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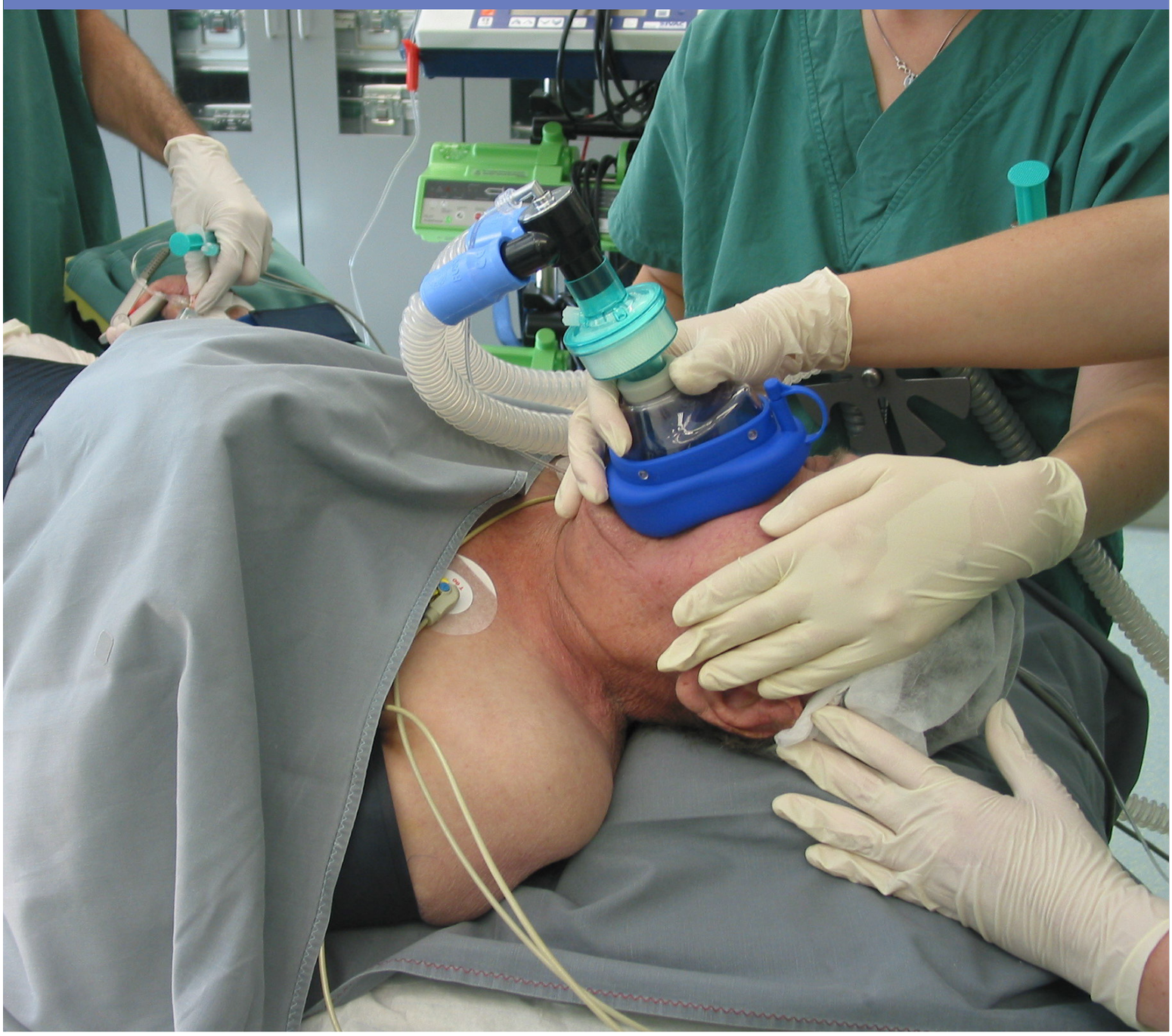

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Volume 4, Issue 2

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Foundation Years Journal is the ONLY journal for Foundation Years doctors and educators, specifically written according to the MMC curriculum. It focuses on one or two medical specialties per month, each issue delivers practical and informative articles tailored to the needs of junior doctors. The Journal closely follows the Foundation Years syllabus to provide the best educational value for junior doctors. In addition to good clinical and acute care articles, assessment questions give junior doctors the chance to gauge their learning.The answers will be published in the next issue, but 123Doc will advance answers to clinical tutor subscribers so they can engage their students in the learning process. Each issue provides comprehensive clinical cases for trainees as well as practical teaching assessments for educators. Readers will benefit from:

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FOUNDATION YEARS JOURNAL 2010

Volume 4, Issue 2

Welcome To This Edition Of The Foundation Years Journal

This issue covers a very wide range of subjects from audit to nutrition and management of obstetric emergencies and head injuries. The eagle-eyed among you will have spotted that the majority of the authors are anaesthetists or have critical care training. This was completely intentional for this edition, as anaesthesia as a subject covers such a broad range of areas and impacts on 60% of hospital activity, such that anaesthesia skills and expertise range from managing the acutely ill patient to chronic pain settings.

Despite this, very few Foundation training programmes involve anaesthesia or critical care as specific posts, therefore we hope we have been successful in illustrating not only the breadth and relevance of anaesthetics as a specialty, but also in providing you with a broad educational range of subjects mapped to the Foundation curriculum for your reading.

The authors trust that the practical nature of anaesthetics will come across in the writing and that you find these articles not only easy to read and understand, but also directly useful in your own practise, regardless of which programme of training you have started or intend to embark upon.

It is almost impossible to overstate how useful a training programme that involves anaesthesia or critical care would be to any medical career, and if the contents of this journal inspire enough curiosity in those of you that hadn't ever considered anaesthetics to embark on a training programme containing it, then as editors we will be very pleased indeed.

Enjoy!

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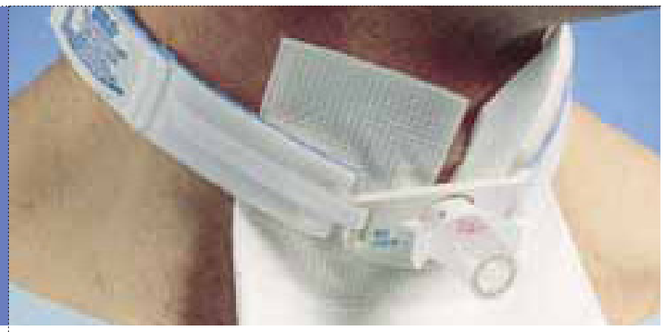
TRACHEOSTOMY MANAGEMENT

Prashast (JC1) and Daniele Bryden

Tracheostomy is a procedure that involves making an opening into the trachea.
Practical Procedures.



Tracheostomy is a procedure that involves making an opening into the trachea. Previously this was performed exclusively by Ear, Nose and Throat (ENT) surgeons but now an increasing number of tracheostomies are being performed by intensive care physicians. Tracheostomy in a critical care environment is performed in patients with upper airway obstruction, in respiratory failure to facilitate weaning from mechanical ventilation, in patients with absent airway reflexes and those unable to clear their airway secretions. It is important for Foundation trainees to have a basic understanding of the procedure and complications, as some patients discharged from critical care units will have had a tracheostomy and in some hospitals patients with tracheostomies still in situ may be managed outside of critical care areas.

Basic anatomy of the adult trachea

The airway can be divided into the upper and lower airway. The lower airway begins at the level of the vocal cords, and so the trachea forms a part of the lower airway. It is easily accessible in most patients and therefore an obvious choice for securing the airway surgically. The trachea begins at the lower end of the cricoid cartilage and then continues posteroinferiorly to the carina where it divides into left and right mainstem bronchi. Tracheal length varies between individuals from about 10–13cm, but averages about 11cm in an adult. It is a fibrocartilaginous structure with 18–22 cartilage rings that are deficient posteriorly[1].

There are a number of important structures in the vicinity of the trachea. The thyroid isthmus, inferior thyroid veins and branches of the anterior jugular vein all lie anterior to the trachea in the neck. The intrathoracic trachea lies posterior to the manubrium sternum, and the remains of the thymus, the arch of the aorta and its branches and the cardiac plexus are also close by. The oesophagus lies posterior to the trachea and the recurrent laryngeal nerves lie between these two structures.

Indications for a tracheostomy

There are four main general indications:

1. Upper airway obstruction: for example, tumour or abscess obstructing the upper airway. Such cases are usually managed by ENT surgeons as elective or urgent procedures. Cricothyroidotomy, which involves making an opening into the cricothyroid membrane, can be performed in a case of emergency airway obstruction.

2. Weaning: in mechanically ventilated patients who require a prolonged period of ventilation, a tracheostomy helps to decrease sedation, is considered to be more comfortable for the patient, and enables better mouth care than the alternative of an oral endotracheal tube.

3. Long-term mechanical ventilation: for example, patients with high spinal cord injuries who require long-term mechanical ventilation.

4. Inability to clear secretions or risk of aspiration: tracheostomy protects the airway from oral secretions and provides an easy access to the lower respiratory tract in patients who are unable to clear their own secretions (e.g. patients with cervical spine injuries or those with neuromuscular disease).

Procedure

The procedure for the modern surgical tracheostomy was first standardised by Chevalier Jackson in the early 1900s. Tracheostomy can be performed either surgically or percutaneously, but most tracheostomies performed on patients in a critical care environment are of the percutaneous type. Practice, however, varies between hospitals and if you are going to be looking after patients with tracheostomies, it is a good idea to familiarise yourself with the method and types of tube favoured in your hospital.

Both surgical and percutaneous tracheostomy access the trachea at the level of the second or the third tracheal ring. Surgical tracheostomy involves accessing the trachea after making a skin incision, retracting the strap muscles and then making a stoma in the trachea by cutting the tracheal rings[2].

Percutaneous tracheostomy requires a horizontal or vertical skin incision and then placement of a guide wire into the trachea under bronchoscopic guidance. The tracheal rings are separated using a dilator, and a tracheostomy tube is inserted once adequate dilation has been achieved. Different techniques of percutaneous tracheostomy involve different type of dilators but all involve the same basic principle of dilating around a guide wire.

TRACHEOSTOMY MANAGEMENT

Prashast (JC1) and Daniele Bryden

Tracheostomy tubes vary depending on the needs of the patient and the problems that the tracheostomy was inserted to overcome.
Practical Procedures.

Types of tracheostomy tubes

Tracheostomy tubes vary depending on the needs of the patient and the problems that the tracheostomy was inserted to overcome. Tubes are usually made of plastic (although occasionally metal) and documentation of the size and type of tube inserted should be in the patient's notes for you to check.

A tracheostomy tube consists of a curved tube with a flange and an obturator for insertion, which is removed immediately after insertion[3]. The tube keeps the stoma open and the flange secures the tube in place by means of ties.

Some tracheostomy tubes may have a cuff which is inflated when using it with mechanical ventilation to provide a seal. The cuff is deflated when used with a speaking valve so that the patient can speak.

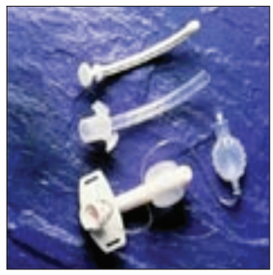
Tracheostomy tubes on the wards should have an inner tube which can be removed and cleaned, and also locks into the outer cannula so that it is not dislodged during coughing. Some tracheostomy tubes do not have an inner cannula and are therefore at greater risk of blockage.

