Fast-Bleeped: A to E Series – Disability: Coma

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A medical educational series comprising practical instructional pieces on how to approach undifferentiated clinical problems in the acute setting

CASE PRESENTATION

You are the emergency department (ED) foundation year 2 (FY2) working in a busy district general hospital. A nurse approaches you because they are concerned about Mr K, a 44-year-old male, who presented with a sudden-onset, "thunderclap" headache. You are informed that the only past-medical history is of adult polycystic kidney disease (APKD). A computerised tomography (CT) scan of his head without intravenous contrast was ordered by one of your colleagues, but there was a hold up at the CT machine due to a concurrent major trauma emergency. They are concerned that over the last 15 minutes he has become more confused, unable to speak and has developed a reduced level of consciousness. They are worried and want you to urgently review him.

You are handed his national early warning score (NEWS) chart with the following observations:

- Heart rate (HR): 108 beats per minute (BPM)
- Blood pressure (BP): 185/97mmHg
- Respiratory rate (RR): 22 breaths per minute
- Oxygen saturations: 96% on room air
- Temperature: 37.2 degrees Celsius

You decide this patient needs urgent review. You go with the nurse to the patient's trolley in the majors cubicles and help to move him to the resuscitation room for further assessment and management.

WHAT SHOULD BE YOUR INITIAL APPROACH TO THIS PATIENT?

As you are moving to the resuscitation area, you remind yourself that with all patient encounters, there are two parallel trains of thought: diagnostic as well as managerial thinking. This patient has deteriorated in a short time whilst in the ED, which demonstrates a worrying trajectory. You also consider that in this case, a patient presenting with headache, there are already two red flags that should put you in a heightened state of awareness: sudden onset history of presentation and a drop in consciousness level.[1] This is probably someone who needs management before, or at the very least alongside a diagnostic process because you may need to intervene quickly to avoid further deterioration.

On arrival, the patient appears to be unresponsive to your greetings. Signs of life are present; the patient is breathing but in a laboured fashion and his pulse is present and regular but fast. All of this worries you. You ask the nursing team to immediately find a senior doctor to attend with you. You remind yourself of the importance of a systematic approach. You begin your ABCDE assessment according to advanced life support (ALS) guidelines.[2]

(A)irway: you look, listen, and feel for signs of breathing. You apply a non-rebreathe oxygen mask attached to 15 litres per minute of oxygen and notice that although the mask is misting, you hear snoring (stertor). There is no stridor or swelling around his mouth. He is not speaking in response to you, nor does he respond to a painful stimulus applied at the supraorbital notch. You are worried that this patient is at least partially obstructing his airway and you identify that with this level of consciousness there may be a threat to losing the airway patency further.[3]

You begin to think that you may need specialist help to support his airway. The ED registrar arrives and immediately walks to the head end of the trolley and supports the patient's airway with a jaw thrust manoeuvre. The patient's snoring stops. The nurse grabs an oropharyngeal airway (OP), and the ED registrar inserts it into the airway. He also provides some suction to remove oral-pharyngeal secretions. High-flow oxygen is then applied via a non-rebreathe mask. Mr K appears to be breathing more easily and you move on with your assessment.[2]

(B)reathing: You observe the monitor and check Mr K's RR and pattern. His RR is 23 breaths per minute, and he appears to be breathing irregularly. Oxygen saturations are 98% on high-flow oxygen. There is no use of accessory muscles of respiration and there is an equal chest rise and fall. The trachea is central. On auscultation of the chest there are normal breath sounds throughout the precordium with no added sounds such as crepitations or wheeze. There is a resonant percussion note which is equal bilaterally.[2] His irregular breathing pattern along with clear alteration in consciousness level points to an intra-cranial problem.[4]

(C)irculation: There is no intravenous (IV) access yet, so you ask the nurse to prepare to insert a large bore intravenous cannula and to send off a full blood count, urea, electrolyte, liver, and clotting profile as well as a c-reactive protein level. You also ask for a venous blood gas (VBG) to be run so that you have some useful results available immediately. Whilst this is taking place, you auscultate for heart sounds, which are normal. You feel the patient's hands and feet and are reassured that they are warm. The peripheral and central capillary refill time (CRT) is 3 and 2 seconds respectively. The patient has a pulse of 110

beats per minute and is regular. The BP is 180/125mmHg. You are worried about the hypertension and ask the nurse to show you how to automatically cycle the BP every 5 minutes on the monitor for continuous up-to-date readings. You recognise that the tachycardia and hypertension signify a syndrome of sympathetic nervous system overactivity.[4]

