roberts, 1991).

Temperature

Peripheral temperature can reflect tissue perfusion and is affected by vasoconstriction and low cardiac output. However, caution must be taken in patients with sepsis, as they are often warm to touch, vasodilated and have poor peripheral perfusion. In A&E, patients’ core temperature can be monitored at the tympanic membrane, axilla or rectum. There is an increased gradient between core and peripheral temperature in shock states.

Checking temperature gradients is a very useful means of non-specific monitoring, as it is for arterial blood pressure. Thermistors are commonly used for monitoring a patient’s core temperature.

Electrocardiography

An electrocardiogram (ECG) is a useful adjunct to diagnosing ischaemia, arrhythmias, electrolyte imbalance and drug toxicity. It is important to ensure that the machine’s 12 leads are positioned correctly on the patient. Modification of these leads can detect both arrhythmias and ischaemia. Many monitoring systems now automatically recognise a variety of arrhythmias (Harrison and Daly, 2001).

References


Pulse oximetry
Pulse oximetry is a valuable monitoring tool. It measures oxygen saturation colour metrically, although it does not measure ventilation or the partial pressure of oxygen. Advances in this technology mean that the light absorbed by the pulsatile component of haemoglobin in the finger or ear lobe can be measured (SpO₂), so allowing estimates of arterial saturation (SaO₂) to be made. In healthy patients, the pulsatile component of the signal is around only 2 per cent of the total absorption, making the signal to noise ratio of the measurement poor. At low haemoglobin levels, the measurement becomes unreliable. In critically ill patients, peripheral perfusion is often reduced, which further degrades the signal to a point at which it is not possible to make reliable measurements of saturation.

It is important to note that a saturation reading of around 97 per cent from the pulse oximeter is only suggestive that the PaO₂ is probably in excess of 9kPa (Hinds, 1994). It is important to note that some beds such as the brain and kidney may become impaired in critical illness, autoregulatory mechanisms in vascular beds such as the brain and kidney may become impaired and perfusion to these organs will be pressure-dependent (Hinds, 1994). It is important to note that some patients are hypotensive intrinsically, or as a result of medication, and still maintain adequate perfusion of the state of the circulation.

Haematocrit and haemoglobin concentration
Low haematocrit tends to be associated with improved peripheral perfusion because of reduced viscosity, although how this relates to perfusion in a patient is largely unknown. Oxygen delivery to the tissues may be compromised owing to reduced oxygen capacity.

Serial decline in haematocrit indicates continued bleeding, but haemodilution with crystalloids can also result in a fall in the haematocrit. The percentage of red cells present in a blood sample gives an indication of adequacy of blood replacement following trauma and surgery. In the critically ill patient an ideal haematocrit is probably 35 per cent, with a haemoglobin concentration of 12–14g/dl (Hinds, 1994).

Arterial blood pressure
Arterial blood pressure is proportional to cardiac output when peripheral resistance is constant, and is affected by changes in the blood volume status of the patient, vasoconstriction and cardiac output. Blood pressure is maintained by physiological compensation in the face of changes in blood volume and cardiac output. Indeed blood pressure may be normal despite grossly impaired cardiac function and, therefore, is only a crude indicator of the state of the circulation.

However, in most trauma patients, if blood pressure is low, tissue perfusion will be inadequate. Furthermore, in critical illness, autoregulatory mechanisms in vascular beds such as the brain and kidney may become impaired and perfusion to these organs will be pressure-dependent (Hinds, 1994). It is important to note that some patients are hypotensive intrinsically, or as a result of medication, and still maintain adequate perfusion.

Blood flow to tissues is crucially dependent on the mean blood pressure. This is not simply an average of the systolic and diastolic pressure but is weighted more towards the diastolic pressure, so that it is one-third the sum of the systolic pressure plus twice the diastolic pressure. Knowledge of the mean arterial pressure is also required to calculate systemic vascular resistance. This is often given automatically with electric blood pressure and cardiac output monitors.

Indirect methods of measuring blood pressure include palpation, auscultation and oscillotonomometry. Direct arterial pressures can be recorded by inserting a cannula in the radial and femoral artery and connecting it to a zeroed and calibrated transducer. The presence of air bubbles, leaks in the system or blocked cannulae can produce an inaccurate trace.

Central venous pressure
Central venous pressure (CVP) is a useful tool for assessing a patient’s blood volume status. CVP is the most common parameter used to guide fluid therapy in a patient with hypovolaemia following trauma, shock, burns, or sepsis (Kumar and Clark, 1999). CVP catheters can be inserted at different sites but in each case the tip of the catheter should be intrathoracic. Sites used for the insertion of the catheter include the external jugular vein, the internal jugular vein and the subclavian and femoral veins. Most commonly, the Seldinger technique is used. This involves puncturing the vein with a needle, then inserting a J-wire through the needle before removing it and passing the catheter over the wire after prior dilatation of the site, if necessary.

It is preferable to use the subclavian site, since it is associated with fewest infective complications, although there is a greater incidence of haemothorax using this method. The value of the CVP can be obtained using a saline-filled manometer, zeroed to the mid-axillary line as the reference point, or by using a pressure transducer. A normal CVP ranges between 4 and 10cmH₂O (Kumar and Clark, 1999). However, in some patients there can be a discrepancy between CVP and left-side heart-filling pressures. False high CVP can be seen in patients with high right-side heart pressures (Box 2).