Patients with long-term tracheostomies may have a fenestrated tube inserted which allows the patient to talk.

Different types of tracheostomy tubes that you may encounter therefore are:

1. Cuffed tracheostomy tubes without cannula: these tubes are used temporarily after a tracheostomy and are usually changed to tubes with an inner cannula before the patient is discharged to the ward.

2. Cuffed tracheostomy tubes with an inner cannula: the inner cannula in these tubes could be disposable or reusable depending on the type of tube. These tubes can be kept for a longer time and are the type most frequently seen in a ward environment.



3. Metal tracheostomy tube: they are uncuffed and are rarely used nowadays due to their high cost.

4. Tracheostomy tube with an adjustable flange: this is again a single cannula tracheostomy tube, it has a neck with a screw that can be used to adjust the length of the cannula that stays inside the trachea. These are temporarily used in special circumstances, for example, where the distance between the neck and the trachea is greater than normal and there is an increased risk of tracheal tube displacement with a conventional tube.

TRACHEOSTOMY MANAGEMENT

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Information on plastic tracheostomy tubes, about their size and dimensions, should be located on the flange of the tube. Different manufacturers produce tracheostomy tubes of the same size with different dimensions, so you should always ensure a tube of the same size and from the same manufacturer is available on the ward.

General care

Tracheostomy cuff pressures should be monitored and maintained in the range of 20–25cms of water. Higher pressures impair tracheal mucosal perfusion and predispose to tracheal stenosis and tracheomalacia. Cuff pressure should only be assessed using a pressure monitoring device and not by feeling the pilot balloon or hearing for minimal leak. If there is a leak around the tracheostomy cuff, the cuff pressure should be checked. If there is a leak in the cuff or persistence of leak around the cuff despite adequate pressure, the tube needs to be changed. In the latter case the tracheostomy tube should be replaced with a larger tube or a tube with a higher volume cuff. If the leak still persists tracheomalacia should be suspected[4].

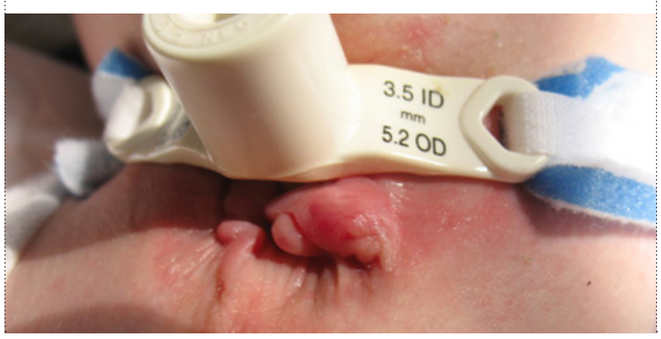
Tracheal suction should be performed when indicated rather than on a fixed time basis. The suction catheter should be inserted just beyond the tip of the tracheostomy tube, as insertion can lead to mucosal injury. The duration of suction should be as short as possible. Nebulised saline can be used to loosen thick tenacious secretions. Any inner cannula should be taken out and cleaned regularly.

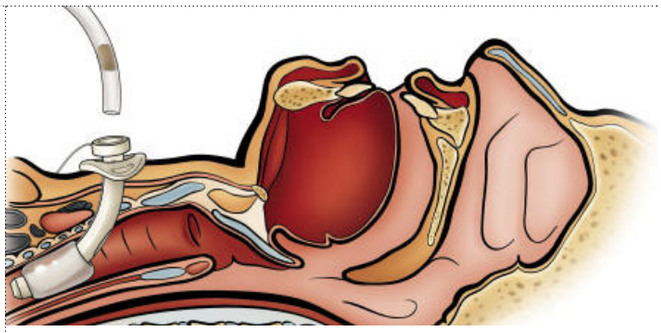
Tracheostomy leads to bypassing of the upper airway and therefore the gases reaching the trachea are colder and dryer. This could lead to desiccation and infection of the tracheal mucosa. Heat and moisture exchangers should therefore be used to protect the tracheal mucosa from drying up. The initial tracheostomy tube should be left in place for 5–7 days, and the first change should be performed by an experienced person.

Complications

Complications of tracheostomy can be divided into early and late – in all cases the patient should be assessed using the "ABCDE" approach, ventilation should be ensured and the patient discussed with a person experienced in the management of tracheostomies.

It is, however, reassuring that most ward-based tracheostomy management is straightforward if basic suctioning and humidification are maintained. Tracheostomy tubes should only be changed by medical and nursing staff with the appropriate training, and so you should not plan to undertake this procedure unsupervised.





Early complications

1. Tube obstruction: this could be caused by secretions, blood or abnormal positioning of the tube. The tube should be suctioned and any inner cannula cleaned. If the above manoeuvres fail, urgent expert help should be sought from an anaesthetist as tube repositioning using a bronchoscope or a change to a different type of tube may be required.
2. Haemorrhage: if minor, it may require packing or a suture to tighten the stoma. However, if it is significant the patient may require surgical intervention. Blood from the tracheostomy on suctioning may be related to the trauma of suctioning (which is self-limiting) or due to a structural problem (e.g. a tracheal granuloma). Treat haemorrhage from a tracheostomy stoma seriously and ask for advice.
3. Wound infection: this normally requires only simple therapy (e.g. cleaning and appropriate antibiotics after a wound swab), although in the case of a wound breakdown the patient may require intubation and debridement as a surgical procedure.
4. Subcutaneous emphysema: expert help should be sought and an urgent chest X-ray done to rule out pneumothorax. The airway should be maintained, endotracheal intubation may be required and therefore anaesthetic help should be sought if a patient develops this.
5. Tube dislodgement: if the patient is self-ventilating and maintaining their own airway, supplement with oxygen. If the patient is in respiratory distress, try to insert the same or a smaller tracheostomy tube if the tracheostomy site is well formed (i.e. more than 10 days since the tracheostomy was formed). If this cannot be done easily, occlude the stoma with a dressing and maintain the airway with a bag and mask. These patients may need endotracheal intubation so you will need to call for urgent help from an anaesthetist.

Late Complications

1. Swallowing problems and aspiration: speech and language assessment should be done although can be difficult with the tracheostomy in situ. Problems with swallowing could arise due to oesophageal compression from a tracheostomy cuff.
2. Tracheal stenosis: normally this does not lead to problems if mild. However, it might require specialist intervention if significant (e.g. affecting respiratory function).
3. Tracheoesophageal fistula: this is suspected in a patient who gets repeated chest infections, and there is failure wean from the ventilator or decannulate (see below) on the ward. It may require surgical correction.
4. Granulomas: form due to a foreign body reaction to the tracheostomy tube.
5. Persistent stoma: occurs in long-standing cases due to epithelialisation and may require surgical closure.

TRACHEOSTOMY MANAGEMENT

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Decannulation

Decannulation is the final process of removing the tracheostomy tube. It should be performed by an experienced person. If the tracheostomy was performed for upper airway obstruction, it should only be removed if the airway has been re-established. Patients should be clinically stable without any respiratory distress, they should be able to expectorate and have adequate swallowing.

To assess if the patient is ready for decannulation, the tube lumen can be blocked by placing your finger over the tube with the cuff down so the patient can breathe around the tracheostomy tube. The patient is then observed for any signs of respiratory distress. A Passy-Muir valve can be used for the same purpose (i.e. it occludes the tube and allows the patient to vocalise). This can be done for varying lengths of time depending on the duration of the tracheostomy and the capacity of the patient to breathe with the tube occluded and the cuff deflated. Never occlude a tracheostomy tube manually or with a valve when the cuff is inflated as the patient will have no route for exhalation. Once satisfied with the patient's breathing ability the tracheostomy tube can be removed. For patients with copious secretions a mini-tracheostomy tube can be left in place. In others the stoma can be covered with an occlusive dressing. The stoma gradually heals over a period of a few weeks although it may close within 24–48 hours.

Summary

Tracheostomy is a common procedure performed in intensive care patients. It is likely that as a Foundation doctor you would come across patients with tracheostomies on the wards in some hospitals. It is important to become familiar with the different types of tracheostomy tubes used in your hospital, and be aware of tracheostomy-related complications and ways to deal with them, including when to get expert assistance. In cases of doubt or in an emergency, the basic principle of maintaining an airway either orally or through the tracheostomy and calling for help holds true in all circumstances.

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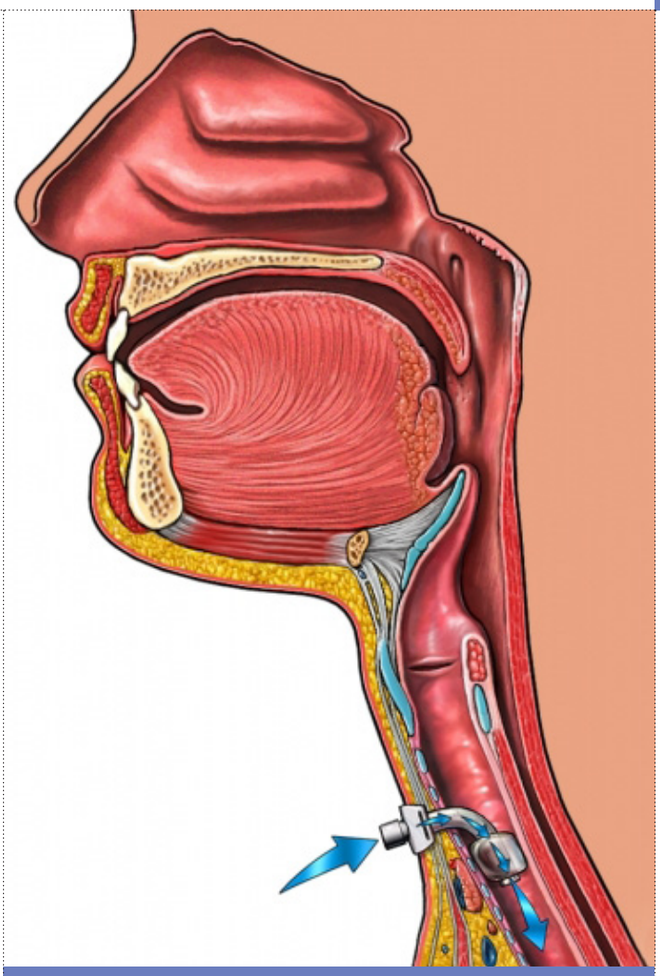


Tracheostomy is a common procedure performed in intensive care patients.
Practical Procedures.

PREOPERATIVE INVESTIGATIONS AND THE ROLE OF THE PRE-ASSESSMENT CLINIC

J Campbell and K Scott

Patients for elective surgery under anaesthetic will require a preoperative evaluation, which often includes investigations.
Good Clinical Care.

Abstract

Patients for elective surgery under anaesthetic will require a preoperative evaluation, which often includes investigations. These tests have several benefits: they can assess a known disease; help plan perioperative care; or discover an unknown condition in the hope that this will reduce perioperative morbidity. However, routine preoperative investigations rarely change a patient's management, and may have disadvantages. Literature now suggests that investigations should be indicated rather than routine. This article examines the most recent evidence and guidelines available regarding common preoperative investigations and their indications, and discusses the role of pre-assessment clinics in this area.

