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# HAEMODYNAMIC DYSFUNCTION AFTER ABDOMINAL AORTIC ANEURYSM REPAIR

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## CASE SCENARIO

A 72-year-old man is admitted directly to the intensive care unit (ICU) following emergency abdominal aortic aneurysm repair. On his arrival in ICU, his systolic blood pressure is 115 mmHg but falls to 85 mmHg over the next 15 min. Intra-operatively he lost an estimated 4000 ml blood during a 4-h operation. His wife informs you that he had a 'heart attack' 3 years ago and can now only walk half a mile before getting chest pain. He takes aspirin and atenolol, and uses a glyceryl trinitrate spray about three times a week. He has no other significant medical history. He had been conscious on arrival at the Accident and Emergency Department but had a tachycardia (120 beats.min$^{-1}$) and hypotension (systolic arterial pressure 90 mmHg).

## DEFINITION AND DESCRIPTION OF THE PROBLEM

Hypotension is defined as a systolic arterial blood pressure of less than 90 mmHg in a previously normotensive patient, or a fall in systolic arterial blood pressure of more than 25% of the patient's usual blood pressure. Hypotension is clinically significant if vital organ perfusion is compromised (e.g. myocardial ischaemia, oliguria and confusion). Patients presenting with a ruptured abdominal aortic aneurysm often have a number of pre-existing problems that add to the high operative mortality associated with haemorrhagic shock and lower torso ischaemia. These include increasing age, cardiovascular and cerebrovascular disease, smoking-related respiratory disease, diabetes mellitus and renal disease. In order to minimise organ dysfunction secondary to hypoperfusion, blood pressure should be

maintained at or close to the patient's normal value. The usual systolic blood pressure may be obtained from old hospital notes or the general practitioner records, but if these sources are unavailable an educated guess is required. Normal systolic blood pressure in patients over 50 years of age varies between 120 and 150 mmHg. However, in patients with ischaemic heart disease and peripheral vascular disease, a relatively non-compliant vascular system will be associated with a higher blood pressure and a target pressure of 150 mmHg should be set.

## PATHOPHYSIOLOGY OF THE PROBLEM

Since

$$\text{mean arterial pressure} = \text{cardiac output} \times \text{systemic vascular resistance}$$

and

$$\text{cardiac output} = \text{stroke volume} \times \text{heart rate},$$

hypotension may result from reduced heart rate, stroke volume or systemic vascular resistance. However, it is sometimes difficult to establish which parameter is the most deranged. The heart rate may be easily obtained from the continuously monitored electrocardiogram (ECG). In a patient with a history of ischaemic heart disease, a rate of 60–80 beats.min$^{-1}$ permits the maintenance of cardiac output while minimising myocardial work. Drugs affecting the cardiovascular system, such as atenolol, may blunt physiological responses and impair the ability to compensate for hypovolaemia.

A low stroke volume may be caused by changes in preload, myocardial contractility or afterload. Starling described the relationship between preload (initial myofibril length) and the force of contraction. The length of the cardiac muscle fibres at the start of contraction is related to the end-diastolic ventricular volume, which is mainly dependent on venous return. Venous return is reduced by hypovolaemia, head-up posture, increased intra-thoracic pressure (e.g. tension pneumothorax, positive pressure ventilation), venodilating drugs (e.g. nitrates) or cardiac tamponade. Central venous pressure (CVP) can be used as an index of right ventricular preload and, with normal cardiac function, will also reflect left ventricular preload. However, right atrial pressures do not reflect left heart function in patients with left ventricular failure, severe bundle branch block, pulmonary hypertension, chronic pulmonary disease, interstitial pulmonary oedema and valvular heart disease; here the pulmonary capillary wedge pressure (PCWP) may be used as an estimate of left ventricular preload.