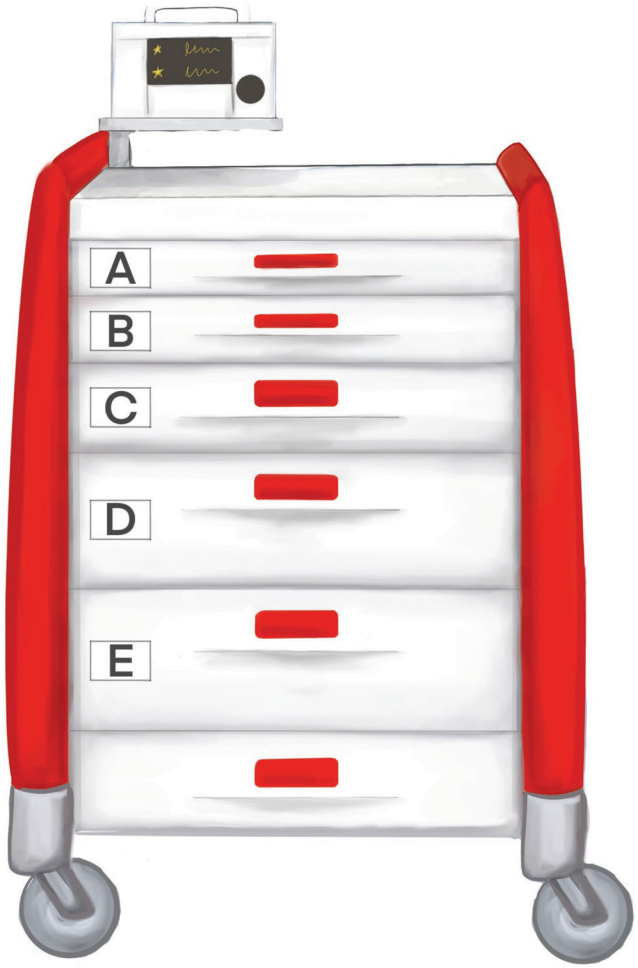
(D)isability: You already know there is a decreased consciousness level. You would like to quantify this so that the team can objectively track any improvement or deterioration in the level of consciousness over time. There are many ways to do this, for example, you could apply the alert, verbal, pain and unresponsive (AVPU) scale and note that the patient is (U)nresponsive because they do not respond to painful stimulus applied at the supra-orbital notch.[5] The Glasgow coma score (GCS) is made up of a score for (E)ye, (V)erbal and (M)otor response. It is only validated in traumatic head injury but is commonly used for other purposes in hospital.[6] To start to assess the GCS you look at the patient's eyes, they are closed and do not open to voice or painful stimulus. You score the patient as E1. The patient has not made any verbal response either so therefore you score V1. You note that the patient did not raise their arms to their head when applying your supra-orbital pressure – he is therefore not localising to pain. You perform a painful stimulus peripherally at the fingernail bed to look for a flexion-withdrawal, an abnormal flexion, or an abnormal extension response. When compressing a nail bed of the right hand there is no response, but on the left side the patient extends so you score the patient M2. You examine the patient's pupils; the right side is dilated to 5mm and is not responsive to light. Given you have identified some lateralising signs (a sign present on one side of the body not seen on the other) you suspect that the cause of this patient's coma could be a structural, intra-cerebral problem.[7] You ask the ED registrar how to organise a CT head urgently. Nevertheless, you remind yourself of anchoring bias, and the importance to stay open to other potential diagnoses.[8] For this reason, you continue your systematic assessment by quickly scanning the patient's ED prescription chart to check whether any sedative or analgesic drugs were given to the patient before their GCS change. He was only given 1 gram of paracetamol 1 hour ago. You would also like to get some collateral history from someone who knows Mr K to reassure yourself that there was no chance of an accidental or intentional overdose of medication before admission. You also specifically check the VBG for the venous blood glucose level because you recognise that hypoglycaemia can be a cause of reduced consciousness level.[9] The patient's venous blood glucose is 5mmol/L.

