Cerebral challenge 1

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Case 1

A 72-year-old male with known ischaemic heart disease is admitted to recovery after cystectomy. He wakes up from the anaesthetic complaining of chest pain. A 12-lead electrocardiogram (ECG) is recorded.

1. What does this ECG show?

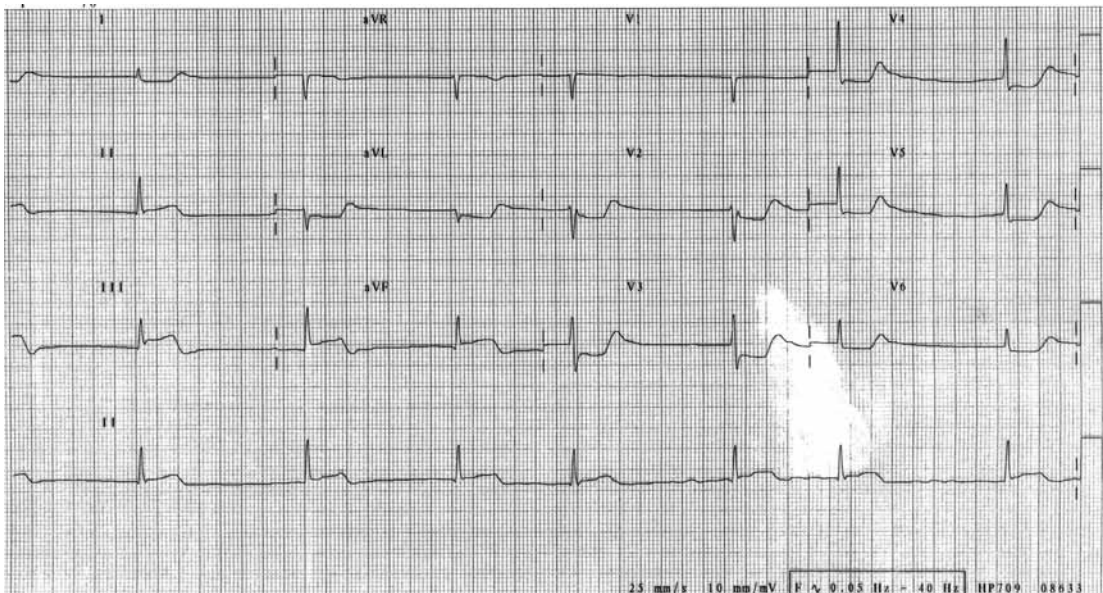2. How would you manage this patient given his recent major surgery?

Figure 1. *ECG of patient 1*

Case 2

A 19-year-old male motorcyclist is brought to the Emergency Department following a road traffic accident. On examination, his oxygen saturations are

95% on high flow oxygen with a patent airway, blood pressure is 90/70mmHg, heart rate is 118 bpm, and GCS is 7 (E2, V2, M3). You note that his GCS was 14 (E3 V5 M6) at the scene. His temperature is 34.2ºC. Tracheal intubation is performed. He is then taken to the CT scanner.

1. What does the CT head scan show?
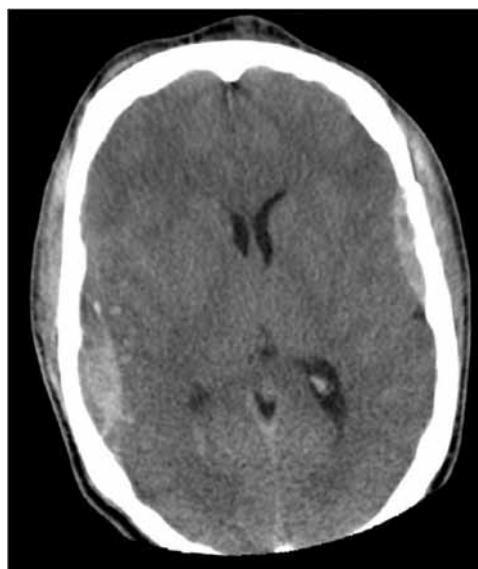
2. What are the critical care priorities for this patient?