Introduction

A patient presenting for elective surgery under anaesthetic will require a preoperative evaluation, including at least a history and examination[1], but often incorporating investigations also. The investigations may be blood tests, a chest X-ray, invasive cardiac imaging, or all of these and more. These tests have several benefits: they can assess or quantify a known disease; they can help plan perioperative care; they could even identify an unknown condition that may affect perioperative care[1]. It is hoped that this will reduce morbidity and minimise a patient's incapacity[2].

For a Foundation doctor, a sound understanding of preoperative investigation is relevant to the job and the curriculum. The curriculum includes matters underlying this discussion, such as decision making, creating a management plan and communication. But the curriculum includes topics with a more obvious link, such as avoiding unnecessary investigations, and acting on results[3]. The choice and management of preoperative investigations is a good subject for workplace-based assessments or teaching.

The problem with preoperative investigations

The benefits of preoperative investigations have been listed above, but are there disadvantages? If more tests give more information about the patient, it seems logical that by performing more tests we will improve safety and outcomes for our patients. Yet in low-risk patients it has been demonstrated that preoperative investigations do not change mortality and morbidity[5]. Preoperative investigations rarely change a patient's perioperative management, potentially making tests futile[1]. The percentage of patients in which management is altered varies widely, and often there is no definition of a significant "change in management". If we believe that an investigation has been futile, we have wasted time and money, using up resources and slowing down the service we offer[4]. This causes delays and inconvenience for the patient and other staff[2], with associated costs. Also, these inappropriate tests may not all have normal results. Some will be false positives, and risks are associated with further tests or treatment for this initial result[2, 6], plus more delays and inconvenience. Patients have even undergone major surgery due to false positive results[6].

Over-investigation is also a medico-legal risk. This is because an abnormality not documented and/or investigated further is a greater liability than if the test was never performed[2, 6] – and large percentages of preoperative results are not documented[7, 6].

PREOPERATIVE INVESTIGATIONS AND THE ROLE OF THE PRE-ASSESSMENT CLINIC

J Campbell and K Scott

With all this in mind, we should aim to carry out appropriate, indicated tests. We cannot abandon all preoperative investigation because indicated tests provide information that is valuable, or even necessary before some cases. Under-investigation carries its own risks for the patient and staff. Therefore it is important to recognise when investigations are indicated preoperatively, and this article will aim to clarify that.

Guidelines

Over recent years preoperative investigation has moved away from "routine" tests performed for everyone, often ordered only because they were easily available[4]. However, ordering tests "as indicated" requires a consensus on the indication for each test. With a rationalised consensus, guidelines can be created which reduce the number of tests requested and reduce workload[4, 7]. However, it will require updating and may prevent the user thinking about the tests, so that the obvious may be overlooked[8].

Unfortunately evidence in the literature is lacking[1] and we have no widespread protocol to follow. The closest in the UK is the NICE (National Institute for Clinical Excellence) clinical guideline for preoperative tests[9]. This was published in 2003 and, when contacted (July 2009), NICE stated a review date is set for June 2010. In its simplest form this is a collection of tables. It combines the patient's age, ASA grading, co-morbidity and invasiveness of surgical procedure, to show tests as either recommended, not recommended or to be considered. The ASA (American Society of Anesthesiologists) grading is a loose scale to rate a patient's fitness for anaesthesia and is unofficially linked to a patient's functional capacity[9]. The official scheme has grades 1 to 6, but the NICE guideline uses only 1 to 3, as defined in Table 1[10]. Invasiveness of surgery is graded from minor to "major+" procedures, shown in Table 2[9]. Both of these scales are subjective, and this, along with the large number of tables, can make the guideline difficult to use.

ASA Grade	Definition	Example of disease
1	Normal healthy patient	None (i.e. "fit and well")
2	Patient with mild systemic disease	Well-controlled asthma
3	Patient with severe systemic disease	Poorly-controlled asthma

Table 1: NICE guidelines.

Grade of surgery	Invasive-ness	Example of procedure
Grade 1	Minor	Excision skin lesion; drainage breast abscess
Grade 2	Intermediate	Tonsillectomy; knee arthroscopy
Grade 3	Major	Total abdominal hysterectomy; thyroidectomy
Grade 4	Major+	Total joint replacement; colonic resection

Table 2: Invasiveness ranging from minor to major.

Apart from the NICE guideline, the most common protocols available are those formed locally, within a region or institution[4]. Local guidelines may have the advantage of including a policy for blood product ordering, or guidance regarding specific procedures (e.g. Liver Function Tests before biliary surgery)[11]. Lastly, a task force from the ASA have published advice in the form of a "Practice Advisory"[1], (i.e. a collective opinion of current evidence).

Types of preoperative investigation

Common investigations performed are shown below, in simple groups. This is not exhaustive, but most other investigations performed to assess a patient's fitness for surgery (and/or anaesthetic) fall into specialised cardiac or respiratory categories. Of course, other tests are used to assess the indication for surgery (e.g. ultrasound to confirm gallstones prior to cholecystectomy) but these are specific and not covered here.

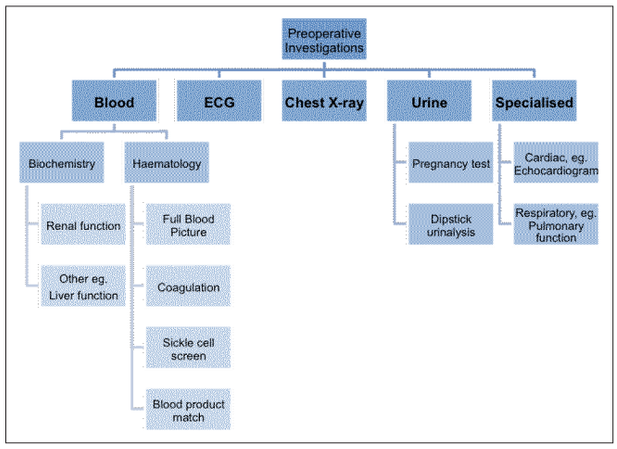


Figure 1: Types of preoperative investigations.

It is important to clarify some terms used here. Blood tests vary by region. We discuss renal function as including urea, creatinine and electrolytes, a full blood picture (hereafter FBP) as including haemoglobin and platelets, among others, and coagulation screen as including at least prothrombin time and activated partial thromboplastin time[3]. We use the abbreviation ECG for electrocardiogram.

As a way of discussing each type of preoperative investigation, below are cases that we hope bring out the relevant points. Investigations required in each case are based on the NICE guidelines. Please bear in mind that some surgical procedures will be sufficient indication for particular investigations, and that NICE guidelines have separate tables for cardiovascular and neurosurgery[9].

PREOPERATIVE INVESTIGATIONS AND THE ROLE OF THE PRE-ASSESSMENT CLINIC

J Campbell and K Scott

Case 1: Blood tests

A 44-year-old female presents for a total abdominal hysterectomy (due to fibroids), with a history of mild renal impairment due to childhood kidney disease.

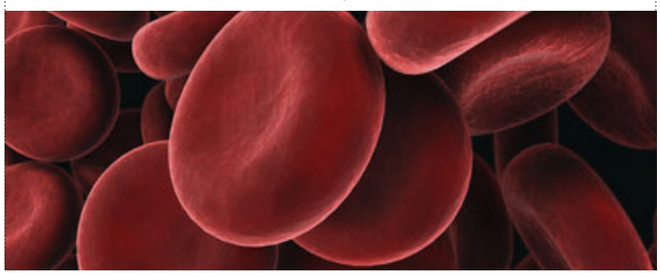
This patient should have blood tests for biochemistry and FBP. A coagulation screen may be considered (see below)[9]. Requests for cross-matching blood products are made by local policy and are not mentioned again.

Renal impairment may cause electrolyte abnormalities and/or difficulties in fluid management and these should be considered by the anaesthetic and surgical team. Her haemoglobin or platelets may be deranged, which may impact on her perioperative management.

Indicators in a patient's history to consider blood tests are listed in Table 3[1, 9], but it should be noted that although blood tests are often considered the most basic of investigations, in many cases they are not required[2]. This is especially so for patients without severe systemic disease, even if elderly[12]. NICE guidelines recommend serum biochemistry most commonly and, although often considered, NICE never recommend a coagulation screen[9], as this is less likely to alter management than other blood results and has been demonstrated not to predict perioperative bleeding[1, 13].

	Biochemistry	FBP	Coagulation
Clinical indicators	Renal disease	Renal disease	Renal disease
	Liver disease	Liver disease	Liver disease
	Cardiovascular disease	Cardiovascular disease	
	Endocrine disease	Respiratory disease	
		Anaemia	
		Haematological disease	
		History of bleeding	Bleeding disorder
	Drug History (e.g. diuretics)*		Drug history (e.g. warfarin)*
	Age	Age	
	Type of surgery	Type of surgery	Type of surgery

Table 3: Blood tests according to a patient's history.
*Remember to include alternative therapies.



Case 2: ECG

A 57-year-old man who smokes 20 cigarettes per day with a past medical history of hypertension (controlled with an ACE inhibitor) attends for a knee arthroscopy.

This patient has several risk factors for cardiovascular disease, and following NICE guidance should have a preoperative ECG[9]. NICE guidelines suggest any patient with cardiovascular disease (including hypertension) should have an ECG prior to surgery, no matter how minor the procedure.

This statement, however, is counterbalanced by more recent guidelines from the American College of Cardiology and American Heart Association (ACC/AHA)[14] in 2007 which state an ECG is not indicated in an asymptomatic patient before low-risk surgery. Their recommendations are summarised below (Table 4)[14]. There are several reasons for this controversial move away from preoperative ECGs. Among these are more accurate biochemical markers of myocardial damage, such as troponin T[19], making a "baseline ECG" less useful[17]. Despite the differences in this guideline, in this case we would still consider an ECG as the NICE guidance is applicable to UK practice, especially if it was part of a local hospital protocol.

	Surgery	Vascular Surgery
ECG recommended	Known cardiovascular disease, for ntermediate surgery	1 or more risk factors
ECG may be considered	1 or more risk factors, for intermediate surgery	Any patient
ECG not indicated	Asymptomatic patient for low-risk surgery	-

Table 4: NICE ECG guidelines.

In relation to this case, we also consider serum biochemistry to be useful due to possible effects of cardiovascular disease and/or his medication.

Case 3: Chest X-ray

A 63-year-old male presents for a total knee replacement. He has angina and uses his GTN spray about twice a week when walking. He lies on three pillows to sleep comfortably.

It is recommended that this patient has an ECG, FBP and biochemical profile[9], but he is also one of few patients for whom a preoperative chest X-ray is recommended. The chest X-ray was the first of the common preoperative tests to be questioned[6]. Studies suggest that most abnormalities on chest X-ray are predictable by chronic conditions (e.g. COPD)[17,18] and that of these abnormalities only evidence of congestive heart failure significantly changes outcome[18]. The usefulness of the X-ray is not its only limitation. It is expensive to perform, subjective to interpretation, abnormalities are easily missed and is not without radiation risk[4, 6].

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A preoperative chest X-ray may be considered in a patient with COPD, cardiovascular disease, recent upper respiratory tract infection and/or a smoker[1]. Age, despite arbitrary thresholds in many institutions, is not an indicator. In this case the history of orthopnoea and smoking makes it important to consider COPD or cardiac failure and the chest X-ray may be useful in making this distinction.