You quickly re-evaluate the airway, breathing and circulation. Your registrar is supporting the airway, the saturations are appropriate, and the circulation remains the same.[2] You continue to assess (E)xposure: The nurse takes a temperature, and it is 37.3 degrees Celsius. You ask for the door to be closed to ensure patient dignity whilst you and your team begin exposing the patient's body and looking for any additional signs. There are no signs of bleeding, abdominal guarding, or rashes on the body.

As you begin to wrap up your assessment, the intensive care registrar arrives, and you deliver a handover in a situation, background, assessment, and recommendation (SBAR) fashion:

> This is a 44-year-old male, who presented to the department with a sudden-onset, thunderclap headache with a past medical history of polycystic kidney disease. Whilst he awaited his CT head scan, he deteriorated with reduced consciousness level. I am not sure of his functional baseline, but I will have a look at his general practice records on the computer system. I suspect he is not maintaining his airway, so my colleague is supporting it with a jaw-thrust, and we are giving high-flow oxygen. He is tachycardic and a hypertensive. He is unresponsive with a GCS of 4 with abnormal extension of his left arm. His right pupil is dilated and unresponsive to light. I'm worried there is an intracranial cause for this, I think he needs airway support and an urgent CT scan of his head.

The intensive care registrar assesses Mr K and decides that urgent airway management is needed. He performs a rapid sequence induction (RSI) to secure the airway with a tracheal tube before transporting the patient to CT. The radiologist confirmed in his report that there was a large subarachnoid haemorrhage. The intensive care team take over the care of Mr K and start neuroprotective measures and contact the local neurosurgical centre.



What are the differentials of a patient with an acute reduced consciousness level or coma?

Common causes of reduced consciousness level include hypoxia, hypercapnia, analgesia/sedative drugs and cerebral hypoperfusion.[2] Coma can be defined as "an eyes-closed state of deep unconsciousness with an inappropriate response to stimulation that lasts for a prolonged period of time".[10]

Coma can be divided clinically into various phenotypes (see table 1):

Coma Types	Conditions
Coma with focal or lateralising features e.g., fixed gaze palsy, or hemiplegia.	• Cerebrovascular event (ischaemic or haemorrhagic) • Trauma • Space occupying lesion
Coma without focal or lateralising features but with meningism e.g., neck stiffness	• Meningo-encephalitis • Subarachnoid haemorrhage
Coma without focal/ lateralising features and without meningism.	• Toxins • Organ failure • Endocrine • Seizures • Pseudo-coma

Table 1: The various coma types and the conditions that tend to be associated with such.[11]

What are the general principles around assessing a patient with decreased consciousness level and coma?

Always seek help with a patient with acute decreased conscious level or coma. These patients may need urgent airway support. Without this, rapid hypoxia can follow.[12] An ABCDE assessment and full neurological examination is vital. The main principles[2,13] in the neurological examination of a comatose patient are:

- An assessment of the level of consciousness e.g. use of the GCS (table 2)
- Pattern of breathing
- Pupillary size and assessment of pupil reactivity
- Eye movements and vestibular ocular response
- Motor responses such as abnormal posturing (decerebration and decortication)
- Meningism (neck stiffness, photophobia)
- Administered drugs e.g., sedative agents, opiates.
- Past medical history

Initial investigations include full set of bloods and VBG, looking for electrolyte and acid-base derangements. A non-contrast CT of the head is also an urgent investigation, firstly to look for acute bleeding, but also any evidence of space occupying lesions or suggestion of raised intra-cranial pressure (ICP).[13]

How can we assess consciousness level using the Glasgow Coma Score and AVPU?

There are multiple scores and scales available to assess consciousness but the most widely known and used are the GCS and AVPU scale. AVPU is used as a quick and simple way to assess brain perfusion and conscious level. AVPU is the acronym for awake, verbal, pain and unresponsive.[5]

An alternative and commonly used scoring system is the GCS which was initially developed by Teasdale and Jennett in 1974 and has since been subjected to multiple reviews and updated approaches to improve its reliability.[6] The GCS helps in assessing the severity of brain dysfunction on initial presentation which then helps in decision making. Repeated assessments of the patient's score can demonstrate injury progression and helps with prognostication. The components of the GCS are shown in the table below.

