# Assessment of haemodynamic disturbance

Haemodynamic disturbance is a common occurrence in critically ill patients and is most exaggerated in patients with shock. Shock is defined as a life-threatening, generalized maldistribution of blood flow resulting in failure to deliver and/or use adequate amounts of oxygen, leading to tissue hypoxia (Holley et al, 2012a). The inadequate oxygen delivery typically results from poor tissue perfusion, which aggravates the clinical situation in a critically unwell patient with high metabolic demand. *Table 1* highlights the most common types of shock.

Irrespective of the underlying cause of shock, the haemodynamic assessment and initial resuscitation is the same. The aim is to ameliorate multiorgan dysfunction (by improving organ perfusion) while concurrently addressing the underlying cause.

In this two-part series, this first article will outline how to assess haemodynamic status in order to use early resuscitation efforts to resolve haemodynamic instability. The second article uses two common examples of shock to illustrate this structured approach. The articles focus on bedside assessment and initial management for the UK core trainee on the ward, so the use of complex invasive monitoring and intensive care therapies are out of scope of these articles.

## Haemodynamic disturbance: parameters to guide therapy

The aim of haemodynamic assessment is to address the adequacy of end organ perfusion and tissue oxygen delivery. To facilitate early detection and management of shock, one should adopt a timely and systematic approach, using a range of individualized resuscitation targets.

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**Table 1. Common types of shock and the principal haemodynamic disturbance**

| Type | Subtype examples | Haemodynamic disturbance |
|---|---|---|
| Hypovolaemic | Haemorrhage<br>Third space loss, e.g. pancreatitis<br>Fluid loss, e.g. diarrhoea | Loss of intravascular volume<br>Reduced preload, stroke volume and cardiac output<br>Initial increased systemic vascular resistance |
| Cardiogenic | Primary pump failure, e.g. myocardial infarction<br>Valve disease | Reduced cardiac output |
| Obstructive | Examples: pulmonary embolus, tension pneumothorax and cardiac tamponade | Reduced cardiac output |
| Distributive | Septic<br>Anaphylaxis<br>Neurogenic | Decreased systemic vascular resistance with abnormal distribution of blood flow<br>Cardiac output usually preserved |

Outside the intensive care unit setting, macrohaemodynamic assessment is crudely used as a surrogate for microcirculatory perfusion. To determine the haemodynamic status of a critically ill patient, one must assess the volume status, volume responsiveness and whether manipulation of the other components involved in the relationship of tissue perfusion is necessary. These components are cardiac output, heart rate, stroke volume, mean arterial pressure and systemic vascular resistance. In the initial resuscitation phase, only certain components can be easily determined (mainly heart rate, mean arterial pressure and central venous pressure) and some can be manipulated but not readily determined, such as systemic vascular resistance. In the intensive care unit, dynamic tools are used which predict the cardiac output, systemic vascular resistance and stroke volume over time in response to interventions.

In addition to measuring the determinants of perfusion, one should assess adequacy of perfusion, which is not solely dependent on cardiac output. This is where measurements of central venous oxygen saturation and lactate clearance can add critical information regarding the global balance in oxygen delivery and consumption.

A simple schematic arises for resuscitation in shock, emphasizing that therapy should be titrated to clear clinical endpoints and guided by parameters that reflect the adequacy of perfusion (*Figure 1*).

Each of these parameters is now presented individually. As each has its own pitfalls, no one measurement should be used in isolation. Trends are more useful than a single measurement of any modality and a helpful way to use haemodynamic monitoring is to evaluate the response to therapeutic interventions.

## Basic assessment

A thorough clinical examination is the critical initial step in identifying shock. Look for signs of hypoperfusion: skin mottling, cool extremities, poor peripheral pulses and reduced consciousness (*Table 2*).