Myocardial contractility is reduced by hypoxaemia, hypothermia, hypercapnia, ischaemic heart disease, myocardial infarction, acidosis, electrolyte imbalances and negatively inotropic drugs (e.g. inhalational anaesthetic agents and anti-arrhythmic drugs). Afterload is the ventricular wall tension required to eject the stroke volume during systole; increased afterload results in increased myocardial work and oxygen

consumption and a decreased stroke volume. Afterload is increased by increased ventricular volume (according to Laplace's law), decreased elasticity of the large blood vessels (e.g. with age), aortic stenosis and increased systemic vascular resistance. Systemic vascular resistance is determined by the diameter of arterioles and is under both neural and humoral control.

Hypovolaemia or myocardial dysfunction is the most likely cause for the hypotension seen in this patient. He has sustained large, peri-operative blood loss (4000 ml recorded losses in theatre) and is likely to have both consumptive and dilutional coagulopathies; these will be exacerbated by hypothermia. He has a history of ischaemic heart disease and may have developed myocardial dysfunction secondary to the increased afterload produced by the cross clamp applied to his aorta during surgery. During surgery, reperfusion of the lower limbs results in the release of lactic acid which depresses myocardial function. Similarly, hypothermia due to prolonged peri-operative exposure and large volume intravenous infusions will also depress the myocardium. Circulatory compensation mechanisms, particularly tachycardia and elevated systemic resistance, increase myocardial work and oxygen requirements at the very time that supply is threatened; this can also worsen myocardial dysfunction. Although not present in this patient, epidural anaesthesia can contribute to hypotension by sympathetic blockade.

## THERAPEUTIC GOALS

The therapeutic goals in this patient include:

1. The prompt reversal of hypotension to avoid hypoperfusion of vital organs.
2. The empirical treatment of hypotension, while instituting further monitoring to identify precisely the most prominent cause.
3. The frequent assessment of the patient's response to treatment.

## THERAPEUTIC OPTIONS BASED ON PHYSIOLOGICAL AND PHARMACOLOGICAL EFFECTS

The initial approach to the patient should follow the airway breathing circulation (ABC) algorithm developed for adult cardiac life support.

### Airway

Although the patient has been transferred from the operating room, intubated, ventilated and sedated, the position of the tracheal tube should be checked by auscultation of the chest and chest X-ray.

## Breathing

Breathing is maintained by controlled ventilation, adjusting the $F_iO_2$ to maintain the $P_aO_2$ between 9 and 12 kPa. If a tension pneumothorax is suspected of causing the hypotension, treatment involves immediate decompression by a large bore cannula in the anterior, second intercostal space at the mid-clavicular line, followed by formal tube thoracocentesis.

## Circulation

The circulation can be assessed clinically by the rate, rhythm and volume of the pulse. A difference >5°C between the peripheral temperature (hand skin probe) with the core temperature (oesophageal or nasal probe) suggests significant peripheral vasoconstriction. Pale, cool peripheries, and oliguria (urine output < $0.5\,ml.kg^{-1}.h^{-1}$) indicate hypoperfusion. The arterial line inserted in theatre allows direct, continuous measurement of blood pressure, but should be checked for accuracy by re-zeroing and examining the waveform for damping, as both could cause inaccuracies in measurement.

In establishing the cause of hypotension, the assessment of ventricular preload by CVP or pulmonary artery catheter (PAC) is crucial. CVP is increased by raised intra-thoracic pressure, impaired cardiac function, venoconstriction and circulatory overload. The effect of a 250-ml fluid challenge on the CVP reading is more useful than its absolute value. A smaller bolus (100 ml) should be used if it is likely that the hypotension is of cardiac origin. A volume challenge causing a persistent rise in CVP of over 3 mmHg suggests a well-filled circulating volume or poor ventricular function in a normovolaemic patient. Hypovolaemia is suggested if the fluid challenge increases the CVP value by <3 mmHg or causes a rise sustained for less than 10–15 min. If CVP measurement does not help in assessing ventricular preload, the use of a PAC or transoesophageal Doppler probe may provide further guidance.