Figure 2. *CT head scan of patient 2*

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Case 3

You are called to the Emergency Department, where a 20-year-old male has collapsed following an overdose. He was intubated at the scene with a GCS of 6. His SpO_2 is 84%, heart rate 96bpm, blood pressure 105/62, with equal reactive pupils. The paramedics report he has been 'difficult to bag' and on examination you note poor air entry over the left chest wall. A chest Xray has been performed.
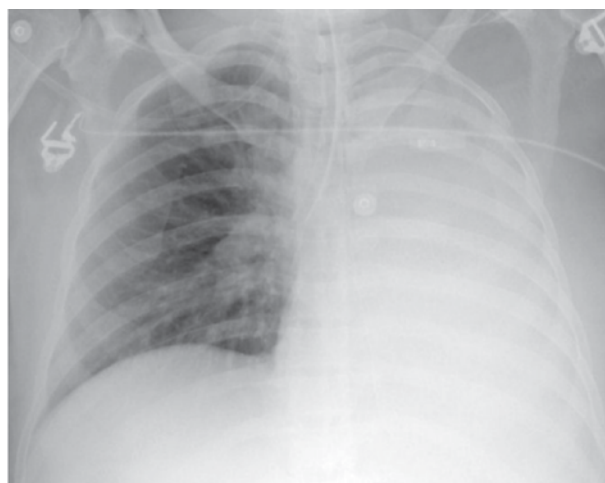
1. What does the chest radiograph show?

2. What are the differential diagnoses of this appearance on chest Xray?

3. What is the immediate management of this situation?

4. What are the risk factors that can predispose to these findings?

Figure 3. *Chest Xray of patient 3*

DISCUSSION

Case 1

The ECG in Figure 1 shows ST elevation in the inferior leads (leads II, III and aVF) indicating inferior ST elevation myocardial infarction (STEMI). There are with reciprocal anterior-lateral ST changes and slow atrial fibrillation.

STEMI is diagnosed by a history of characteristic chest pain and an ECG showing one of:

* ST-segment elevation of >1mm (1mV) in two or more adjacent limb leads (i.e. leads I, II or III).

* Elevation of >2mm (2mV) in two or more adjacent precordial leads (V1-V6).

* New onset left bundle branch block (LBBB).

These diagnostic criteria are important, as they are generally required to persuade a cardiologist to undertake PCI (percutaneous coronary intervention, i.e. angioplasty and stent insertion) in settings where this is available.

The term 'nodal rhythm' is used in situations where the atrioventricular (AV) node rather than the sinoatrial (SA) node sets the rate of contraction of the heart. This may be caused by a variety of pathologies affecting the SA node, however right coronary artery occlusion is a relatively common cause since the SA node is supplied by the right coronary artery (RCA) in 90% of individuals. In the remainder it is supplied by the circumflex artery. All myocardial cells have their own inherent rhythmicity - i.e. they set their own rate of contraction if left unstimulated. In health the natural faster rate of the SA node predominates, stimulating cells further down the conducting system of the heart at a faster rate than their own inherent rate. Unstimulated ventricular cells will generally contract at around 30-40bpm and so this is the rate seen in complete heart block. In this patient the cells with the next fastest inherent rate have taken over the pacing of the heart (as so-called 'escape pacemaker'). As narrow QRS complexes have been generated, this escape pacemaker lies within the usual conducting system and may be nodal (i.e. the AV node, sometimes called junctional) or infra-nodal.

Figure 4 illustrates that the RCA supplies the right atrium, right ventricle, and also, in the majority of patients, the atrio-ventricular (AV) node (in addition to the SA node).

Therefore patients with RCA ischaemia may present with:

* bradyarrhythmias (atrial fibrillation, heart block) due to AV node ischaemia, and
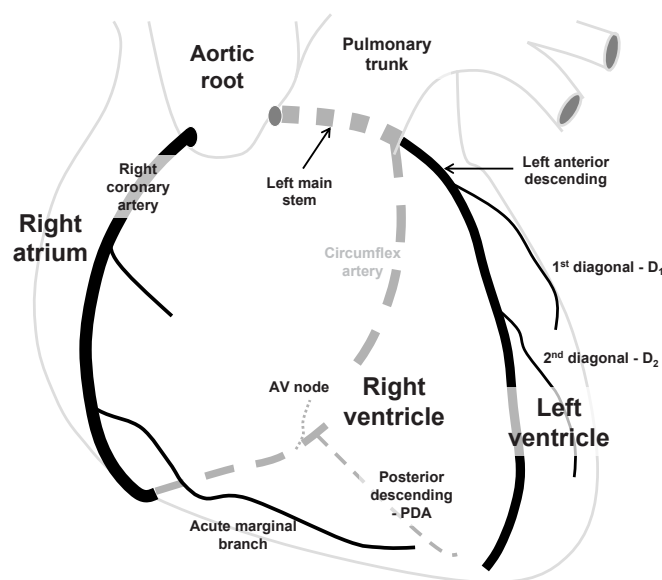
* right ventricular failure.