## Volume assessment and fluid responsiveness: optimizing preload

The next fundamental step is volume resuscitation, but studies have shown that 50% of patients with shock will not respond to a fluid bolus (Marik et al, 2009). As overzealous fluid resuscitation has deleterious consequences, it is of critical importance that fluid status and responsiveness be determined. Assessing fluid responsiveness is essentially trying to establish whether the

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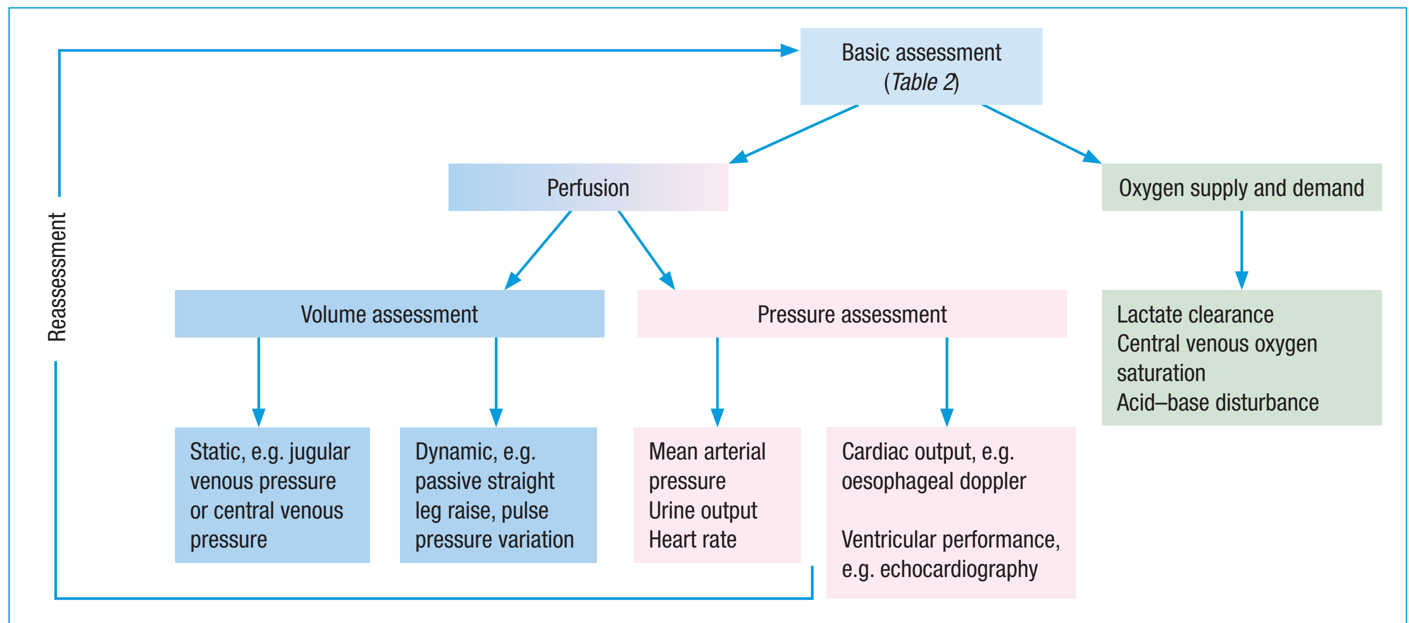
Figure 1. Schematic to guide initial resuscitation in patients. After a basic bedside assessment to detect signs of hypoperfusion, therapy should be targeted towards distinct clinical parameters that determine adequacy of perfusion. These can be divided into those that indicate intravascular volume, pressure and oxygen supply and demand.




failing patient is on the ascending portion of the Frank–Starling curve and therefore should be administered fluid (*Figure 2*).

Initially fluid status is rapidly assessed at the bedside using vital signs, jugular venous pressure and identifying any evidence of fluid overload (e.g. pulmonary oedema). This can give some indication of global fluid distribution and whether fluid challenges can safely be given. However, intravascular volume is more difficult to assess and often needs invasive monitoring, which is outside the scope of this article.

