Update in
Anaesthesia

Cerebral challenge 2

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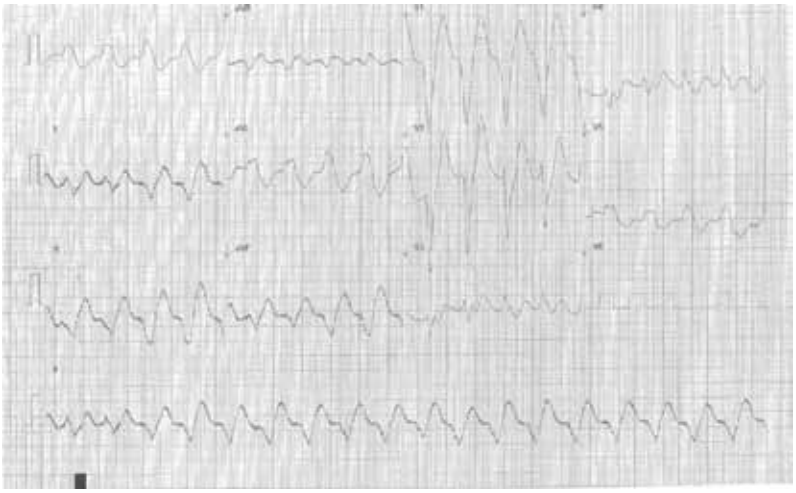
Case 1

You are called to the emergency department, where a 43-year-old female with no medical history has been brought in by ambulance after taking an intentional overdose. She was found with a Glasgow Coma Score of 3/15 and was intubated by the paramedics at the scene. Two empty packets of amitriptyline were found beside her. In the ambulance she had a palpable brachial pulse but the ECG monitor showed intermittent irregular broad complexes. A 12-lead ECG (Figure 1) was recorded in ED shortly after her arrival.



1. What does the ECG show?
2. What is the likely cause of these abnormalities?
3. How would you manage this patient?
4. What new drug is gaining credibility in the treatment of such an overdose?

Figure 1. *ECG of patient 1*

Case 2

A previously well 52-year-old woman is brought to the emergency department having been trampled by her horse. She walked briefly after the injury, but complained of severe chest and abdominal pain when the paramedics arrived. While on her way to ED, she became hypotensive, tachycardic and drowsy. Examination findings on arrival were: BP 70/40, HR 130min^{-1}, cool peripheries, tense abdomen and unresponsive to painful stimulus. The admitting doctor began resuscitation, including assisted ventilation with a bag and mask. You have been called to secure the airway, and the trauma team has been summoned. The surgeon suspects a ruptured spleen. As part of the primary survey, a chest Xray was taken following intubation (Figure 2).

1. What are the main abnormalities that this chest Xray demonstrates?
2. How should the team investigate and manage the suspected splenic rupture?