These patients can have a high CVP measurement, yet be hypovolaemic. For this reason, comparing changes in a CVP measurement with a baseline measurement is more beneficial than noting the initial recording.

Respiratory system monitoring
Monitoring the respiratory system is a very important activity, but for patients who have been admitted to A&E

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**BOX 2. CONDITIONS ASSOCIATED WITH FALSE HIGH CENTRAL VENOUS PRESSURE**

- Tricuspid valve regurgitation
- Congestive cardiac failure
- Pulmonary valve stenosis
- Pulmonary artery hypertension
- Severe pulmonary embolism
- Chronic obstructive pulmonary disease (COPD)
Respiratory rate; Arterial blood gases; Peak flow; Manually recording ventilatory rate. Increasing respiratory rate, shallow breathing patterns, paradoxical respiration, use of accessory muscles, tachycardia and excessive sweating all indicate inadequate ventilation and impending respiratory failure.

Gas exchange
Pulse oximetry is a simple and non-invasive method of monitoring oxygen saturation (SpO$_2$) and is used to assess adequacy of gas exchange. Screening for arterial blood gases and pH are useful tests of pulmonary function. Often, the first laboratory signs of impending lung problems are seen as changes in PaO$_2$, PaCO$_2$, and pH. A PaO$_2$ value of less than 8.0kPa while breathing room air suggests type 1 respiratory failure. Coupled with a PaCO$_2$ greater than 6.0kPa, type 2 respiratory failure is suggested. Critically ill patients are usually receiving supplemental oxygen, and their PaO$_2$ should always be interpreted in relation to the inspired oxygen tension (FiO$_2$). Patients with chronic pulmonary disease and type 2 respiratory failure can tolerate abnormal blood gas values owing to compensatory mechanisms, but they do not generally tolerate more than 28 per cent FiO$_2$.

Patients with normal lungs should be given supplemental oxygen if they are at all hypoxic, followed by ventilatory support if the respiratory insufficiency does not improve. Intermittent analysis of patients’ blood gases gives useful information during mechanical ventilation and weaning.

On-line intravascular monitoring of PaO$_2$, PaCO$_2$ and pH is now available, but its usefulness over and above intermittent analysis has yet to be established.

Renal system
Oliguria is usually the first indication of renal impairment in a patient and should prompt immediate attempts to optimise cardiovascular function, particularly by expanding the circulating volume and restoring blood pressure to normal values.

Hourly urine output can be a very useful guide to the adequacy of cardiac output, splanchnic perfusion and renal function. Normal renal function produces 0.5ml/kg of urine per hour in adults. Measurement of the specific gravity and osmolality of the urine is used to differentiate between pre-renal and renal failure.

Plasma and urine electrolytes, urea and creatinine trends of blood urea, and creatinine and serum electrolytes are useful for evaluating the progress of renal function. However, urea can rise in the absence of renal dysfunction in conditions such as gastrointestinal bleeding, high protein intake and increased catabolism.

Acute and chronic renal failure results in rising urea and creatinine levels. The concentrating ability of the kidney can be estimated by comparing the blood and urine sodium, potassium and urea. A urine/plasma osmolality ratio of less than 1.2, urea ratio of less than 10 and a urinary sodium of more than 40mmol/l indicate acute renal failure.

The use of mannitol and loop diuretics should be taken into account as they can cause electrolyte abnormalities.

Hepatic system
Damage to the liver may not obviously affect its activity because of a considerable functional reserve. Consequently, tests of liver function alone are insensitive indicators of the degree of liver disease. Indicators of cell damage are frequently used instead, for example measurement of hepatic enzymes. The liver synthesises albumin, clotting factors, anti-thrombin III and protein C, all of which can be used to assess liver function. Albumin, because of its long half-life, is not a sensitive measure of acute liver dysfunction.

Defects in clotting are reflected in the standard tests, and prothrombin time is a useful guide for the monitoring of liver function. Factor VII has a half-life of four to eight hours and its measurement can be used to assess the severity of coagulopathy, even in cases where fresh frozen plasma has been given. A raised serum bilirubin concentration is frequently, though not invariably, seen in liver disease. Indeed, in acute hepatic failure patients are rarely jaundiced and have normal or only slightly abnormal serum bilirubin levels. Greatly increased serum transaminase activities are characteristic of hepato-cellular damage, while raised alkaline phosphatase activity is seen in biliary obstruction.

Haematological monitoring
Haemostatic failure and acquired coagulopathies, such as disseminated intravascular coagulation, are regularly seen in patients in A&E. The main causes of clotting factor deficiencies are liver disease, vitamin K deficiency, anti-coagulant drugs, disseminated intravascular coagulopathy and massive blood transfusion. Clotting function is usually assessed by measuring prothrombin time, activated partial thromboplastin time, fibrinogen concentration and either fibrin degradation products (FDPs) or D-dimer assessment.

Conclusion
It is essential that A&E nurses realise the importance of monitoring trauma patients and have an understanding of physiological parameters, so that they are able to offer high quality care and achieve positive patient outcomes. Different patients respond differently to trauma and have different physiological reserves, and it is important that A&E nurses are aware of this when assessing physiological status. Many of the treatment options used require some form of physiological monitoring in order to gauge their effectiveness. An understanding of the normal and abnormal variants in parameters and vital signs will enable nurses to recognise that the condition of the patient is deteriorating, which empowers them to intervene promptly and appropriately.