## Pulmonary artery catheter

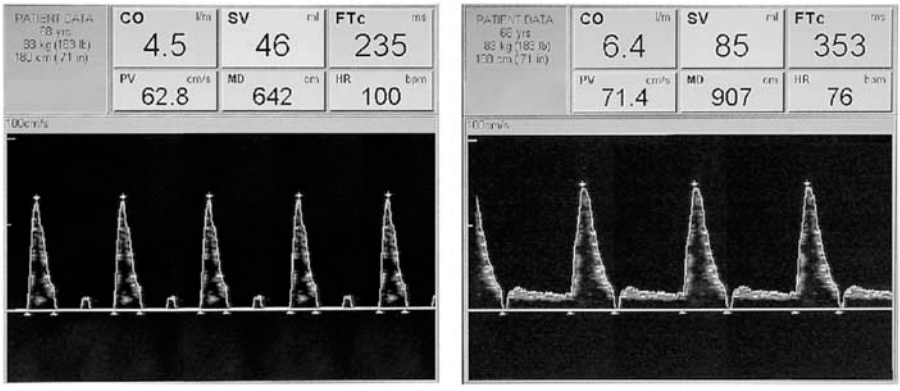
In some clinical situations, CVP values do not correlate with left atrial pressures (see above). In the described patient, this is a consideration because his previous myocardial infarction may have altered left ventricular compliance. PCWP is measured by wedging a flow-directed balloon-tipped catheter into a small pulmonary artery. When wedged, a continuous column of blood connects the catheter tip to the left atrium via the pulmonary capillaries and veins, and the measured pressure reflects left ventricular preload in most situations. Values should be interpreted with caution in left ventricular failure, mitral valve disease, raised intra-thoracic pressure, non-compliant left ventricles and aortic regurgitation. As with the CVP, response to a fluid challenge is more helpful than absolute values, but initial PCWP target values of 15–18 mmHg are appropriate. PACs allow intermittent

or continuous measurement of cardiac output by thermodilution. However, their use in critically ill patients is controversial. Complications of their use include arrhythmias, catheter knotting, pulmonary infarction, pulmonary artery rupture and infection.

## Transoesophageal Doppler

Transoesophageal Doppler ultrasound is an increasingly popular alternative to PAC insertion and is a less-invasive way of assessing circulatory volume status and cardiac function. Blood flow in the descending thoracic aorta is monitored via a probe placed in the distal oesophagus and gives a characteristic waveform (Figure 1.1).

The area under each waveform (i.e. the stroke distance) represents the stroke volume with 85% accuracy when corrected for patient's age, weight and height. The peak velocity measures myocardial contractility and normally declines with age. The flow time corrected for heart rate (FTc) is inversely proportional to systemic vascular resistance and is therefore low in hypovolaemia and arterial constriction, and gives a guide to fluid status. A fluid challenge increases FTc and stroke distance in hypovolaemia, but reduces peak velocity in poor ventricular function. Measurement




(a) (b)

*Figure 1.1* – Transoesophageal Doppler monitor screen demonstrating a patient: (a) with hypotension due to hypovolaemia. In this patient, the SV is relatively low (46 ml) and is associated with low FTc (235 ms) and the waveform can be seen to be peaked and narrow suggesting that flow is only occurring in the earliest part of systole. HR is also elevated; however, the PV is maintained at the near-normal for this patient suggesting poor contractility is not a problem. (b) following fluid resuscitation. After appropriate volume loading, the CO and SV have improved substantially with a reflex fall in HR. FTc is now at the normally targeted value (350–400 ms) and the waveform looks wider and fuller. PV has increased slightly showing that the heart was capable of better contractility with increased preload.

Abbreviations: CO, cardiac output; SV, stroke volume; FTc, flow time corrected; PV, peak velocity; MD, mean distance; HR, heart rate.

of stroke volume before and after the administration of a fluid challenge is a safe and effective method to evaluate post-operative hypotension. Fluid boluses should be continued until the rise in stroke volume is less than 10%, implying that the slope of the preload–contractility curve has flattened. Although extremely safe in normal usage, transoesophageal Doppler is contraindicated in patients with oesophageal pathology or marked coagulopathy. There are few reported complications even with prolonged use.