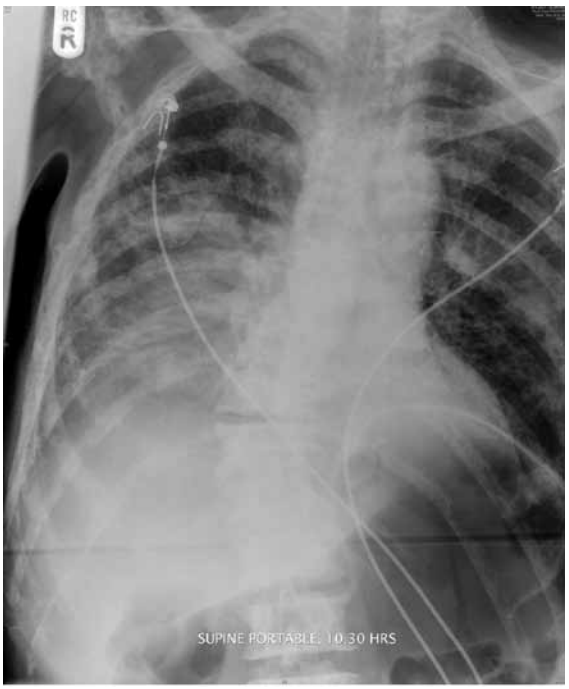


Figure 2. *Chest Xray of patient 2 after tracheal intubation*

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Case 3

A 14-year-old old boy presents into ED with a history of general malaise and reduced exercise tolerance over the last 10 days. His parents are concerned by his progressive lethargy and shortness of breath and have brought him in to ED today after he complained of abdominal pain. He is normally fit and well. On examination he looks unwell and is lethargic. He is afebrile, tachycardic and has a blood pressure of 76/40. Heart sounds are normal but quiet. He has cool peripheries, a central capillary refill time of 3 seconds and a weak pulse. Examination of his abdomen reveals diffuse discomfort to palpation with a palpable liver edge but no evidence of peritonism.

You order a chest Xray and an ECG.

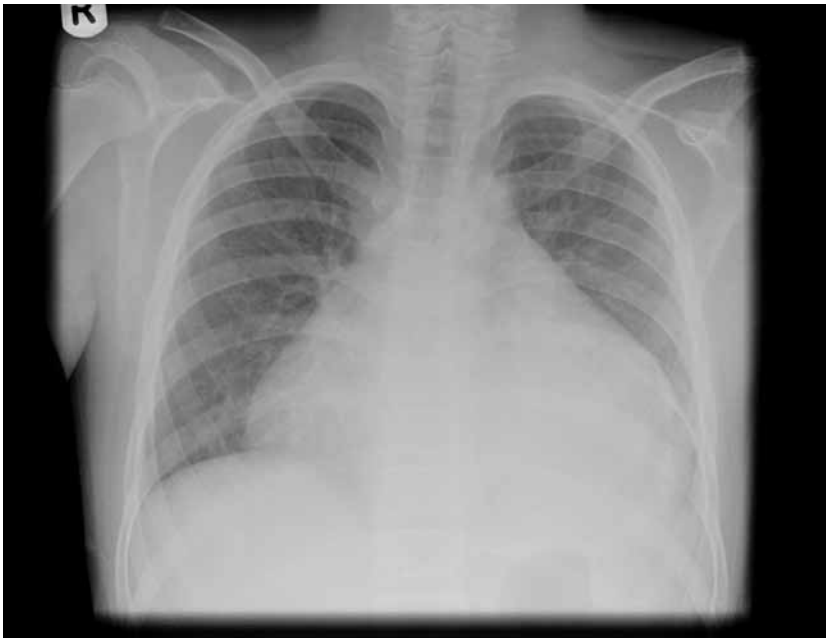


Figure 3. *Chest Xray of patient 3*

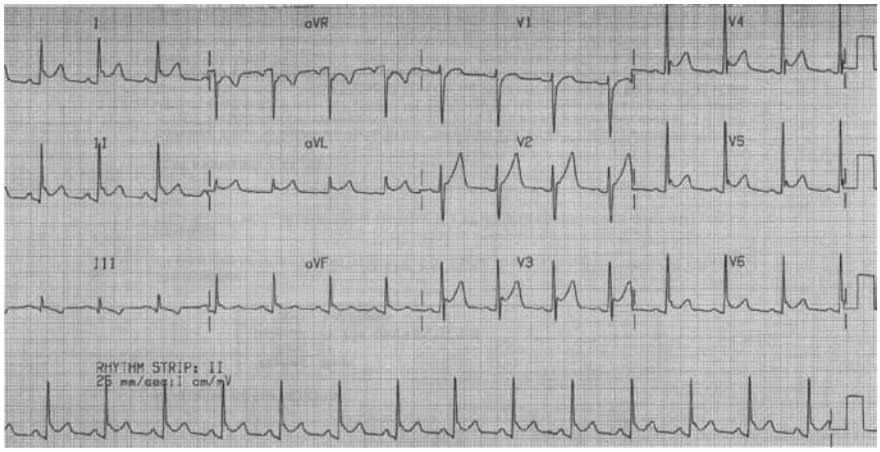


Figure 4. *ECG of patient 3*

1. What abnormality does the Xray show?
2. What does the ECG show?
3. Given the history and the findings of the Xray and ECG, what is the likely diagnosis?
4. What other possible findings on examination are consistent with this diagnosis?
5. What are the main causes of the most likely diagnosis?
6. What are the anaesthetic implications of caring for a child like this?