Due to its common and widespread use, it is important to understand some of the problems and limitations of this scoring system as we use it. Although it is usual for health-care professionals to present GCS as a single score, this is not the intended way nor is it reliable to be presented as such. This is because the same GCS's but with points scored for different components of the score (for e.g., E1V1M2 vs E2V1M1) will have different mortality rates.[6,14] GCS is also commonly used in settings where reduced or loss of consciousness is affected by other factors such as drugs, alcohol, and other sedatives where its use is not validated. It is also important to note that GCS is not applicable for paediatric patients and so in such cases, a structured clinical assessment with the AVPU score should be used.[6,14]

What are the different types of intracranial haemorrhages and key features?

Intracranial bleeds are typically divided into four categories: epidural, subdural, subarachnoid and intraparenchymal haemorrhage.[15]

Epidural haemorrhage usually happens following a blunt force head trauma or penetrating head injury. Most commonly patients sustain a skull fracture (85-95%) with concurrent damage to the middle meningeal artery. This results in bleeding into the potential epidural space between dura and inner skull. Although usually arterial, venous bleeding happens in one third of all cases, commonly in paediatric patients. The dura is tightly associated to the inner skull, meaning it takes time for an epidural haematoma to develop in size, this explains the classic presentation of an initial loss of consciousness at the time of injury, followed by a lucid interval before the haematoma is of a size that will cause subsequent neurologic deterioration.[15]

Subdural haemorrhage occurs when a high force impact

Eyes (1-4 points)	Verbal response (1-5 points)	Motor response (1-6 points)
Open spontaneously (4)	Orientated (5)	Follows commands (6)
Open to voice (3)	Confused (4)	Moves to localised pain (5)
Open with painful stimulus (2)	Inappropriate words (3)	Flexion to withdrawal from pain (4)
Eyes not open (1)	Incomprehensible sounds (2)	Abnormal flexion (3)
	No sounds (1)	Abnormal extension (2)
		No response (1)

Table 2: Summary of the GCS.[6]

head trauma causes significant brain motion within the skull. This then causes the vessels traversing from brain to skull to stretch and rupture, resulting in bleeding into the subdural space. A subdural haemorrhage happens in 5-25% of significant head injury with the incidence increasing with age. In older patients with cerebral atrophy the traversing vessels are more vulnerable and even mild head injuries can result in significant subdural bleeding. Some causes of subdural haemorrhage include head trauma, coagulopathy, and vascular abnormality rupture.[16]

Subarachnoid haemorrhage (SAH) on the other hand can be divided into traumatic and atraumatic haemorrhage. An atraumatic bleed, as the name suggests, is when the bleeding occurs spontaneously and would fall under the definition of stroke (accounting for 5% of all cases of stroke). Atraumatic SAH can be further categorised into aneurysmal and non-aneurysmal. Typically, patients present complaining of a worst headache of their life or the textbook, thunderclap headache.[15]

Finally, intraparenchymal haemorrhage can be defined as bleeding into or within the brain parenchyma. It accounts for 10-20% of all strokes and classically presents with a history of stroke symptoms. The most common cause of non-traumatic intraparenchymal haemorrhage is hypertension causing spontaneous intra-cerebral haemorrhage. Other causes include trauma (penetrating or non-penetrating), arteriovenous malformation, coagulopathy, trauma, vasculitis, aneurysmal ruptures, tumour, or venous outflow obstruction.[15]

How are intracranial bleeds managed?

Management of patients with suspected or confirmed intracranial bleeds always begin with ensuring a secure airway, breathing and circulation. Intravenous access should be secured. The patient's GCS should be continuously monitored and any deterioration in neurological status or drop of GCS to less than 8, should prompt you to consider intubating the patient.[6, 15] You should then immediately consult the neurosurgical team in the region regarding definitive management of the bleed.