Figure 4. *Anatomy of the coronary arteries*

In most cases, the posterior descending artery that supplies the inferior left ventricular wall is also a branch of the RCA. Therefore some RCA infarcts, affecting predominantly the inferior part of the heart, also have posterior extension. Standard leads do not overlie the posterior aspect of the heart, however 'reciprocal' or opposite changes may be seen in the leads that lie over the anterior wall of the heart. So posterior
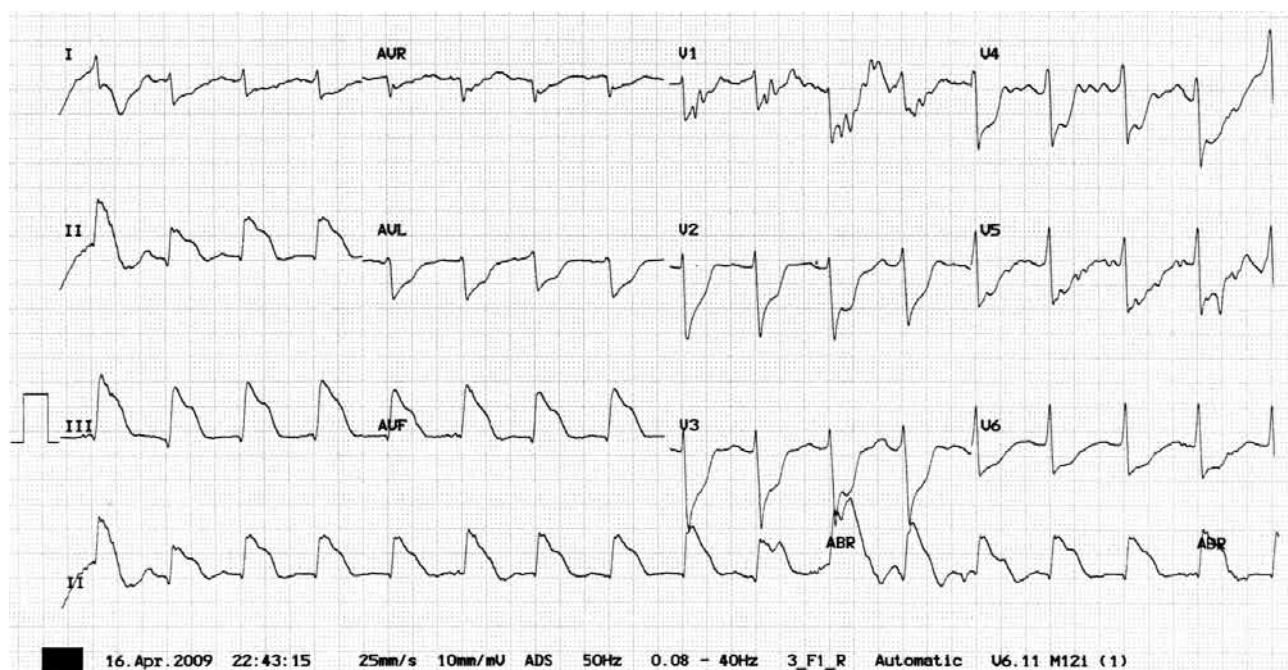
Figure 5. *'Tombstone' ST elevation in the inferior leads*

wall infarction may be seen as ST depression (the reciprocal of ST elevation) and R waves (the reciprocal of Q waves) in leads V1 and V2. This also explains why people describe the ST depression seen in leads V2-6 in Figure 1 as 'reciprocal' - these leads are opposite those directly over the affected inferior wall of the heart and so show the opposite changes.