DISCUSSION

Case 1

The ECG shows broad QRS complexes (lasting 0.19 seconds or 190ms); the normal QRS duration is less than 0.12 seconds (3 small squares with a standard ECG recording).

There is also a prolonged QT interval (0.55 seconds, 550ms). The QT interval is measured from the beginning of the Q wave to the end of the T wave (Figure 5) and, as it varies with heart rate, it should be corrected for this. The normal corrected QT interval (QTc) is less than 0.45 seconds in males and less than 0.47 seconds in females. The QTc may be calculated using the formula QTc = QT/√R-R interval.

Prolonged QTc is associated with an increased risk of developing life-threatening arrhythmias such ventricular tachycardia and ventricular fibrillation.

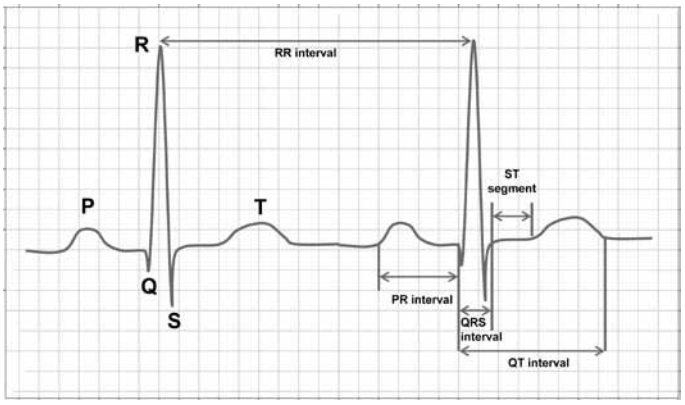


Figure 5. *A typical ECG complex with normal morphology showing standard intervals. Normal values are: PR interval, 0.12s (3 squares) - 0.2s (5 squares); QRS interval, <0.12s (3 squares); QT interval, <0.45s (males), <0.47s (females)*

Given the history, the likely cause of these abnormalities is an amitriptyline overdose. Amitriptyline is a tricyclic antidepressant (TCA) whose main mechanism of action is serotonin and norepinephrine reuptake inhibition at the postganglioninic membrane. In addition, it inhibits sodium channels, L-type calcium channels, certain voltage-gated potassium channels and has anticholinergic effects. This broad spectrum of actions accounts for its central and cardiovascular toxicity.

Table 1. *Causes of a prolonged QT interval*

Genetic	long QT syndrome eg. Romano-Ward, Jervell-Lange-Nielson
Drug induced	amiodarone, quinidine, sotalol, haloperidol, clarythromycin, tricyclic antidepressants, alcohol and many others
Metabolic	hypothyroidism

Table 2. *Symptoms and signs of TCA toxicity*

Peripheral	dry skin, urinary retention
Central	drowsiness, hyper-reflexia, respiratory depression, seizures
Cardiovascular	intraventricular conduction delay (QRS, PR & QT prolongation), hypotension, ventricular tachycardia/fibrillation

Your management of this patient should follow an 'ABCD' approach. In this case, particular attention should be paid to:

1. Correcting hypoxia caused by a depressed conscious level or seizures.
2. Treating arrhythmias by correcting any acid-base disturbance. Adults with a tachycardia >100bpm and/or ECG changes should be treated with 50mmol of 8.4% sodium bicarbonate solution even in the absence of acidosis. This interferes with the binding of TCAs to the myocardium.
3. Amiodarone should be avoided when treating arrhythmias as it prolongs the QT interval, and may exacerbate hypotension.
4. Seizures should be controlled with benzodiazepines. Phenytoin should be avoided as it blocks sodium channels and exacerbates TCA mediated conduction delay.
5. Resuscitation may be prolonged, due to the duration of action of amitriptyline.