Case 4: Sickle cell screening

A 38-year-old male attends for excision of a large lipoma under general anaesthetic. He is fit and well. He is second generation Nigerian.

This patient would require no investigations other than considering a sickle cell screen[9]. Family history may add useful information. Patients with ethnic origins from North/West/South Africa or Afro-Caribbean are recommended to be tested, but other ethnic backgrounds should also be considered (e.g. Middle East, Asia)[9,19]. Approximately 10,000 people in the UK carry sickle cell disease[19].

Genetic testing shows either heterozygous "sickle trait" or homozygous sickle cell disease. Homozygous patients have a decreased life expectancy, are much more likely to require surgery and suffer more complications (including typical pain crises and pulmonary compromise)[19]. These are believed to be due to a vicious cycle of sickled cells causing vaso-occlusion, hypoxia and ischaemia, potentiating more sickling. Other theories exist, but management remains the same: these patients require adequate warmth, hydration and oxygenation to prevent sickling[20].

Another aspect is preoperative transfusion, to "dilute" or replace sickle cells. A Cochrane review in 2007 could not recommend transfusion (either top-up or exchange regimes) but stated that it may decrease morbidity and/or mortality[19].

Genetic testing requires counselling before and after testing. It is useful to ask permission to inform family members of results, and/or offer testing, depending on results[9].

Case 5: Pregnancy testing (and urinalysis)

A 21-year-old female presents for tonsillectomy. She is fit and well, however, her history allows the possibility of pregnancy.

Pregnancy testing is different from other investigations. Currently any other "routine" preoperative investigation is unlikely to change perioperative management, but a positive pregnancy test changes virtually all management for elective procedures[1].

It is estimated that surgery is performed during 1.5–2% of pregnancies, usually without consequence, and there is no absolute contraindication to surgery during pregnancy[21, 22]. The risks of the anaesthetic and surgery are not quantified. There are concerns of triggering labour in later pregnancy and of teratogenicity[21]. Also in the first trimester, up to 15% of known pregnancies miscarry, and the possibility of this being attributed to the procedure is a medico-legal risk. Therefore it is recommended that any surgery be postponed if possible[23].

A pregnancy test should be considered in all females of childbearing age and it has been suggested that pregnancy testing should be routine[9, 22]. A b-HCG test (urine or blood) will be positive as early as 8-days post-conception, when a patient will be unaware if she is pregnant[1, 6]. Consent for this test is of course important, with counselling available.

Other urine tests (i.e. urinalysis, microscopy and culture) are not felt to be indicated preoperatively except if a patient is having a prosthetic implant (higher risk of infection), a urology or urogynaecology procedure, or has symptoms of urinary tract infection[1, 6, 9].

Case 6: Other investigations

A 72-year-old lady attends for a sigmoid colectomy (for diverticular disease) with two previous myocardial infarcts (MI). She states that she had stents inserted after her last MI.

Other investigations (FBP, biochemistry, ECG and/or CXR) will be appropriate here, as discussed earlier. "Other" preoperative investigations fall mostly into either cardiac or respiratory tests. For either of these a specialist consultant opinion (i.e. cardiologist or respiratory) is often valuable. This consultant can best discern the risk from points in the history and may provide valuable advice or referral for specialist tests[24].

Specialised testing of cardiac status may include an echocardiogram (with or without dobutamine), exercise stress testing, angiography or radionucleotide imaging. Specialised respiratory investigations include pulmonary function tests and home pulse oximetry[1]. A preoperative arterial blood gas is never recommended in the NICE guidelines, but should be considered in patients with co-morbidities[9].

Case 7: Children

A 6 year old, who is otherwise fit and well, is admitted for an umbilical hernia repair (i.e. intermediate surgery).

It is now recognised that there are few indications for tests in children[6, 9]. This is reflected in the NICE guidance, in which even for "major+" procedures, no tests are recommended and only basic blood tests and urinalysis are even considered. This will be a relief to those who understand the difficulties and distress any investigations can cause for the child, parents and staff.

Who, where and when

Many systems now exist for the arrangement of preoperative investigations. For example, relevant tests may be performed at the outpatient clinic when booking for surgery. Many patients are still admitted to the ward the day before surgery to allow any tests to be performed. Recently, however, there has been an increase in Preoperative Assessment Clinics (abbreviated here to PAC)[24].

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The PAC takes various forms in different institutions, but usually the patient will attend a standalone appointment in order to optimise the patient for surgery when they are admitted. This usually includes most preoperative investigations required, but may also include consent, patient education and optimising medications[8, 25]. In the rapidly increasing field of day surgery, a PAC can be invaluable[26]. In some centres a telephone interview or postal questionnaire is used to filter out patients who do not need to attend. Anaesthetists, surgical juniors, other doctors, specifically trained nurses, or any combination of these may staff the PAC[4, 27].

Often a protocol is devised to standardise the tests performed. Otherwise, the number and type of investigations varies depending on who sees the patient. We audited preoperative investigations in 100 elective admissions for general surgery, ENT or gynaecology procedures in our district general hospital in early 2009 before the introduction of a PAC. We found patterns of investigation aligned to each specialty. Overall, in gynaecology most patients had a FBP and blood grouping carried out but few other tests; in ENT patients fewer than recommended tests were performed; and in general surgery tests were done that were unnecessary according to the NICE guideline. The latter is staffed by F1s and this follows literature suggesting that more junior doctors order more investigations[4, 7].

The growth in number of PACs demonstrates their benefits to patient satisfaction and hospital economics[8, 25]. Advantages and disadvantages of a PAC compared to ward admission on the day before surgery are shown in Table 5 [4, 8, 24, 25].

Advantages	Disadvantages
Reduced delays on day of surgery	Requires more patient time (inc. travel)
Allows admission on day of surgery, reducing bed pressures	Allows admission on day of surgery, increasing time pressure on arrival
Patient/staff less likely to cancel	Staff and start-up costs
Better adherence to protocols	Risk duplication of work (e.g. history, tests) on admission
Reduced investigations	Risk recalling for simple reasons (e.g. haemolysed blood sample)
Allows time to treat abnormalities	Risk results being out of date
Allows time for results to be seen	Blood product matching will be invalid
May centralise several specialties	Unlikely to see own anaesthetist/surgeon

Table 5: Advantages and disadvantages of PAC compared to ward admissions before surgery.

The age of results can be an issue. Investigation results from the time of PAC attendance may change before admission, but it is unlikely that previously normal results will become abnormal by the time of the surgery[6]. A survey of the American Society of Anesthesiologists showed that over 90% were content to use test results from patient records, mostly 3 months old or more, if the patient's history had not changed significantly[1].

Acting on results

There will be some results that require treatment (e.g. hyperkalaemia) but the most common issue around actioning results is communication.

It can be forgotten that the result is more important than doing the test. With reduced working hours it is increasingly likely that the person requesting a test will not see the result, but it is essential that this is handed over properly. This may involve only writing it in the notes. But an abnormal result may involve further action, such as communication with a senior colleague, an anaesthetist or another specialty for advice. If further action is needed (which may be as simple as a repeat test) then it should be clear who will do this and, if the patient's surgery is cancelled, then it should be clear who will inform the patient, surgeon, anaesthetist and theatre staff. On the other hand, a test performed with no consideration of the result is a medico-legal risk[2, 6].

Summary

To conclude, the current literature would suggest that preoperative investigations remain a useful part of preoperative evaluation, but there has certainly been a move away from "routine" investigations toward "indicated" investigations[1], and there is evidence that reducing the number of investigations does not compromise patient care[2]. The indications for tests carried out may be formulated in a local protocol or algorithm, although the nearest to this on a national scale in the UK is the NICE guidance[9]. Algorithms are difficult to produce because there is not enough evidence to set explicit parameters[1]. Therefore it is important that Foundation doctors are educated to rationalise the use of preoperative investigations appropriately[4, 7].

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ACUTE PAIN MANAGEMENT

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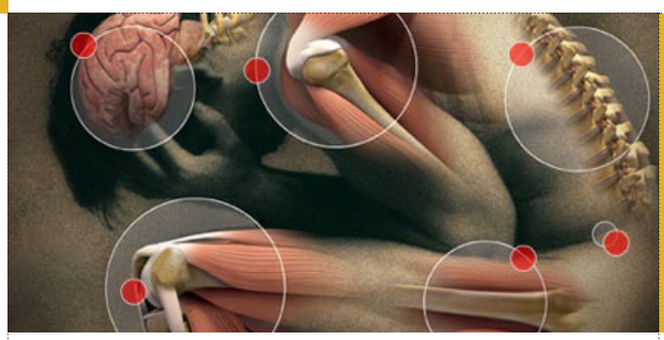

Pain is associated in the presentation of most commonly met medical and surgical conditions and therefore an understanding of how this complex condition can be dealt with is important.
Good Clinical Care.

Pain is associated in the presentation of most commonly met medical and surgical conditions and therefore an understanding of how this complex condition can be dealt with is important to allow delivery of good quality daily clinical practice. Advances in many areas of medicine and surgery lead to different approaches to established conditions. Pharmacogenetics is just such an area and may in the future help clinicians target analgesia appropriately and this is both a developing as well as an exciting field.

The International Association for the study of pain defines pain as – "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." This indicates the need to address the emotional content of the pain experience and be aware of the concept of "total" pain. Pain is not generic. There are many ways to subdivide what we know as pain; acute and chronic, nociceptive and neuropathic. These are differences that should be made in practice as their approach and management differs.

This article will concentrate on the pharmacological management of acute pain, for while complimentary interventions, such as acupuncture and TENS, can have a role, their use is often secondary to the immediately available tools at our general disposal on the wards and which Foundation doctors are most easily able to utilise.

Pain Definitions:

Acute: peaking rapidly and then receding over a short time.

Chronic: lasting 3 months longer than expected.

Nociceptive: pain originating from a nociceptor! Can be seen as "normal" or inflammatory pain.

Neuropathic: pain due to abnormality or disturbance of nerves; for example, nerve trauma, degenerative neurological diseases, metabolic, cancer, to name a few.

Assessment

There are several tools used in assessing pain severity. The most commonly used is the visual analogue scale (VAS), where scores of 0–3, 4–6, 7+ out of 10 are seen as mild, moderate or severe respectively. The VAS has been adapted for use in children and those with complex needs (see Figure 1).

Figure 1: Examples of Visual Analogue Scales.

Many find a verbal descriptor, such as mild, moderate or severe, an easier approach and while reflecting the VAS is perhaps more user-friendly.

Sedation scores are correctly being seen as a vital sign to record, and combined with the patient's respiratory rate will suggest any required modification to the patient's regime (see Table 1).

Sedation scores may be part of local early warning scoring systems for the deteriorating patient as well as for pain assessment:	
0	Awake
1	Drowsy
2	Rousable
3	Unrousable

Table 1: Sedation scores.

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Treatments

The WHO analgesic ladder has been in existence since 1986 and although originally designed for cancer pain management, it has been adopted almost universally as the standard approach for all pain treatments with drugs (see Figure 2). It is worth remembering that the "ladder" should be used as a guide to management, as many clinical situations may mean a move directly from the first level to the third level (e.g. pancreatitis, renal stones).