Figure 5 is another illustration of an acute inferior MI, with so-called 'tombstone' ST elevation in the inferior leads. The 'tombstone' effect is due to markedly prolonged R waves secondary to a build up of potassium and extracellular metabolites in ischaemia. This accumulation depolarises the myocardium but then inhibits sodium channels, causing delayed transmural action potential transmission. The name of this ECG finding is related to the associated mortality.

The main treatment aims are of physiological optimisation and the reduction of further myocardial ischemic damage. The initial assessment and general early management should be rapid. For this patient, who has just undergone major body cavity surgery, all available therapies should be considered, but consideration of the risk and benefit may preclude them.

Management of acute MI in the perioperative setting:

Early therapies

- Oxygen therapy at 2-5L.min^{-1}

- Analgesia (morphine 2-10mg or diamorphine 2.5-10mg intravenously with accompanying antiemetic).

- Sublingual/intravenous nitrates if blood pressure allows. Be cautious in inferior MI if there are signs of right heart failure (high JVP, hepatic engorgement, peripheral oedema) - venodilation will reduce right ventricular filling and result in profound hypotension.

- Consider antiplatelet therapy (ideally aspirin 300mg and clopidogrel 300mg). This must be a joint decision between surgeon, cardiologist and anaesthetist, in order to weight the relative benefits of limiting myocardial damage, against the risk of postoperative bleeding.

- Immediate discussion with a senior cardiologist regarding patient suitability for reperfusion therapy (thrombolysis or PCI). Cystectomy will preclude this therapy in this patient, with the risk of catastrophic bleeding outweighing any potential benefit until the patient is outside the window for such therapy. Of note, coronary artery stent placement also has an essential component of antiplatelet therapy to prevent stent thrombosis, limiting its use in this setting.

- Beta-blockade (e.g. metoprolol 1-2mg up to 15-20mg) should be considered in the setting of tachycardia. Ensure that hypovolaemia and pain are not contributing factors. Aim for a heart rate 60 90bpm and a systolic blood pressure >100mmHg. Some experts recommend waiting 48-72hours before commencing beta blockade and this would be sensible in view of his major surgery. If already taking a beta-blocker this should be continued preoperatively.

- Intensive care unit admission for monitoring for 24-72 hours.

Later therapies

- Introduction of a statin (for cholesterol lowering and coronary artery plaque stability) and an ACE inhibitor to enhance ventricular function.

- Formal echocardiography to assess left and right ventricular performance.

- Formal assessment of coronary disease (angiography) once patient stability achieved.

Case 2

The CT head scan shows bilateral extra-dural haemorrhages (EDH) with mild ventricular compression indicating raised intracranial pressure, and a small volume of blood in the left ventricle. The larger haemorrhage on the right is confined posteriorly by the lambdoid suture and by the coronal suture anteriorly. No bony fractures are evident, but the scan should be viewed on 'bone windows' to exclude this. The mid-line is not shifted, but this may be a false reassurance, as bilateral injuries disguise this.

An extra-dural haemorrhage most commonly occurs following tempero-parietal trauma and is often secondary to laceration of either the middle meningeal artery and/or accompanying venous vessels. Less commonly, a dural venous sinus tear is responsible.

A history of decreasing consciousness is a 'red flag' warning sign of rising intracranial pressure (ICP) and EDH should be suspected. Classically, there will be a lucid interval shortly after the injury, prior to a fall in consciousness. Other symptoms are nausea, vomiting, worsening headache, confusion and seizures. On examination, neurological signs such as worsening GCS, unilateral or bilateral up-going planters, brisk reflexes, hemiparesis, and ipsilateral pupil dilation may be present. Other external signs of possible intracranial haemorrhage are scalp lacerations and depressed skull fractures. 'Coning' due to raised ICP manifests clinically as bradycardia and hypertension (the Cushings reflex) & are pre-terminal signs. Deep irregular breathing with bilateral spastic paresis occurs shortly before respiratory arrest.

EDH is a neurosurgical emergency. Management of EDH is focussed on stabilisation and resuscitation before urgent transfer to the closest neurosurgical unit for clot evacuation.

Whilst awaiting transfer to theatre or another centre, there are several aspects of his care that can be optimised to limit secondary brain injury, by maintaining cerebral perfusion pressure above 70mmHg.