There is increasing evidence for the use of Intralipid® (lipid emulsion) in the management of a variety of drug toxidromes. First used in 2006, this lipid emulsion is now the first line treatment for local anaesthetic toxicity and its use has been successfully extended to treat TCA, calcium channel antagonist, beta-blocker and antipsychotic overdose. Its mechanism of action remains uncertain, but may be via the absorption of lipid-soluble drugs into fat droplets, effectively removing them from the circulation. Intralipid® may be considered in severe cases of TCA overdose which are refractory to conventional treatment.[1]

Intralipid® dose guide

There is no robust evidence based regimen for the use of intralipid in TCA overdose, but a protocol guiding the use of intralipid in local anaesthetic toxicity may be used. This guideline, endorsed by both the American Society for Regional Anaesthesia and the Association of Anaesthetists of Great Britain and Ireland, is shown below.[2]

1.5ml.kg^{-1} bolus of 20% lipid emulsion over 1 min

followed by

15ml.kg^{-1}.h^{-1} infusion of 20% lipid emulsion

If cardiovascular instability persists after 5 min or a previously stable circulation deteriorates:

A maximum of 2 repeat boluses of **1.5 ml.kg^{-1}** may be given at 5 min intervals

The infusion rate may be doubled to **30ml.kg^{-1}.h^{-1}**

Continue the infusion until cardiovascular stability is restored or the maximum dose of **12 ml.kg^{-1}** 20% lipid emulsion is reached

REFERENCES

1. www.lipidrescue.org
2. www.aagbi.org/sites/default/files/la_toxicity_2010_0.pdf

Case 2

The Xray shows a number of abnormalities. Most prominent is the significant bilateral air space shadowing, predominantly in the right mid-zone and base; this is most likely to be due to significant pulmonary contusion. There is a very enlarged gastric bubble and a coincidental finding of a marked thoracic scoliosis.

Gastric insufflation can often occur during resuscitation as a result of hand ventilation through a partially obstructed airway. Meticulous attention to preserving airway patency will decrease gastric inflation. Useful measures to achieve this include:

1. Head tilt/chin lift or jaw thrust with in-line stabilisation where cervical injury is suspected.

2. Early use of airway adjuncts (e.g. oropharyngeal or nasopharyngeal airway device, noting that the latter is contraindicated in suspected base of skull fracture).

3. Synchronisation of bag-squeezing with any patient respiratory effort.

4. Avoidance of excessive inflation pressures and tidal volumes.

If bag and mask ventilation has been necessary for a significant period, gastric decompression via a nasogastric or orogastric tube should be performed as soon as possible if there are no contraindications. The tube position must be confirmed on Xray or trauma CT scan.

There is nothing on the chest Xray to explain the degree of haemodynamic compromise in this patient. The surgeon is probably correct to suspect abdominal injury and bleeding. Despite its position under the left ribcage, the spleen is the organ most commonly injured in blunt abdominal trauma. The adult spleen weighs 150-200g and receives approximately 5% of cardiac output.

Splenic injuries should be treated non-operatively where possible. Conservative management is currently favoured for those patients who are haemodynamically stable, have no other significant injuries, are aged under 55 years and maintain a steady haemoglobin level for 12 to 48 hours. A CT scan is the investigation of choice for these patients. The American Association for the Surgery of Trauma (AAST) have the following injury grading system.

Those with significant compromise require urgent intervention. Your patient falls into this category. In many hospitals, an urgent laparotomy and splenectomy would be the intervention of choice. Recently splenic angio-embolization by an interventional radiologist has gained popularity in centres where this facility is available at short notice. This approach avoids the surgical risks associated with a laparotomy, but the patient is still likely to require an anaesthetic. A surgeon and operating room facilities should remain on standby when unstable patients are managed in this way.