## Hypovolaemia

Patients may be hypovolaemic following surgery due to continuing or inadequate replacement of losses, or redistribution of intravascular volume. To enable rapid fluid resuscitation, at least two large venous cannulae should be sited. In patients with difficult venous access, insertion of a PAC introducer into a major vein provides wide bore venous access and allows subsequent flotation of a PAC if required. The primary objective is to restore the circulating volume as guided by the patient's response to fluid boluses (i.e. changes in pulse rate, blood pressure, peripheral perfusion, urine output, CVP and PCWP). A combination of colloids (i.e. gelatins, starches or albumin) and isotonic crystalloids can be used. Debate continues as to the 'best' fluid for resuscitation and maintenance in critically ill patients and, in the absence of strong evidence, no firm recommendations can be made. However, it must be remembered that colloids will expand the intravascular volume three times more than an equivalent volume of crystalloid due to their tendency to remain in the intravascular space. The results of blood tests taken on ICU admission (e.g. full blood count, haematocrit and coagulation) will determine blood products requirement. Transfusion is rarely indicated when the haemoglobin concentration is over $10\,g.dl^{-1}$. However, in this patient, an inability to correct hypovolaemia may suggest ongoing bleeding, in which case transfusion should be instigated. Transfusion is almost always indicated when the haemoglobin concentration is below $6\,g.dl^{-1}$. The platelet count should be kept above $50 \times 10^9 l^{-1}$ as platelet function may be impaired by aspirin therapy in this patient. Activated partial thromboplastin time (APTT) and prothrombin time (PT) greater than 1.5 times the normal values require correction with fresh frozen plasma (FFP). Continued coagulopathy may require cryoprecipitate, especially if fibrinogen levels are less than $0.8\,g.l^{-1}$. Antifibrinolytic agents (e.g. aprotinin) may occasionally have a role. Fluid warming and warm air blankets will correct hypothermia. As the patient warms, there is an initial rise in lactate representing washout from previously underperfused tissues and an increased need for further intravascular volume expansion as the peripheries dilate. Sympathomimetics with vasoconstricting action are occasionally required to increase vascular tone transiently while fluid resuscitation occurs. However, long term or high doses of vasoconstrictors must be avoided in hypovolaemic patients. An urgent surgical review should be sought if abdominal girth increases or there is continuing blood loss into surgical drains, as a second laparotomy may be required.

## Myocardial dysfunction

If the hypotension persists despite adequate circulating volume (as indicated by CVP and PCWP) and there is no suspicion of sepsis, the cause is likely to be cardiogenic. Arrhythmias, myocardial infarction (old or new) and ongoing ischaemia (ST segment depression, T-wave inversion) can be assessed by a 12-lead ECG performed on admission to ICU. Biochemical abnormalities (e.g. hypokalaemia and hypomagnesaemia) predispose to arrhythmias and require correction as arrhythmias reduce ventricular filling and, hence, cardiac output. If arrhythmias persist despite high normal serum potassium (4.5–5.5 $mmol.l^{-1}$) and magnesium (1.0 $mmol.l^{-1}$) levels, mechanical stimulation should be excluded by withdrawing the CVP catheter and PAC into the superior vena cava. If this fails to correct the rhythm, direct current (DC) cardioversion (for atrial fibrillation, ventricular tachycardia), pacing (for complete heart block) or an amiodarone infusion may be indicated.

Laboratory measurement of cardiac enzymes may help to confirm or refute suspected myocardial infarction. Myocardial specific creatinine kinase (CKMB) rises 6 h after myocardial infarction. Troponin I is a more sensitive and specific marker of acute myocardial infarction than CKMB; it is normally undetectable and rises 6 h after myocardial infarction. It peaks at 12 h and falls between 5 and 9 days after the

*Table 1.1* – Cardiovascular drugs commonly used in patients following emergency abdominal aneurysm surgery.