Fluid responsiveness is defined as an increase in stroke volume and cardiac performance with a fluid bolus. Preload represents the load present before ventricular contraction has started, which is provided by venous return. An increase in preload can be used as an indicator of fluid responsiveness, but this is controversial as many factors complicate this relationship. As doctors are limited by what is available on the wards, the authors suggest using a surrogate marker of right ventricular preload in the form of jugular venous pressure (central venous pressure if available). The authors recommend using the changes in jugular venous pressure with fluid boluses as an indicator of fluid responsiveness, although it is important to be aware of its limitations.

Fluid responsiveness can be also be measured by dynamic methods, which are more sensitive and specific, but most of these require invasive monitoring.

**Table 2. Bedside detection of organ dysfunction in patients with suspected shock**

| | |
|---|---|
| Respiratory | Tachypnoea (can be compensatory) |
| | Arterial hypoxaemia ($PaO_2$ [mmHg]/$FiO_2$) <300. Note: desaturation is a late sign |
| Cardiovascular | Tachycardia |
| | Hypotension (systolic blood pressure <90 mmHg, mean arterial pressure <70 mmHg or decrease of systolic blood pressure of >40 mmHg in adults) |
| | Decreased central capillary refill (unreliable if vasodilated) |
| Other | Altered mental state |
| | Substantial oedema |
| | Acute oliguria (<0.5 ml/kg/hr) |
| | Paralytic ileus |
| Tests | Fever or hypothermia (>38°C; <36°C) |
| | Hyperlactaemia (lactate >2 mmol/litre) |
| | Metabolic acidosis (base deficit) |
| | Altered glycaemic control |

*$FiO_2$ = fractional inspired oxygen; $PaO_2$ = arterial partial pressure of oxygen.*

### Static measurement: jugular venous pressure

This should only be used as an indicator of volume status in initial resuscitation. Jugular venous pressure is higher in patients with conditions that lead to raised right atrial pressures, e.g. tricuspid regurgitation and pulmonary hypertension. User variability adds to its limitations. Observing a non-sustained rise in jugular venous pressure suggests that the patient is fluid responsive, whereas a persistently elevated jugular venous pressure may indicate that the patient is volume replete.

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Figure 2. Frank–Starling curve. The Frank–Starling law states that the stroke volume of the heart increases in response to an increase in the volume of blood filling the heart (the end diastolic volume) when all other factors remain constant. After a certain point, increasing the end diastolic volume and thus increasing the 'stretch' of the cardiac muscle fibres does not improve the stroke volume. At this point further fluid resuscitation is unlikely to cause an improvement in cardiac output.

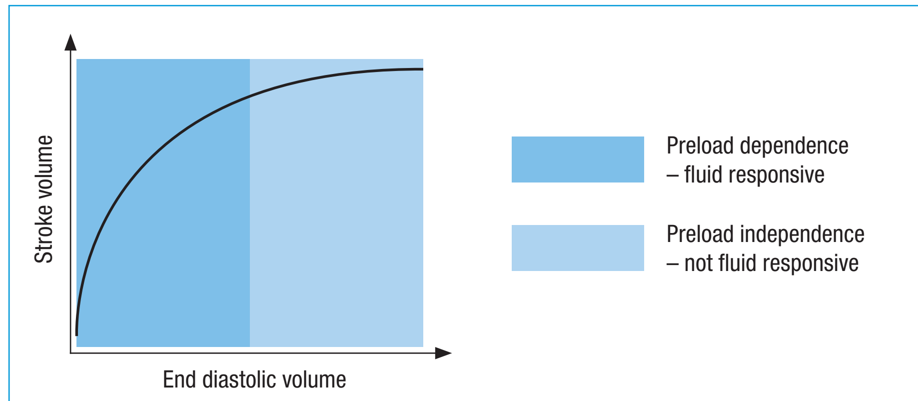



Figure 3. Arterial line waveform, demonstrating a 'swing' over time caused by changes in pulse pressure during inspiration and expiration. This variation in pulse pressure can be exaggerated in hypovolaemic patients.