The role of CT scanning for unstable patients with suspected high grade splenic injuries has also evolved alongside the development of splenic angio-embolisation (SAE). It was previously accepted that delaying transfer to theatre in order to perform a CT scan would cost valuable time and lead to increased morbidity and mortality. Now, with the advent of much faster scanners and in hospitals with suitable facilities, it can be possible to perform a rapid trauma CT scan followed by urgent SAE. In these centres, imaging may be considered even in those patients presenting with cardiovascular instability.

Tranexamic acid in trauma

Adult regime:	loading dose 1000mg in 100ml 0.9% saline over 10 min
	infusion 1000mg in 100ml 0.9% saline over 8hrs

Grade I

- subcapsular haematoma < 10% of surface area
- capsular laceration < 1cm depth

Grade II

- subcapsular haematoma 10 - 50% of surface area
- intraparenchymal haematoma <5cm in diameter
- laceration 1 - 3cm depth not involving trabecular vessels

Grade III

- subcapsular haematoma >50% of surface area or expanding
- intraparenchymal haematoma >5cm or expanding
- laceration >3cm depth or involving trabecular vessels
- ruptured subcapsular or parenchymal haematoma

Grade IV

- laceration involving segmental or hilar vessels with major devascularization (>25% of spleen)

Grade V

- shattered spleen
- hilar vascular injury with devascularised spleen

A recent study of the value of tranexamic acid in trauma (CRASH-2), showed that administration of this drug within 4 hours of injury is associated with decreased mortality.[4] Tranexamic acid is an antifibrinolytic agent whose mechanism of action is to inhibit the conversion of plasminogen to plasmin, the molecule responsible for fibrin degradation.

The spleen is the main site of production of IgM antibodies required for the body's immune response to encapsulated micro-organisms. Patients who undergo splenectomy following trauma have an increased susceptibility to infection by *H. influenza, S. pneumonia* and *meningococcus*, and should be vaccinated against these bacteria at least 14 days post-operatively. Earlier immunisation has been associated with lower subsequent antibody function and is not recommended.[5] Prophylactic daily penicillin is recommended in asplenic children and should be considered in otherwise immunocompromised adults although regional guidelines vary.

REFERENCES

1. Tinkoff G, Esposito TJ, Reed J et al. American Association for the Surgery of Trauma Organ Injury Scale I: spleen, liver and kidney, validation based on the National Trauma Data Bank. *J Am Coll Surg* 2008; **207**: 646.

2. The CRASH-2 collaborators. The importance of early treatment with tranexamic acid in bleeding trauma patients: an exploratory analysis of the CRASH-2 randomised controlled trial. *Lancet* 2010; **376**: 23-32.

3. Shatz DV, Schinsky MF, Pais LB et al. Immune rsponses of splenectomised trauma patients to the 23-valent pneumococcal polysaccharide vaccine at 1 vs 7 vs 14 days after splenectomy. *J Trauma* 1998; **44**: 760.

Case 3

The Xray shows the patient's heart is large and globular with sharp outlines. Given the history, the diagnosis is likely to be pericardial effusion with cardiac tamponade. Pericardial collections of greater than 250ml cause the heart shadow to enlarge and become globular or pear shaped in appearance.

The typical ECG findings associated with a pericardial effusion are low-voltage QRS complexes and a sinus tachycardia. Large effusions can allow the heart to change position within the pericardium from beat to beat (known as a 'swinging heart'). This can occasionally be seen as beat to beat changes in the appearance of the QRS complex, a phenomenon known as 'electrical alternans' (Figure 6).

Pericarditis is the most common cause of a pericardial effusion seen in the UK and an ECG is diagnostic (although it may be normal in 10% of cases). Acutely, raised ST segments with a 'saddle shape' are often seen, as shown in Figure 4. The ST segment is always depressed in aVR. PR segment depression is sometimes seen and whilst it is highly diagnostic it has to be specifically sought.

ST elevation caused by pericarditis is often misdiagnosed as an ST elevation myocardial infarction in leads V2, V3 & V4. Infarct related ST elevation is seen in territorial leads, e.g. anterior or inferior and rarely in multiple territories, while ST elevation seen in pericarditis should be global. Thrombolysing a patient with acute pericarditis, misdiagnosed as acute MI, can cause a catastrophic haemopericardium.