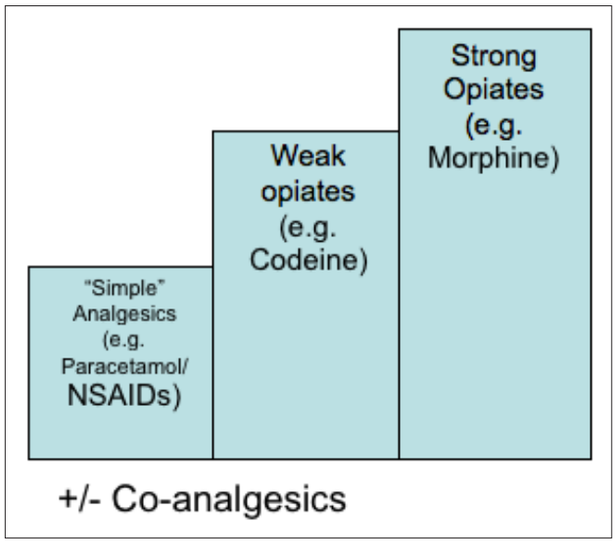

Figure 2: WHO Analgesic Ladder.

A positive clinical approach to managing acute pain is integral to optimising benefits. There is good research evidence that positive suggestion and expectation will facilitate any employed strategies to work optimally. The Foundation doctor should involve the patient in their pain management, explain the options and ensure the patient is aware that the doctor is confident that the pain can be controlled.

Drugs

Paracetamol

The exact mode of action of paracetamol (acetominophen) is unclear. COX3 activity, cannabinoid and 5-HT^3 receptors have all been suggested to play a role. Clinically, as a mild analgesic and antipyretic, regular administration should be encouraged for several reasons. There is an opioid sparing effect, it is part of a balanced analgesic approach and is cost-effective.

Routes of administration are oral, intravenous and rectal, and the dose for an adult is established in the UK as 1gm 6 hourly, for children a dose of around 20mg/kg given 6 hourly.

Significant side effects are rare although the catastrophic results of overdosage, especially liver failure, are well known. Caution is required in patients with liver disease or with a history of heavy alcohol intake.

NSAIDs and COX-2 inhibitors

The mode of action of these classes of drugs has been well researched and is via inhibition of both subtypes of COX enzymes – COX-1 and COX-2. By reducing cyclo-oxygenase (COX) enzyme activity and decreasing prostaglandins in the inflammatory reaction to injury, including surgery, a reduction in peripheral sensitisation of pain fibre endings can be achieved leading to a reduction in pain. Non-selective COX-1 inhibitors have been established in clinical practice for decades whereas the newer, more selective COX-2 inhibitors are more recent. Both enzymes are expressed and inducible in the human body and therefore while some side effects are reduced in COX-2 use compared to COX-1, others remain.

Effects on the gut are well recognised and can be problematical whichever route of administration is used. Renal effects are more covert but just as significant and can lead to acute renal failure requiring support if not recognised early. Creatinine clearance was shown in studies to be reduced by 16ml/min one day after surgery. If planning to use non-steroidals in older patients with pre-existing renal risk factors, close monitoring of renal function is mandatory, although most of the subtle changes, such as a reduction in creatinine clearance, are difficult to elicit on readily available e-GFR tests.

Use of non-steroidals can be beneficial in the first 24 hours in some clinical situations where, for example, straining due to constipation may adversely affect outcome, such as spine or neurosurgeries. There is up to a 50% pain reduction in acute management with a number needed to treat (NNT) figure of 2.2 and between a 30–50% reduction in opioid requirements, but their use should be considered in each individual patient and not prescribed as "routine".

Opioids

There are many opioids available for use in the UK. Morphine is reasonably seen as the gold standard against which all others are judged. There are many issues with the use of "strong" opioids that are raised in any clinical setting. As doctors, we should not be influenced by the almost superstitious beliefs held with regard to the correct and reasonable administration of this class of medication. Morphine exerts its action mainly at the mu receptor, which via G protein action, induces changes involving calcium and potassium ions. Morphine can be administered via practically any route, but most commonly by mouth or parenterally. The clinical state of your patient will influence which is the most appropriate route to use. In the acute setting, parenterally is generally the most common route.

The "correct" dose of morphine to use is impossible to define. When given via patient controlled devices (PCA), an available dose of 1mg IV every 5 minutes is usual. Where a PCA is not used, regular assessment of the patient's needs is required as the correlation between size, age and dose is unpredictable. Using locally agreed protocols for intramuscular or subcutaneous administration offers flexibility of total dose and open access depending on the patient's requirements.

Why bother?

- Multimodal approach often helps overall management and acknowledges different pain mechanisms.
- We feel happier when less "controlled" drugs are used!
- We should see opioid reduction as an endpoint in itself.

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Opioid side effects are well known, so informing patients of commonly met issues should be part of the prescribing procedure and including medication to mitigate such anticipated effects (particularly nausea and vomiting) should be routine.

Others medications

A popularly prescribed drug is nefopam. Others are gabapentin, amitryptiline, etc. In general terms it is easiest to see other adjuncts to the above drug classes as "the antis", i.e. antidepressants and antiepileptics. The main roles for these drugs are in neuropathic pain when the NNT figure is in the region of 3–4 for around 50% benefit. Their use in acute pain management is usually best taken under advisement from the local pain team.

Other approaches

Various regional anaesthetic techniques for surgery and post-operative pain control are used. These are not to prove how clever the anaesthetist is in blocking abstruse nerves! Patient comfort and outcomes are both positive endpoints in the use of regional anaesthesia.

Epidurals can reduce the risk of deep vein thrombosis, respiratory complications of opioids and are an important part of most colorectal procedures as they reduce opioid induced constipation and improve anastamosis integrity. Most epidural prescriptions consist of low concentrations of local anaesthetic and opioid, to give both axonal and neuronal blocking effects.

Normally, epidurals are prescribed by the anaesthetic team and managed by the acute pain team, but it is not uncommon to be asked to review a patient with an epidural in situ. Using a standard "ABC"-type assessment approach can ensure that other factors contributing to apparent pain problems are not missed (e.g. bleeding). The charts are generally self-explanatory and include sedation scores, sensory levels and general vital sign recordings. Changes that trigger an intervention are documented on the relevant prescription charts and may vary from unit to unit. Further assessments are best left to the pain team, having followed any immediate safety procedures usually indicated by local protocols on the epidural charts.

Clinical examples

I am called to see a post-operative knee replacement patient whose epidural is not working. It is 10pm and I see the epidural catheter tip has fallen out from the back of this 78-year-old man.

He is talking but obviously in pain and distressed with a heart rate of 90 and BP of 100/75. There is about 600mls of blood in the knee drain. What should I do?

Follow the basic ABC rules. A and B seem to be OK. You have already identified that there is probably significant blood loss and the vital signs suggest he might be marginally hypovolaemic.

If using the appropriate device, re-transfusing the drain blood and/or a fluid challenge would seem a good idea while specific pain treatment is being arranged.

At his age and with possible hypovolaemia, initial avoidance of NSAIDS would seem reasonable especially if there is a raised creatinine in the history. Ensuring paracetamol has been given regularly and then small boluses of strong opioid (morphine) with anti-emetic cover should be the next intervention. Getting control of the pain by using 2mg incremental doses intravenously every 3 minutes should then rapidly allow either subcutaneous or PCA morphine to be adequate.

At a convenient time, alert the pain team of the epidural failure so they can review the situation on their next round.

I have admitted a drug misuser (£50-a-day heroin) with probable femoral arterial embolus following groin injections of drugs. The patient is complaining bitterly of poor pain control and requesting further doses of morphine even before the first dose has been given. The leg is tense and very tender, especially below the knee, and foot pulses are only weakly identified on Doppler. It is late in the evening, the patient's complaints are disrupting the ward for other patients and the staff are being distracted from caring for other patients. What should I do?

This is a difficult situation as it involves distress in more than the single patient. Assessment of any obvious remediable causes for the marked pain needs to be rapidly performed.

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Two immediate possibilities are compartment syndrome and vascular occlusion directly related to the intravascular injections. Both are limb threatening urgencies and if suspected, urgent review by a surgical colleague is mandatory. Ischaemic pain is notoriously difficult to manage from whatever cause and almost invariably opioids are required. In a recreational opioid user, the responsiveness of the mu receptor is likely to be reduced, therefore if morphine is not acting as well as anticipated with reasonable doses (often seemingly very large in comparison to opioid naive patients) then a trial of oxycodone may be considered. This is approximately twice the potency of morphine and has anti-neuropathic efficacy as well as acting on kappa and mu receptors. These problems require multidisciplinary input and early consultation with the local pain team should be seen as an essential part of your treatment plan. Remember to consult early with your acute and/or chronic pain teams for advice.

My consultant has asked for a 60-year-old man with COPD and lung cancer to be admitted for treatment of a chest infection. The patient's notes show there to be metastases in his liver, two ribs and acetabulum on the left. His biochemistry shows moderately raised liver function tests.

He is talking to me and breathing rapidly, being in pain from the diseased ribs and finding it almost impossible to cough. Movement is difficult due to hip pain. He is taking co-dydramol for the pain. What should I do next?

The use of opioids in pre-existing chest disease is not contraindicated! Of course caution needs to be taken, but in this situation without the ability to clear chest secretions due to pain, this patient will be put at more risk than appropriate opioid use will cause.

Liver function is often used as a reason why perceived strong opioids cannot be used; this too is a fallacy as until end stage liver failure is reached, metabolism of opioids will continue, albeit at a reduced rate.

Therefore, intravenous morphine to control the pain and allow sputum clearance, despite a possible reduced respiratory rate, is sensible to try and prevent the high risk of deterioration of lung function during this acute episode. Non-steroidal or COX-2 inhibitors are often found to be useful with bone pain from metastases and as cancer pain can have elements of Nociception, inflammatory and neuropathic pain mechanisms, a trial of an anti-neuropathic agent (e.g. gabapentin) may prove a useful adjunct to the opioid if it proves difficult in obtaining comfort for the patient.

Involvement of the pain team and/or palliative care services would be appropriate especially as the prognosis in a patient with this history sounds guarded.

Conclusions

This article has been an overview of the use of commonly prescribed acute pain medications in hospital and their place within a multidisciplinary team approach to patient care. Foundation doctors have a role to play both in managing acute pain and in assessing patients and situations where specialist input is required.

Truths

- Annually more deaths are attributed to NSAIDs than opioids in clinical practice.
- Co-morbidities are precautions NOT contra-indications.

Myths

- Old patients need less analgesia – they may use less opioid following immediate titration.
- Babies do not need analgesics – distress is distress at whatever age.
- COPD means no strong opioid – patients all need to be comfortable enough to clear their chest.
- Decreased renal function means no opioids – this is a caution, not contra-indication. Some opioids are more "renal friendly".
- Morphine is locked up so it must be dangerous – so is strong potassium which is more immediately dangerous!
- High BMI = need for more analgesia – BMI is only one factor, consider the whole picture of clinical situation.
- Drug misusers are after more morphine – immediate care is not the best time to be judgemental.
- Pain will respond to morphine – you just need a bigger dose.

Is opioid sparing relevant?

- NSAIDS reduce morphine requirements by up to 50%.
- Paracetamol reduces morphine dose by 7–10mg.
- Side effects remain the same!
- Pruritis, retention of urine, nausea, sedation and most oddly – respiratory depression.