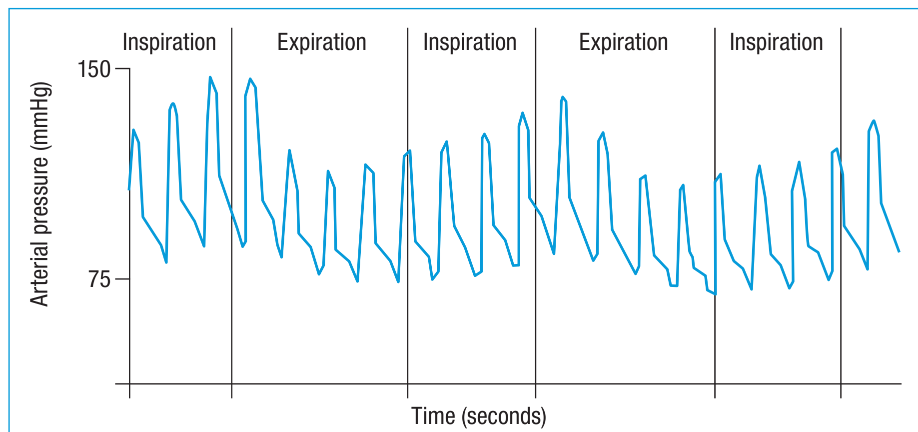
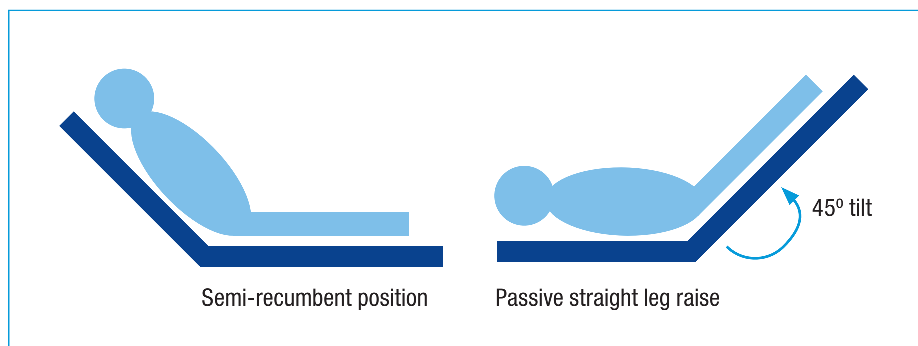



Figure 4. Passive straight leg raise. By tilting a patient 45° from a semi-recumbent position, a hypothetical 'fluid bolus' can be given and parameters such as heart rate and blood pressure can be assessed for fluid responsiveness.




Jugular venous pressure is synonymous with central venous pressure, but the latter is perhaps more sophisticated. Some wards may have access to central venous pressure measurement, which is pressure transduction from the superior vena cava using a thoracic central venous catheter. The normal value is 3–8 mmHg with fluid responsiveness defined as a non-sustained rise in central venous pressure of >2 mmHg with a 250 ml bolus over 10 minutes (Holley et al, 2012a). It is accepted that a very low central venous pressure in the context of a low mean arterial pressure is a good indicator of intravascular volume depletion and fluid boluses may be beneficial.

### Dynamic measurement

Pulse pressure and stroke volume vary with respiration. This pulse pressure variation can be observed on the monitor as an undulation ('swing') in the arterial line waveform (*Figure 3*). Similarly, this can be seen on a pulse oximetry trace. If pulse pressure varies significantly with changes in intrathoracic pressure during respiration, this swing is exaggerated and can suggest relative hypovolaemia (which may be referred to as pulsus paradoxus). It is less useful in spontaneously breathing patients and cannot be used if an arrhythmia is present.

One can perform the passive leg raise test to 45° in a spontaneously breathing patient, which allows for a 'fluid bolus' to be given without the administration of intravenous fluids. By rapidly tilting a patient from a semi-recumbent position to a supine position (*Figure 4*), one can assess vital signs for any changes. This has been shown to predict fluid responsiveness (Preau et al, 2010).