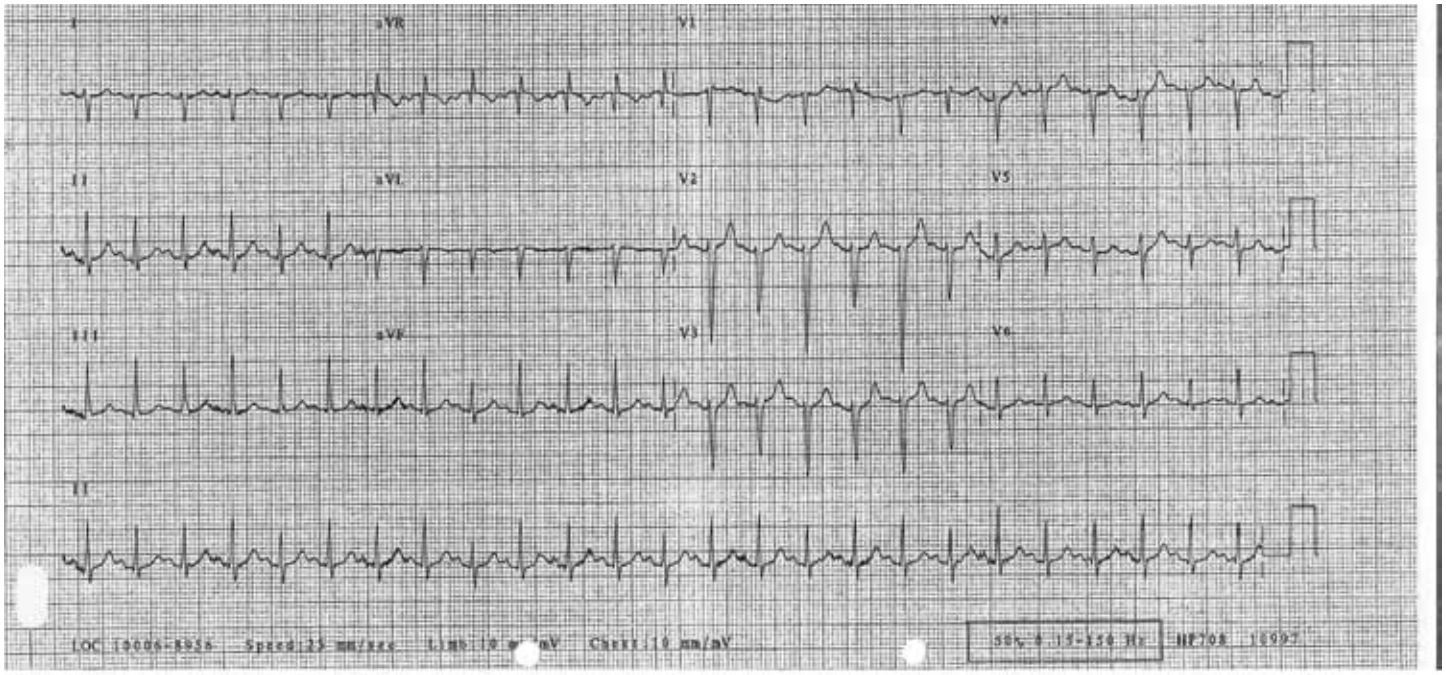
Cardiac tamponade occurs when the volume of the effusion between the pericardial sac and the heart muscle is sufficient to cause compression of the cardiac chambers. The effects of compression are greatest on the right atrium and ventricle, as these form a lower pressure system, therefore the clinical effects of compression are those of right ventricular failure. The main variable that contributes to the magnitude of these haemodynamics effects is the speed of accumulation of the effusion. Acute tamponade in an adult may occur when little as 100-200ml of fluid accumulates in the pericardial sac, whilst chronic effusions can reach volumes of 1000ml without clinical signs of tamponade. The speed of onset is often dependent on age, cardiovascular 'fitness', the cause of the effusion and the response to any treatment. The size of the effusion itself does not correlate particularly well with the degree of haemodynamic compromise.

