

Monitoring

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Cardiovascular - Cardiovascular monitoring at minimum should include continuous heart rate (electrocardiogram, oxygen saturation and pulse waveform and non-invasive blood pressure). Patients whose state of shock is not rapidly corrected with a small amount of fluid should have CVP monitoring and continuous blood pressure monitoring through an arterial line.

Central venous pressure There is no 'normal' CVP for a shocked patient, and reliance cannot be placed on an individual pressure measurement to assess volume status. Some patients may require a CVP of 5 cmHg, whereas some may require a CVP of 15 cmHg or higher. Further, ventricular compliance can change from minute to minute in the shocked state, and CVP is a poor reflection of end-diastolic volume (preload). CVP measurements should be assessed dynamically as the response to a fluid challenge. A fluid bolus (250–500 mL) is infused rapidly over 5–10 minutes. The normal CVP response is a rise of 2–5 cmHg, which gradually drifts back to the original level over 10–20 minutes. Patients with no change in their CVP are empty and require further fluid resuscitation. Patients with a large, sustained rise in CVP have high preload and an element of cardiac insufficiency or volume overload.

Minimum Additional modalities ECG Central venous pressure Pulse oximetry Invasive blood pressure Blood pressure Cardiac output Urine output Base deficit and serum lactate

Cardiac output monitoring allows assessment of not only the cardiac output but also the systemic vascular resistance and, depending on the technique used, end-diastolic volume (preload) and blood volume. Use of invasive cardiac monitoring with pulmonary artery catheters is becoming less frequent as new non-invasive monitoring techniques, such as Doppler ultrasound, pulse waveform analysis and indicator dilution methods, provide similar information without many of the drawbacks of more invasive techniques. Measurement of cardiac output, systemic vascular resistance and preload can help distinguish the types of shock present (hypovolaemia, distributive, cardiogenic), especially when they coexist. The information provided guides fluid and vasopressor therapy by providing real-time monitoring of the cardiovascular response. Measurement of cardiac output is desirable in patients who do not respond as expected to first-line therapy or who have evidence of cardiogenic shock or myocardial dysfunction. Early consideration should be given to instituting cardiac output monitoring for patients who require vasopressor or inotropic support. Systemic and organ perfusion Ultimately, the goal of treatment is to restore cellular and organ perfusion. Ideally, therefore, monitoring of organ perfusion should guide the management of shock. The best measure of organ perfusion and the best monitor of the adequacy of shock therapy

remains the urine output. However, this is an hourly measure and does not give a minute-to-minute view of the shocked state. The level of consciousness is an important marker of cerebral perfusion, but brain perfusion is maintained until the very late stages of shock and hence is a poor marker of adequacy of resuscitation (Table 2.4). Currently , the only clinical indicators of perfusion of the gastrointestinal tract and muscular beds are the global Christian Johann Doppler , 1803–1853, Professor of Experimental Physics, Vienna, Austria, en mixed venous oxygen saturation. Base deficit and lactate Lactic acid is generated by cells undergoing anaerobic respi - - ration. The degree of lactic acidosis, as measured by serum lactate level and/or the base deficit, is sensitive for both diagnosis of shock and monitoring the response to therapy . Patients with a base deficit of more than 6 /uni00A0 mmol/L have a much higher morbidity and mortality than those with no metabolic acidosis. Furthermore, the length of time in shock - with an increased base deficit is important, even if all other vital signs have returned to normal (see occult hypoperfusion below under End points of resuscitation). These parameters are measured from arterial blood gas analyses, and therefore the frequency of measurements is limited and they do not pr ovide minute-to-minute data on systemic perfusion or the response to therapy . Nevertheless, the base deficit and/or lactate should be measured routinely in these patients until they have returned to normal levels. - - Mixed venous oxygen saturation The percentage saturation of oxygen returning to the heart from the body is a measure of the oxygen delivery and extraction by the tissues. Accurate measurement is via analysis of blood drawn from a long central line placed in the right atrium. Estimations can be made from blood drawn from lines in the superior vena cava, but these values will be slightly higher than those of a mixed venous sample (as there is rela - tively more oxygen extraction from the lower half of the body). Normal mixed venous oxygen saturation levels are 50–70%. Levels below 50% indicate inadequate oxygen delivery and increased oxygen extraction by the cells. This is consistent with hypovolaemic or cardiogenic shock. High mixed venous saturations (>70%) are seen in sepsis and some other forms of distributive shock. In sepsis, there is disordered utilisation of oxygen at the cellular level and unciated the Doppler principle in 1842.

TABLE 2.4 Monitors for organ/systemic perfusion. Clinical Systemic perfusion Organ perfusion

Muscle - Gut - Kidney	Urine output	Brain Conscious level	Investigational Base de /f_i cit Lactate
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Therefore, less oxygen is presented to the cells, and those cells cannot utilise what little oxygen is presented. Thus, venous blood has a higher oxygen concentration than normal. Patients who are septic should therefore have mixed venous oxygen saturations above 70%; below this level, they are not only in septic shock but also in hypovolaemic or cardiogenic shock. Although the S O level is in the 'normal' range, it is low v 2 for the septic state, and inadequate oxygen is being supplied to cells that cannot utilise oxygen appropriately . This must be cor rected rapidly . Hypovolaemia should be corrected with fluid therapy , and low cardiac output due to myocardial depression or failure should be treated with inotropes (dobutamine) to achieve a mixed venous saturation greater than 70% (normal for the septic state). New methods for monitoring regional tissue perfusion and oxygenation are becoming available, the most promising of which h are muscle tissue oxygen probes, near-infrared spectros copy and sublingual capnometry . While these techniques pro vide information regarding perfusion of specific tissue beds, it is as yet unclear whether there are significant advantages over existing measurements of global hypoperfusion (base deficit, lactate).

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