| Drug | Dose ($\mu g.kg^{-1}.min^{-1}$) | Action | Site of action |
|---|---|---|---|
| Epinephrine | 0.01–1.0 | +++ heart rate<br>+++ contractility<br>++ arterial constriction | Primarily β1- and β2- with some α-agonism at high dose |
| Norepinephrine | 0.01–1.0 | +/− heart rate<br>+ contractility<br>+++ arterial constriction | Almost exclusively α-agonism |
| Dopamine | 1–20 | ++ heart rate<br>++ contractility<br>++ arterial vasoconstriction | β-agonism at lower doses with increasing α-action at higher doses. Renal effect is seen experimentally but has little proven benefit in patients |
| Dopexamine | 0.5 | ++ heart rate<br>+ contractility<br>++ splanchnic and renal dilatation | β2-agonism and DA-1 agonism with some evidence of benefit on urine output |
| Enoximone | 0.5–1.0* then 5–20 | + heart rate<br>+++ contractility<br>+++ arterial dilatation | Phosphodiesterase inhibition increasing cAMP levels |
| Glyceryl trinitrate | 0.2–5.0 | +++ venodilatation<br>+ arterial dilatation | Veno- and vasodilatation by nitric oxide production |

*$\mu g.kg^{-1}$ bolus.

infarct. Fibrinolytic therapy is contraindicated after surgery and the treatment of myocardial infarction or ischaemia is, therefore, supportive.

The initial management of cardiac dysfunction involves optimisation of preload by fluid challenges to achieve a CVP or PCWP of 16–18 mmHg. This is followed by vasoactive drug support using noradrenaline, adrenaline or dobutamine, to achieve a mean blood pressure of 70–80 mmHg. Haemodynamic monitoring (PAC or transoesophageal Doppler) helps guide choice of inotrope (Table 1.1).

Sympathomimetics increase vascular tone by action on peripheral α-receptors, and heart rate and contractility by their effects on cardiac α- and β-receptors. Phosphodiesterase inhibitors (e.g. milrinone, amrinone and enoximone) improve contractility by increasing cyclic adenosine monophosphate levels but also cause vasodilatation. Vasodilators help decrease PCWP in the presence of pulmonary oedema and improve cardiac output by reducing afterload.

## USUAL OUTCOME

Emergency aortic aneurysm repair is associated with 50–60% mortality; pre-operative hypotension, intraperitoneal rupture, pre-operative coagulopathy and pre-operative cardiac arrest have been identified as independent predictors of increased mortality.

## KEY LEARNING POINTS

1. The intravascular volume should be normalised by the infusion of intravenous fluids.
2. The response of CVP and PCWP to a fluid bolus is a useful indicator of volume and cardiac status.
3. Early surgical review is required if hypovolaemia and hypotension persists despite apparently adequate fluid replacement.
4. Myocardial contractility and vascular tone may require support with inotropic or vasoconstrictor drugs.

### Further reading

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# 2

# TRAUMATIC LUNG CONTUSION

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## CASE SCENARIO

A 23-year-old male is admitted after a motor vehicle crash in which he sustained chest and abdominal injuries including multiple left-sided rib fractures and a ruptured spleen. He required an emergency splenectomy, and was transfused 10 units of blood prior to his admission to the intensive care unit (ICU), where he remains intubated and ventilated. Five hours after admission to ICU, his blood pressure and pulse are stable, but his arterial oxygen saturation has fallen from 96% to 85% despite 100% inspired oxygen.

## DEFINITION AND DESCRIPTION OF THE PROBLEM

This patient has life threatening hypoxaemic respiratory failure, which needs to be corrected urgently. A number of mechanisms may cause respiratory failure in ventilated patients, who have suffered severe chest trauma; these include:

1. *Early complications of chest trauma:*
    - airway injury (e.g. trachea, bronchus and alveolus);
    - pneumothorax and haemothorax;
    - flail chest (i.e. three or more consecutive ribs with each broken in at least two places);
    - pulmonary contusion;
    - diaphragmatic rupture.
2. *Delayed complications of chest trauma:*
    - late presentation of acute complications listed above;
    - atelectasis;