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AUDIT, RESEARCH, SERVICE REVIEW AND CLINICAL GOVERNANCE: WHAT IS THE DIFFERENCE AND WHY DOES IT MATTER?

Sughrat Siddiqui

The following article will seek to clarify the differences between clinical audit, service review, clinical governance and research. Teaching & Training.

Introduction

The following article will seek to clarify the differences between clinical audit, service review, clinical governance and research. These are important processes in the improvement of patient care that are often poorly understood, not only in the sense that the differences between each are sometimes unclear, but the actual importance of each, not only to your own career progression within the Foundation programme, but also to the delivery of excellent clinical care to the patient, is sometimes forgotten.

This article will define each process, give examples and, where appropriate, give a direct comparison to highlight differences and similarities. The purpose of this is to clarify potential areas of confusion and allow you to use your time more efficiently when planning to perform an audit or service review in your Foundation programme.

Clinical audit

Clinical audit is about improving clinical services. One definition that is used is[1]: "Clinical audit is a quality improvement process that seeks to improve patient care and outcomes through systematic review of care against explicit criteria and the implementation of change."

Clinical audit is a continuous process and re-audit aims to measure care against criteria and assess the impact of the implementation of change. Audit asks the following questions[2]:

- Is what is happening what should be happening?
- Are we doing what the evidence says we should be doing?
- Does practice meet the standards that have been set?
- Does practice follow published guidelines?
- Is knowledge or best evidence being applied in practice?

Clinical audit was introduced by Florence Nightingale during the Crimean War (1853–1855)[3]. Sidney Herbert, the British Secretary for War, asked Florence Nightingale, a mathematician, to become nursing administrator and oversee the introduction of nurses to military hospitals[4]. Nightingale, at the end of the war, was able to show positive outcomes from quality of care when few other clinicians at this time used audit.

When Nightingale arrived at Scutari medical barracks in 1854, she was appalled by the unhygienic conditions and high mortality rates among injured and ill soldiers. Nightingale's knowledge of mathematics and statistics were evident as she collected data enabling her to calculate mortality rates. By February 1855 mortality rates had dropped from 60% to 42.7% due to an improvement in hygiene. By spring that rate had dramatically decreased to 2.2% through the provision of fresh water, the purchase of fresh fruit, vegetables and standard hospital equipment (purchased from her personal funds).

In 1912 another famous advocate for clinical audit, Ernest Codman (1869–1940) introduced his "end of plan idea". US physician, Codman, established a plan to enable the tracking of patient treatment outcomes as a way to identify clinical misadventures. He intended it to serve as the foundation stone for improving future patient care in the way that audit, risk management and critical incident reporting are used presently to improve patient care.

What makes a good audit?

As a Foundation trainee you will become involved in audit, both in attending departmental/specialty audits and in helping to perform an audit project of your own. It is worth thinking about audits you have seen and considering the factors common to good audit.

A good audit has:

- Good topic selection. (Does it examine the area which needs to be examined? For example, high-risk care, high volume procedures, new drugs, etc.).
- The standards which are set relate to local or national standards.
- Multi-professional involvement.
- Analysis of the collected data takes place and is compared to set standards.
- Changes are specific, measurable, achievable, realistic and time bound (i.e. SMART goals).
- Changes DO take place.
- The change benefit patients/clients/carers.
- Re-audit of any changes happens to enable comparisons with previous results.

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Conversely, a poor audit can be considered to have:

- Topics chosen that are not relevant (i.e. no benefit to patients or the service) seeming to "tick a box" for no obvious purpose.
- Topics no one wants to do because the topic has been imposed on them rather than the investigator having an involvement or interest in that area.
- Standards that are non-existent, or are either too high or too low.
- Poor methodology.
- Poor data collection.
- Sample sizes which are too large or too small.
- The analysis has not been thought through.
- No change takes place after the audit.

Five stages of audit

When considering undertaking an audit the following steps will be helpful in ensuring that your audit is more likely to be a successful one!

Stage 1: Preparing for the audit

The reason for undertaking the audit may arise from a problem that you might identify from everyday practice, coroner's cases or national practice that people know or feel local practice could be potentially improved upon. It is now often a mandatory requirement for all audits to be registered with the Clinical Effectiveness and Evaluation Department of your Trust, so you will need to make yourself familiar with the registration forms. Registration of audits supports a Trust's Clinical Audit Annual Report.

Stage 2: Selecting criteria

The criterion should be written as a statement, for example:

- All patients requesting an urgent appointment will be seen that day.
- All patients with epilepsy should be seen at least once a year.
- All patients on Phenytoin should have their levels within the recommended limits.

Criteria can be defined from recent medical literature or the best experience of clinical practice.

A standard should be defined in order to make it useful. It should describe the level of care to be achieved for any particular criteria, such as:

- 98% of patients requesting urgent appointments will be seen the same day; or
- 90% of patients with epilepsy should be seen at least once a year.

The level of standard can often be controversial but there are basically three options: A minimum standard – the lowest acceptable performance standard. This can be used to distinguish between acceptable and unacceptable practice.

2. An ideal standard – the care that should be given under ideal conditions and with no constraints. This however can be unattainable for a prolonged period.

3. An optimum standard lies between the minimum and the ideal. Setting these standards requires judgement, discussion and consensus with other members of the audit team. Optimum standards represent the standard of care most likely to be achieved under normal conditions of practice and are often the ones most likely to be chosen for an audit.

Stage 3: Measuring performance

Following data analysis, areas falling below the predetermined standards can be identified, with performance either falling above, below or staying similar to the identified standards.

Stage 4: Making improvements

From the final report's recommendations, key recommendations should be arranged into an action plan and given to the appropriate stakeholders, such as members of the multidisciplinary team, Clinical Directors, Professional Managers, etc., for implementation. You should always aim to present your audit results to some key stakeholders (e.g. at a departmental meeting).

Stage 5: Sustaining improvement and re-audit

Without re-auditing it is impossible to see if any implemented recommendations have lead to an improved level of care. The audit cycle gives a clear checklist of the changes that are required before undertaking a successful re-audit project. The three main areas are: creating a climate for change; engaging and enabling the whole organisation; and finally, implementing and sustaining change[2].

Audit or research?

Audit and research have much in common; they share a rigorous approach to methodology in terms of design, procedure, analysis and interpretation of data. At times, the distinction between research and audit can seem blurred. However, there are a number of major differences and the table below provides some assistance in determining what is audit and what is research[2].

Clinical audit: is a way of finding out whether you are doing what you should be doing by asking if you are following guidelines and applying best practice.

Research: evaluates practice or compares alternative practices, with the purpose of contributing to a body of knowledge by asking what you should be doing.

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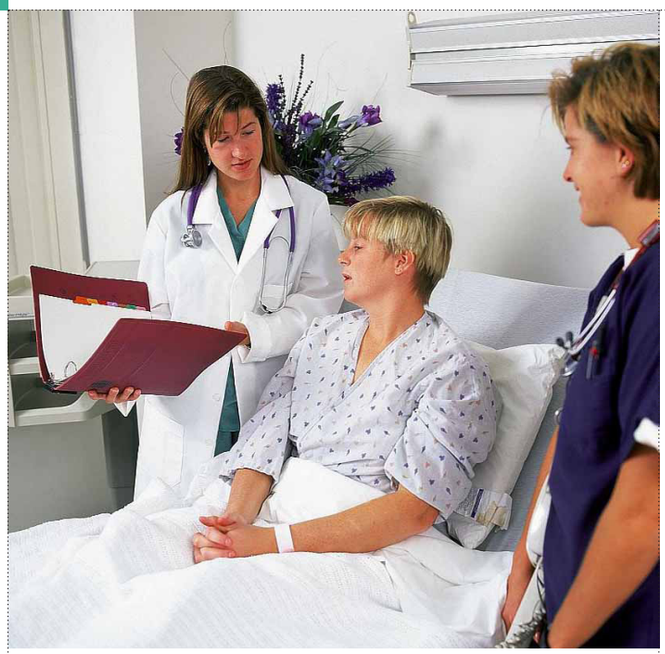

Audit Research

Audit	Research
Never involves experiments, whether on healthy volunteers or patients as volunteers	May involve experiments on human subjects whether patients, patients as volunteers or healthy volunteers
Is a systematic approach to the peer review of medical care in order to identify opportunities for improvement and to provide a mechanism for bringing them about	Is a systematic investigation which aims to increase the sum of knowledge
Never involves allocating patients randomly to different treatment groups	May involve allocating patients randomly to different treatment groups
Never involves a completely new treatment	May involve a completely new treatment
Never involves a disturbance to the patients beyond that required for normal clinical management	May involve extra disturbance or work beyond that required for normal clinical management
May involve patients with the same problem being given different treatments, but only after full discussion of the known advantages and disadvantages of each treatment. The patients are allowed to choose freely which treatment they get	May involve the application of strict selection criteria to patients with the same problem before they are entered into the research study

Research

If research is defined as "the attempt to derive generaliseable new knowledge by addressing clearly defined questions with systematic and rigorous methods"[5] it is another word for "enquiry". It is undertaken to discover facts or relationships and reach conclusions using scientifically sound methods.

Medical research can be divided into two general categories: the evaluation of new treatments for both safety and efficacy in what are termed clinical trials; and all other research that contributes to the development of new treatments. The latter is termed preclinical research if its goal is to elaborate knowledge specifically for the development of new therapeutic strategies.

Preclinical research

Preclinical research is research in basic science, which precedes the clinical trials and is almost purely based on theory and usually involves tissue or animal experiments.

New treatments come about as a result of other, earlier discoveries that are often unconnected to each other, and in various fields. Sometimes the research is done for non-medical purposes, and only by accident contributes to the field of medicine, for example, the discovery of penicillin. Clinicians use these discoveries to create a treatment regimen, which is then tested in clinical trials.

Clinical trials

A clinical trial is a comparison test of a medication or other medical treatment, versus a placebo, other medications and devices, or the standard medical treatment for a patient's condition. Clinical trials vary greatly in size: from a single researcher in one hospital or clinic to an international multicenter trial with several hundred participating researchers on several continents. The number of patients tested can range from as few as a dozen to several thousands.

Funding

Research funding in many countries comes from research bodies which distribute money for equipment and salaries. In the UK, funding bodies, such as the Medical Research Council and the Wellcome Trust, derive their assets from UK tax payers, and distribute this to institutions in a competitive manner.

Regulations and guidelines

Medical research is highly regulated. National regulatory authorities oversee and monitor medical research, such as for the development of new drugs. In the USA, the Food and Drug Administration oversees new drug development; in Europe, the European Medicines Agency; and in Japan, the Ministry of Health, Labour and Welfare. The World Medical Association develops the ethical standards for the medical profession, involved in medical research. The International Conference on Harmonisation of Technical Requirements for Registration of Pharmaceuticals for Human Use (ICH) works on the creation of rules and guidelines for the development of new medications, such as the guidelines for Good Clinical Practice (GCP). All ideas of regulation are based on a country's ethical standards code. This is why investigation and treatment of a particular disease in one country may not be allowed, but is in another (e.g. there are different rules governing stem cell research in the USA from that in Europe).