Epidural haemorrhages are managed by evacuating the haematoma and stopping further bleeding. However, small haematomas are typically managed non-surgically and monitored for resolution.[15]

Like epidural haemorrhage, subdural haemorrhages can be managed surgically with decompression via burr hole. Other approaches include craniotomy or twist drill hole. Management decisions depend on the size, location, resultant neurological injury, and patient factors like past medical history and baseline functional status. Patients with complex medical history and poor baseline function are at higher risk of surgical complications and therefore a non-surgical, conservative approach to management may be taken. Such approaches may be repeating imaging to monitor progression, stopping anticoagulation and reversal where necessary, platelet transfusions in cases of platelet dysfunction and a good control of blood pressure. Patients should also be repeatedly assessed for any neurological deterioration which might prompt change in management.[15, 16]

Definitive management of SAH differs depending on its cause. In aneurysmal SAH, initial medical management includes the use of nimodipine to prevent vasospasm. Emergency measures to alleviate raised ICP can be used such as mannitol or hypertonic saline.[17] Sometimes it may be necessary to insert an external ventricular drain to decompress the cerebral ventricles and lower ICP. This device also allows direct measurement of ICP which can help guide neuro-intensive care management. As with subdural haemorrhage, repeated imaging to assess improvement and reversal of anticoagulation or antiplatelet use should be done where applicable. Management of non-traumatic SAH varies based on its aetiology which could be clipping or coiling of an aneurysm or treating an arteriovenous malformation.[15, 17]

Finally, with intraparenchymal haemorrhage, initial management should be as all other bleeds with the aim to stabilise the patient first and foremost.[15, 17] In certain cases, aggressive surgical decompression is needed but in others, a craniectomy may be performed instead to help with cerebral swelling. Other options include the different catheter-based dissolutions of haemorrhage.[15]

Outcome of case

The patient was intubated and ventilated following an RSI. Due to the suspicion of an intra-cerebral catastrophe, neuroprotective measures to limit escalating ICP were initiated.[18] It is advised that patients should be nursed in a head up position and endotracheal tube ties around the neck should be avoided to encourage venous drainage via the jugular veins. Drugs should be administered to ensure deep sedation to limit cerebral oxygen demand and prevent any seizures or patient actions that could spike ICP e.g., endotracheal tube biting. A considered ventilation strategy should be adopted to avoid hypoxia and/or hypercapnia. $PaCO_2$ should be controlled to within the normal ranges because high $PaCO_2$ would cause cerebral vasodilatation and worsen ICP. A mean arterial pressure (MAP) should be agreed to ensure an adequate cerebral perfusion pressure. However, in an unsecured aneurysm there is a risk of ongoing/further bleeding, so a careful balance must be struck.[18]

A CT head scan without contrast demonstrated blood within the arachnoid space consistent with SAH. Following this, a contrast scan was performed which revealed an anterior communicating artery aneurysm. ADPKD is a common autosomal dominant genetic disorder causing progressive renal failure. The disease is characterised by multiple cysts that form within the cortex of both kidneys, but cysts can also be seen in other organs such as the liver and pancreas. There is also an association with cerebral aneurysm formation.[19]

A decision was made by the critical care team that transfer to a tertiary neurosurgical centre was required. The team acted quickly to ensure provision of portable equipment including a ventilator, oxygen supply and monitoring devices. They also needed to ensure that there were enough ongoing sedative drugs with a back-up supply of propofol 1% and fentanyl continuous intravenous infusions. The intensive care registrar inserted an invasive arterial line to allow continuous blood pressure monitoring and a means for easy blood draw for serial blood gas analysis. A urinary catheter was also inserted before transfer for urine output monitoring and to manage the patient's continence.

On arrival to the tertiary unit, endovascular coiling of the aneurysm was undertaken by the interventional radiology team.[17] On neurocritical care, the team started regular nimodipine via a nasogastric tube; which has been shown to reduce the risk of secondary vasospasm that can cause delayed cerebral ischaemia.[17] There was close haemodynamic and neurological monitoring and after several

days, the critical care team weaned the patient from his sedation. Unfortunately, the patient had sustained a significant neurological injury and never reached a point whereby the critical team could guarantee that he would maintain his own airway and ventilatory status post-extubation. Despite no sedation the patient remained completely tolerant of the endotracheal tube. Brain stem death was confirmed by two of the consultants on the critical care unit. The family were closely supported by the team, and the specialist nurses in organ donation (SNOD) were asked to speak to the family about the possibility of organ donation. The family agreed to stop ongoing life support and decided to donate Mr K's organs.

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ACKNOWLEDGEMENTS

I would like to thank Zuzanna Nowinka, a final year medical student at Lancaster University for producing the brilliant illustrations for this issue of Fast-Bleeped.

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Queen's Jubilee tree planting, picture courtesy of Greg Lambert and Beyond Radio.