## Perfusion assessment: maintaining perfusion pressure

A concurrent assessment should be made to assess the adequacy of the perfusion pressure. Components that can be measured to assist the assessment are set out below.

### Mean arterial pressure

Mean arterial pressure is (systolic blood pressure + 2x diastolic blood pressure)/3 – a simple measure of end organ perfusion.

Diastolic blood pressure contributes more to mean arterial pressure than systolic blood pressure. Therefore initial changes in pulse pressure are more important than the systolic blood pressure, e.g. septic shock reduces diastolic blood pressure with a drop in mean arterial pressure; hypovolaemic shock increases diastolic blood pressure with an initial relative preservation of mean arterial pressure.

Non-invasive measurements are affected by the cuff size, altered peripheral vascular tone and calcification of arteries. Therefore invasive dynamic monitoring is preferred.

In hypotensive states, autoregulation in regional vascular beds is compromised and flow is more dependent on pressure.

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## TOP TIPS

- Early communication with the senior team is imperative.
- Regularly reassess parameters once any intervention has been administered.
- Consider bedside echocardiogram in assessing fluid status.
- Aim to move persistently unstable patients to a critical care environment.
- Do not use a single parameter in isolation to assess haemodynamic status and fluid responsiveness.

Optimal mean arterial pressure depends on the type of shock and the patient's pre-existing physiology, e.g. higher mean arterial pressure is required for those with premorbid hypertension; lower mean arterial pressure is needed for those with haemorrhagic hypovolaemic shock (may reduce bleeding and allow thrombus formation).

### Heart rate

Tachycardia and bradycardia can compromise cardiac output. Tachycardia can be appropriate to the physiology and degree of illness.

## Global oxygen supply and demand assessment

Once volume and pressure deficits have been addressed, the possible imbalance between global oxygen supply and demand should be assessed. All patients in shock should receive oxygen, keeping saturations ≥94% (caution to avoid oxygen toxicity).

### Central venous oxygen saturation

Take a superior vena cava sample from a thoracic central venous catheter. The normal value is approximately 70%. This shows the percentage of oxygen extracted by the upper body and reflects the balance between global oxygen delivery and consumption. However, interpretation is a challenge as a result of multiple variables. Variables that can be modified are fractional inspired oxygen, cardiac output and haemoglobin concentration.

Central venous oxygen saturation <70% may indicate an oxygen supply or demand mismatch. Central venous oxygen saturation ≥90% could represent poor oxygen extraction as a result of tissue necrosis or shunting and is regarded as a poor prognostic factor (Pope et al, 2010).

### Lactate clearance and base deficit

Inadequate oxygen delivery results in anaerobic respiration and lactate production. Serum lactate is affected by factors other than perfusion, e.g. hepatic clearance.

Lactate clearance can imply adequate resuscitation – failure to clear correlates with increased morbidity and mortality (Holley et al, 2012b). Tissue hypoxia results in metabolic acidosis, so serial blood gases should be obtained. Correct other causes of metabolic acidosis.

## Conclusions

Haemodynamic disturbance is a frequently encountered problem in hospital, so it is essential that junior doctors are armed with a structured approach to tackle it. By understanding the physiology of shock, one can use the parameters discussed to detect it, assess it and swiftly initiate goal directed resuscitation. Despite the evidence supporting the use of certain parameters being contentious, when used together they provide a good framework to guide clinical practice on the general ward. BJHM

*Conflict of interest: none.*

## KEY POINTS

- Haemodynamic disturbance is when a patient's disease process causes deviation from the physiologically 'normal' parameters.
- Shock leads to inadequate oxygen delivery and ultimately causes tissue hypoxia.
- Early detection and resuscitation targeted to individualized parameters is paramount in preventing multiorgan dysfunction.
- A three-staged approach addressing volume, pressure and oxygen mismatch forms the backbone to the assessment and evaluation of the degree of disturbance.
- Obtaining the relevant parameters and striving to correct them guides early goal-directed therapy as well as helping communication with critical care.

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