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Service evaluation

"A set of procedures to judge a service's merit by providing a systematic assessment of its aims, objectives, activities, outputs, outcomes and costs" (NHS Executive 1997). Service Evaluation may include audit and/or research and tends to use very similar data collection methods to research and audit.

Sometimes the results of the service evaluation are intended for use to influence services or practices outside the population or setting where the evaluation is based. If this is the case, the evaluation should be managed within the Research Governance Framework (i.e. go through peer review and ethical processes).

Service evaluation is undertaken to benefit those who use a particular service and is designed and conducted solely to define or judge current service. Your participants will normally be those who use the service or deliver it. It involves an intervention where there is no change to the standard service being delivered (e.g. no randomisation of service users into different groups). This does not require ethical approval although in many hospitals it is required to register service evaluations as you would an audit.

It is possible to use data collected from participants during a service evaluation for later research as long as:

- the data is completely anonymous;
- it is not possible to identify participants from any resulting report;
- use of the data will not cause substantial damage and distress.

Clinical governance

Clinical governance is the term used to describe a systematic approach to maintaining and improving the quality of patient care within a health system. The term became widely used in health care after 1995 when an anaesthetist, Dr Stephen Bolsin, exposed the high mortality rate for paediatric cardiac surgery at the Bristol Royal Infirmary. It was originally elaborated within the United Kingdom National Health Service, and its most widely cited formal definition describes it as[6]:

"A framework through which NHS organisations are accountable for continually improving the quality of their services and safeguarding high standards of care by creating an environment in which excellence in clinical care will flourish."

This definition is intended to embody three key attributes: recognisably high standards of care; transparent responsibility and accountability for those standards; and a constant dynamic of improvement.

"Clinical governance" does not mandate any particular structure, system or process for maintaining and improving the quality of care, except that designated responsibility for clinical governance must exist at Trust Board level, and that each Trust must prepare an Annual Review of Clinical Governance to report on quality of care and its maintenance. Beyond that, the Trust and its various clinical departments are obliged to interpret the principle of clinical governance into locally appropriate structures, processes, roles and responsibilities.

Clinical governance is composed of at least the following elements:

- education and training;
- clinical audit;
- clinical effectiveness;
- research and development;
- openness;
- risk management.

It is clear therefore that audit and service review have important roles in the system of clinical governance, and that all clinical research must be subject to governance procedures.

Conclusion

For all postgraduate medical trainees, audit and service review is mandatory and being able to differentiate one from the other, and both from research, is essential to protect patients, safeguard our professional standards and not waste your own precious time. Pursuing audit that is in fact research or seeking ethical approval for an audit that is in fact a service review would be hugely frustrating and could be potentially professionally difficult, which is why a clear understanding of the differences will hopefully lead you all to enjoy the processes while understanding the importance of why we are doing it. Good luck!

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THE FOUNDATION TRAINEE'S GUIDE TO ASSESSING AND RESUSCITATING THE OBSTETRIC PATIENT

Andrea Hards

Obstetric anaesthesia is a sub-speciality where learning points are not just specific to anaesthesia trainees.
Good Clinical Care.

Obstetric is a sub-speciality where many learning points may seem specific to anaesthesia trainees. However, pregnant patients are seen in many branches of medicine, especially general practice and the emergency department, and the "anaesthetic approach" to an obstetric patient can be very useful. It is important for the Foundation trainee to know how to deal with an acutely ill pregnant patient and this article aims to help you to do that.

What are the key differences in pregnant patients?

These are due to the presence of an additional patient (the foetus) and the numerous physiological changes that occur in pregnancy[1-3]. The best way to consider them is by using a systems-based approach. The key issues that result and can cause problems, or confuse assessment, are highlighted.

Cardiovascular

- Cardiac output increased by 50% by the end of the second trimester. This is a combination of a 30% increase in stroke volume and a 25% increase in heart rate.
- Systemic and pulmonary vascular resistance reduced by up to 35%.
- Diastolic blood pressure fall by around 25%, with the systolic blood pressure minimally affected. Both values then return to normal near term.
- From around 13-weeks gestation, the gravid uterus compresses the inferior vena cava and aorta.
- Reduced cardiac output due to decreased venous return in the supine position; this can be enough to cause patients to feel sick, faint and sweaty.
- Lower extremity and uterine artery hypotension (the latter with potential foetal distress).

Respiratory

- Capillary engorgement of nasopharyngeal and laryngeal mucosa can occur.
- Increased risk of hypoxaemia, especially in the supine position and as the pregnancy progresses.
- Functional residual capacity is reduced by 30% from the upwards displacement of the diaphragm by the gravid uterus.
- Oxygen consumption is increased by as much as 60% due to foetal, uterine and placental metabolic demands.
- Increased risk of airway bleeding and the difficult airway scenario.
- Increased minute ventilation increases because of a rise in tidal volume and, to a lesser extent, respiratory rate.
- $PaCO_2$ reduced to 3.7–4.2kPa, serum bicarbonate concentration falls to 18–21mmol/L.
- NB Dyspnoea with no cardio-respiratory disease is common.

Gastrointestinal

- Reduced lower oesophageal sphincter pressure due to progesterone effects.
- Increased intra-gastric pressure from the gravid uterus.
- Reduced "barrier pressure".
- Delayed gastric emptying in labour though probably normal at other times.
- Very high incidence of gastro-oesophageal reflux.
- Increased risk of aspiration and subsequent pneumonitis, probably from around 16–20-weeks gestation.

Renal

- Increased renal plasma flow.
- Increased glomerular filtration rate.
- Increased sodium and water retention.
- Increased urinary protein losses.
- Decreased plasma concentrations of urea and creatinine decrease.
- Glycosuria and proteinuria can occur in the absence of disease.
- Oedema, especially in the presence of other disease (e.g. pre-eclampsia) can result in a difficult airway.

Haematological

- Increased plasma volume of 50%.
- Increased red cell volume of 30%.
- Consequent "physiological anaemia of pregnancy" with haemoglobin drop of 15%.
- Increaesed WBC to around 9–11 x 10^9/L.
- Increased platelet turnover, clotting and fibrinolysis.
- Increased circulating volume means that a pregnant woman may lose 1200–1500ml blood before showing any signs of hypovolaemia.
- Pro-coagulopathic state (i.e. prone to thromboembolism).

So how should you assess an acutely ill pregnant patient?

This will hopefully be very familiar to you: it is an A,B,C,D,E approach with early senior help. In many cases you will have time to elicit a reasonable history, even if it is simultaneous with your examination.

Also do not forget that illness in the pregnant patient will involve a multidisciplinary team with potential input from obstetrics, anaesthetics and paediatrics as well as the parent specialty.

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It is important to remember that the mother is your first priority in this situation and anything you can do to improve her well-being will also improve things for her baby.

Pregnant women can present with non-obstetric illness and trauma but the following gives an overview of some of the relevant diseases of pregnancy.

Airway

Pregnant patients can be more prone to airway difficulties (e.g. because of the increased size of nasopharyngeal tissues or oedema related to pre-eclampsia). However, the most likely causes of airway problems are still a depressed level of consciousness, trauma or burns.

Your first approach should be to talk to the patient: **an appropriate response indicates a patent airway (as well as stable enough breathing to allow phonation and adequate cerebral perfusion).** You should then adopt the "look, listen, feel" system of assessment:

- ***Look for obvious agitation, drowsiness, cyanosis.***
- ***Listen*** for snoring, gurgling, hoarseness or the silence of complete obstruction.
- ***Feel*** for expired air and tracheal position.

If the airway is obstructed, try the simple measures of head tilt, chin lift and jaw thrust, remembering that simple adjuncts (e.g. Guedel airway) may be necessary. Do not forget that in trauma cases your airway assessment should include cervical spine control and if you need to open the airway, only use jaw thrust.

Because of the increased aspiration risk in pregnant patients, endotracheal intubation should be considered at an earlier time than in the non-obstetric patient, and this should be performed ideally by an anaesthetist as there is a higher incidence of difficult intubation in the pregnant patient.

Breathing

You should administer high flow oxygen as you assess breathing in an acutely unwell pregnant patient. Then follow a systematic approach to examination:

- ***Inspect*** for obvious injury, asymmetry, abnormal breathing pattern, accessory muscle use.
- ***Palpate*** altered chest expansion or mediastinal shift.
- ***Percuss*** for hyper-resonant or dull notes to aid diagnosis.
- ***Auscultate*** including the posterior chest if possible and in the axillae.

Supplementary monitoring includes oxygen saturation and respiratory rate and it may be appropriate to check arterial blood gases (ABG) or a peak flow measurement depending on the situation.

Remember from the physiology review that pregnant women can develop hypoxia more quickly, particularly if they are supine, and that normal ABGs are a little different in pregnancy.

Can you request a radiological investigation, such as a chest x-ray?

The answer to this is "yes", if it will help with/alter your diagnosis and management. There is much less foetal radiation exposure from distant sites (e.g. chest) and the amount of radiation used has reduced over recent years.

The Royal College of Radiologists states[4]:

"If pregnancy is established, or likely, review the justification for the proposed examination, and decide whether to defer the investigation until after delivery, bearing in mind that a procedure of clinical benefit to the mother may also be of indirect benefit to her unborn child and that delaying an essential procedure until later in pregnancy may present a greater risk to the foetus. If a procedure is undertaken, the foetal dose should be kept to a minimum consistent with the diagnostic purpose."

It is worth noting that the **diagnosis of pulmonary embolism should not be missed in a pregnant woman.** The Confidential Enquiry into Maternal And Child Health (CEMACH) collects data from England and Wales for all cases of maternal death and publishes every 3 years. Thromboembolism remains the most common cause of direct death with a total of 41 cases for the period 2003–2005[5].

Circulation

The most common circulatory problem seen in pregnancy is hypovolaemia from haemorrhage: it was the fourth most common direct cause of maternal death in the 2003–2005 CEMACH report. Unless you are working in obstetrics, you are most likely to see an ante-partum haemorrhage (e.g. placenta praevia or an abruption). It is important not to forget, though, that trauma happens to pregnant women too – accidental or not – and they may present with sepsis/anaphylaxis/neurogenic shock although this is much less common.

We have already seen that in pregnancy heart rate increases, blood pressure falls and blood volume increases. This means that:

- Pregnant women may only show the classical signs of hypovolaemia after loss of 1200–1500ml of blood (i.e. when they are already at a critical stage)[6].

Your assessment should include **pulse (rate, rhythm, volume), blood pressure, capillary refill, mental state and urine output.** Always look for any overt signs of blood loss and then consider more occult ones. It is also important to remember that because of the aorto-caval compression we have discussed:

- Anyone visibly pregnant and unwell should be tilted onto her left side as soon as possible to avoid supine hypotension.

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Management otherwise is standard, obtain large bore intravenous access, preferably 2 cannulae, and take bloods, including a full blood count, clotting profile and group and save or cross-match depending on the situation. There is endless debate over the choice of intravenous fluid to give in the first instance, although a fairly standard approach is to give up to 2 litres of Hartmann's solution, followed by plasma expanders until blood is available. If there is a life-threatening haemorrhage, you should transfuse O-negative or type-specific blood pending a full cross-match.

Disability

Your initial assessment tools here are the Glasgow Coma Score along with pupillary size and reaction. In more stable and appropriate cases you should perform full peripheral and/or central nervous system examinations.