Presenting symptoms of cardiac tamponade

Acute	cardiac arrest (pulseless electrical activity) hypotension shock confusion
Sub-acute	shortness of breath (cardiac failure) chest discomfort symptoms of complications of compromised circulation e.g. renal failure, liver/mesenteric ischaemia compression of adjacent structures e.g. hoarse voice

A pericardial effusion has a number of associated clinical features. Individually they are not diagnostic, but together they are suggestive. Beck's Triad of hypotension, distended neck veins and muffled heart

Figure 6. *An ECG (from a different patient), showing electrical alternans. This patient suffered acute pericardial tamponade due to bleeding, on the first postoperative night following coronary artery bypass surgery*



sounds is considered pathognomic of cardiac tamponade, but is only present in a minority of cases.

Signs of a pericardial effusion	
Heart sounds soft and distant	The effusion effectively dampens sound transmission through the thorax
Apex beat obscured	Dampening of the palpable beat by the effusion.
Pericardial friction rub	Often auscultated best over the apex May be present if effusion caused by pericarditis but rub subsides as effusion develops.

Signs of cardiac tamponade	
Raised jugular venous pressure	Caused by high right heart pressures
Hypotension	Secondary to reduced stroke volume (reduced right ventricular output, dictates low left ventricular output)
Pulsus paradoxus	Represents a drop in inspiratory systolic pressure of >10mmHg with respiration
Confusion	Secondary to low blood pressure

Causes of pericardial effusion

There are a large number of causes of pericardial effusion. They may be considered as pathologies that cause 'acute' or 'sub-acute' effusion or tamponade.

This child is haemodynamically unstable and, importantly, he has a fixed cardiac output due to compressive limitation on his right ventricular output. General anaesthetic will abolish any compensatory increase in his systemic vascular resistance and may reduce his heart rate and cardiac contractility. This will further compromise his circulation and possibly precipitate cardiac arrest and death. A general anaesthetic must be avoided if at all possible.

Needle aspiration of the effusion and drain insertion may be achieved under local anaesthesia. Aseptic technique is essential and ultrasound guidance should ideally be used. If time permits, application of local anaesthetic cream to the chest can achieve effective anaesthesia. This can then be supplemented with local anaesthetic infiltration if required. Parental assistance may be very helpful to try to keep the child relaxed and compliant with the treatment.

Monitoring of BP, HR and oxygen saturation is essential throughout any intervention. Poor patient tolerance may threaten the safety and efficacy of the procedure. Ketamine sedation may be considered in such cases with extreme caution. If this is absolutely necessary very small bolus doses should be used, starting with perhaps 0.125mg. kg^{-1} IV.

If a general anaesthetic is unavoidable then the following points should be considered.

1. The risks should be clearly explained and documented.
2. A preoperative echocardiogram to establish the degree of tamponade and impairment of cardiac function is helpful.
3. Central venous pressure and invasive arterial pressure monitoring should be achieved.
4. Pericardial drainage should be sought prior to surgery if possible.
5. Drugs that cause a reduction in systemic vascular resistance (SVR) or cardiac contractility should be avoided and the patient kept normotensive. Consider induction and maintenance of anaesthesia with ketamine, as it increases heart rate, contractility and SVR.
6. As positive pressure ventilation reduces venous return spontaneous breathing should be maintained if possible.
7. Hypovolaemia can precipitate tamponade in an otherwise haemodynamically asymptomatic effusion.
8. IV diuretics are contraindicated and may be fatal.

Acute tamponade/effusion

Myocardial infarction leading to cardiac tissue rupture

Trauma

Aortic dissection

Spontaneous bleeding, may be seen in;

- uraemia (inhibits platelet function)
- thrombocytopenia
- anti-coagulation

Cardiac surgery or pacing (iatrogenic trauma to cardiac tissue)

Sub-acute tamponade/effusion

Malignant disease

Ionising radiation

Systemic lupus erythematosis

Hypothyroidism

Idiopathic pericarditis

Infections

- TB
- bacterial