REMEMBER: CPP = MAP – ICP

(CPP = Cerebral perfusion pressure, MAP = Mean arterial pressure, ICP = Intracranial pressure).

- Normal ICP is 5-12mmHg. In a head injured patient it is reasonable to assume it is 20mmHg and so a mean arterial pressure of 90mmHg is needed to achieve a CPP of 70mmHg.

- Blood pressure should be monitored regularly, and invasively if possible.

- If thoracic and lumbar spine have been cleared then patient should be 30° head up to improve cranial venous drainage.

- Endotracheal tubes should be taped in place rather than tied to prevent ties obstructing venous drainage.

- $PaCO_2$ should be maintained in the low normal range (4.5-5kPa).

- Maintain normoglyacemia.

- Maintain oxygen saturations >95%.

- Any seizures should be controlled (e.g.18mg.kg-1 phenytoin).

- A brief period of hyperventilation (to lower the $PaCO_2$) and/or mannitol 0.5g.kg^{-1} IV can be used to control acute surges in ICP. Hyperventilation should not be sustained as cerebral vasoconstriction may lead to further ischaemia.

An appropriate target is that all patients requiring emergency neurosurgery have a CT head with 1 hour of arrival, and transfer to a neurological centre is complete within 4 hours. This can be practically difficult to achieve. Adequate resuscitation must first be implemented to optimise the patient pre-transfer. However, transfer to a neurosurgical unit should occur as soon as possible to enable potentially definitive and life-saving treatment. Prolonged delay is inappropriate.

During the transfer cervical spine protection must be maintained. The patient should also receive the same standard of ICU physiological monitoring, and pupillary responses must be assessed every 15 minutes.

Case 3

The chest Xray shows right endobronchial intubation and complete atelectasis of the left lung.

The differential diagnoses of a complete hemithorax 'white-out' depend on the position of the trachea:

Position of trachea relative to opacification	Causes
Central	Consolidation Chest wall mass Pleural mass (e.g. mesothelioma) Pleural effusion/haemothorax (small to moderate) - may be a 'veiling opacity' if film taken supine
Towards opacification	Total lung collapse (e.g. endobronchial intubation) Pneumonectomy Pulmonary hypoplasia
Away from opacification	Pleural effusion/haemothorax (large)

Endobronchial intubation is most likely immediately prior to intubation, but may occur at any time during surgery or an ICU stay. It should be suspected in any case with low or falling arterial oxygen saturations, particularly when inflation pressures are high and expansion or air entry is asymmetrical. It is more likely to occur in children, where positioning is more critical due to the short length of the trachea. Where inubation has been difficult or prolonged there is a tendency to over insert the endotracheal tube and this is particularly likely when insertion has been blind, for example over a bougie.

Check that the endotracheal tube (ETT) is at an appropriate expected length for the patient. Deflate the cuff and slowly withdraw the ETT until breath sounds are auscultated on the anterior chest wall of the non-inflating side. This can be done under direct laryngoscopy or using a fiberoptic scope to check the position above the carina, and minimises the chance of inadvertent extubation. If you do not have chest Xray evidence of endobronchial intubation and the above measures do not improve bilateral lung expansion, consider alternative diagnoses. Once you are satisfied that the tube is a suitable length, is patent and you can auscultate equal air entry bilaterally, reinflate the cuff, re-secure the tube. In those with pulmonary disease a lung recruitment manoeuvre (e.g. $40cmH_2O$ for 40 seconds) followed by PEEP will help to reinflate the affected lung.

Other risk factors for bronchial intubation include:

- intubation by non-anaesthetists in emergency situations,

- use of uncut endotracheal tubes

- long insertion depths (>21cm at the teeth in women and >23cm in men),

- prone positioning,

- shared airway surgery,

- operations involving a pneumoperitoneum with steep head down positioning.

FURTHER READING

- Ratib K. Bhatia G. Uren N. Nolan J. Emergency Cardiology, 2nd Ed. Hodder Arnold, UK. 2011. p6-26.

- Ramrakha P. Hill J. Oxford Handbook of Cardiology. Oxford University Press, UK. 2006. p155-73.

- Recommendations for the Safe Transfer of Patients with Brain Injury. 2006. Association of Anaesthetists of Great Britain and Ireland.