Some of the problems you may encounter in pregnant patients are listed below (although this is by no means exhaustive and falling back on the "surgical sieve" in combination with relevant history is essential).

Headache is common and differentiating sinister causes, for example the onset of pre-eclampsia, from more benign ones can be notoriously difficult.

Consider:

- tension, stress, fatigue
- head trauma
- intracerebral bleed
- cortical vein thrombosis
- infection (e.g. meningitis, encephalitis, sinusitis)
- migraine
- pre-eclampsia
- electrolyte imbalance/hypoglycaemia.

Fitting and/or a depressed level of consciousness can be due to any of the "usual" medical causes but a concern is always that the diagnosis may be eclampsia: hypertensive disease of pregnancy was the second most common cause of direct maternal death in the CEMACH report between 2003–2005 with 14 cases reported.

Pre-eclampsia is now defined as hypertension and proteinuria developing after 20-weeks gestation in a previously normotensive and non-proteinuric woman[3]. Oedema is now omitted from the definition although it is often present.

- It is possible to develop non-proteinuric pre-eclampsia and also to have eclamptic seizures with minimal or even no hypertension.

- Although the disease can only be terminated by delivery, pre-eclampsia and eclampsia may both present only afterwards.

- The specific drug for fitting due to eclampsia is magnesium sulphate with a loading dose of 4g over 20 minutes. You must also treat the hypertension if SBP >160mmHg and/or DBP >105mmHg, usually with labetalol as the first line drug[6].

The most likely "D" investigation you will need to arrange is a head CT scan of the head, following local radiology guidelines and those discussed earlier. Do not forget that the CT scanner is classed as a remote site, usually with limited space and equipment, and patients must only be transferred there once they are stable.

Exposure and environmental control[6]

Once you have progressed through your ABCD assessments and treated/reassessed as appropriate, you need to adequately expose the patient to complete a full assessment.

Abdominal problems may well be encountered in your "C" assessment but the most common complaint seen in this group of patients is abdominal pain.

Take a detailed history wherever possible: the acute onset of abdominal pain suggests rupturing or tearing and may include a ruptured ectopic, uterus, abscess or aneurysm or the perforation of an ulcer. **Acute abruption also presents with severe abdominal pain and should be the presumptive diagnosis until it is ruled out.**

Also do not forget that **abdominal injuries in pregnancy are on the increase,** both from accidental and non-accidental causes. In the first trimester, the uterus is relatively well protected from injury due to its thick wall and position within the bony pelvis. As the pregnancy progresses though, it provides some protection to the abdominal contents but the uterus becomes thinner and more vulnerable, with particular risk of abruption due to an elastic uterus and inelastic placenta.

Once you have assessed ABCDE and treated appropriately, then you need to consider foetal monitoring (e.g. ultrasound, foetal heart monitoring, cardiotocograph). It is vital to stress again that improving the mother's physiology will improve her baby's too. If a secondary survey is needed, it is done after assessment of foetal well-being and viability (which may include the decision to deliver).

Worst-case scenario: what if a pregnant patient arrests?

The first thing to say is that the chances of you being involved with this are very small! Resuscitation guidelines for pregnancy are based largely on case series and scientific rationale and management follows the standard Advanced Life Support algorithms. Here to help you remember are the key points that are slightly different[7].

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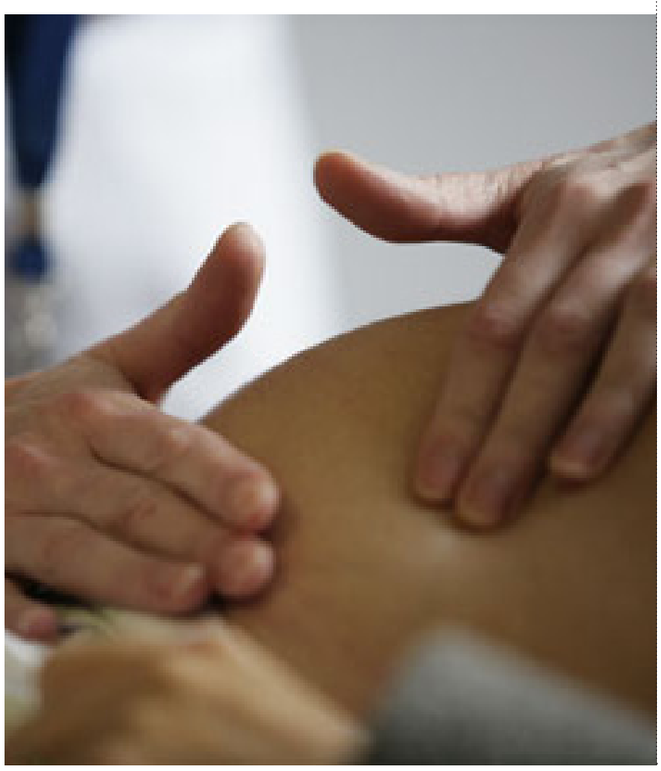
• You must tilt the patient or wedge them onto their left side to minimise aorto-caval compression.
• Hand position for chest compressions may need to be slightly higher.
• You should consider early intubation with cricoid pressure in view of the increased risk of aspiration.
• The Resuscitation Council (UK) has recommended that prompt Caesarean delivery should be considered as a **resuscitative procedure** for cardiac arrest in near term pregnancy (i.e. it improves the chances of maternal resuscitation). It should be begin within 4 minutes and the infant should be delivered by 5 minutes.

Summary

Pregnant patients can present to a variety of specialities with the same problems as the non-pregnant population. Assessment should be a systematic ABCDE approach as for anyone but with a few specific points to remember based on the physiological changes of pregnancy, the key ones are:

• The mother is the first priority.
• Remember the potential for rapid desaturation.
• Apply left lateral tilt to anyone visibly pregnant and unwell.
• A mother with classical signs of hypovolaemia is presenting late and has lost a substantial amount of circulating volume.
• Always bear in mind some of the specific diseases of pregnancy (e.g. eclampsia and placental abruption) in your differential diagnosis.

And most importantly:
Seek senior advice and get multidisciplinary involvement early.



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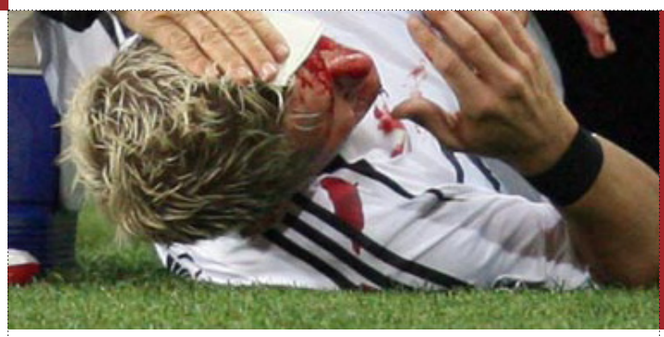
MANAGEMENT OF HEAD INJURY

Karl Brennan and Daniel Stevens



The assessment and management of head injury patients can be a daunting task for Foundation year doctors.
Patient Management.

The assessment and management of head injury patients can be a daunting task for Foundation year doctors. This article emphasises the importance of accurate history taking and examination when assessing these patients and will discuss management of a difficult, yet common scenario.

A 37-year-old plumber is brought to A&E following an alleged assault. He had been drinking all day with friends and was involved in an altercation with another man. He was hit across the head, possibly by a bottle, and fell to the ground. An ambulance was called.

On arrival in A&E, he is placed in a cubicle to allow him to sober up until he is reviewed by a doctor.

What are the principles in assessing this patient?

The patient should be seen and triaged by a suitably trained professional within 15 minutes of arrival. If the patient's Glasgow Coma Scale (GCS) is <15 (see Table 1) he should be seen immediately[1].

The aim is to establish the severity of any head injury sustained and the existence of any other associated injuries. Therefore, every patient should be approached using the ABCDE system of assessment. In the presence of excess alcohol or head injury, or both, the airway can be compromised. This is a medical emergency and requires anaesthetic support immediately. Furthermore, cervical spine injury is common, occurring in 2 to 6% of cases of serious head injury[2]. Full cervical spine immobilisation is required if any of the following are present[1]:

- GCS <15 on initial assessment;
- neck pain or tenderness;
- focal neurological deficit;
- paraesthesia in the extremities;
- any other clinical suspicion of cervical spine injury.

The primary survey performed using the ABCDE system ensures that the most life-threatening injuries are treated first, such as massive haemorrhage, prior to focusing on the head injury. Assessing disability using the GCS is a reliable and objective method of measuring the severity of the neurological insult.

Eye opening	Spontaneous	4
	To voice	3
	To pain	2
	None	1
Verbal Response	Orientated	5
	Confused, disorientated	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Best motor response	Obeys commands	6
	Localises to pain	5
	Withdraws from pain	4
	Abnormal flexion	3
	Abnormal extension	2
	None	1

Table 1: Using the Glasgow coma scale.

The patient's best response in each category is added. The maximum score is 15 and the minimum is 3. A patient with a GCS score of <8 is classed as in a coma and usually requires intubation in order to protect their airway. In this case an anaesthetist should be involved early. Further assessment includes pupil size and reactivity. A sluggish pupil progressing through to an enlarging, then dilated and non-reactive pupil demonstrates progressive 3rd nerve palsy. It must be assumed that this is due to significantly raised intracranial pressure until proven otherwise and requires neurosurgical advice/input immediately.

The primary survey demonstrates that the patient is maintaining his own airway and has equal air entry on auscultation. His respiratory rate is 16 and saturations are 100% on 15L oxygen via a non-rebreather facemask. His capillary refill is one second with a regular heart rate of 90 and blood pressure 160/90mmHg. His GCS is 12: eyes 3, motor 5 and verbal 4. Both pupils are 3cm and briskly reactive to light. His temperature is 36°C, his abdomen is soft and there is no evidence of limb injury. He has a 2cm laceration above his left ear with a small swelling beneath. He smells strongly of alcohol.

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How would you proceed?

It is essential that you gain as much information regarding his risk of a significant head injury using a thorough history and examination. In any case of head injury this can be difficult and a history from an observer, possibly friend or ambulance crew, is very useful. Important specific features in the history are[1]:

• Mechanism of injury – high-energy head injury includes:
- pedestrian RTA or high speed motor collision;
- ejected from motor vehicle or vehicle roll-over;
- fall from greater than 1 metre or >5 stairs;
- bicycle collision.

• Time since the injury.
• Loss of consciousness or drowsiness (e.g. difficulty keeping eyes open).
• Amnesia for events before or after injury.
• Persistent headache.
• Any vomiting since injury, especially if more than one episode.
• Any seizure after injury.
• Any focal neurology (i.e. restricted to a part of body or activity):
- includes balance, weakness, vision, speaking, writing, understanding.

• Drug or alcohol intoxication.
• Altered behaviour or irritability.
• Age >65.
• Coagulopathy or anticoagulation (e.g. warfarin).
• Suspicion of non-accidental injury.
• Previous cranial neurosurgical intervention.

The patient's previous medical history is important. It will help to assess the risk of an intracranial bleed, such as a history of previous brain surgery, coagulopathy or use of drugs that promote bleeding (e.g. warfarin). Additionally, it may help to diagnose how the patient obtained the head injury, such as a seizure, arrhythmia or hypoglycaemia in the diabetic patient.

When performing a clinical examination in a head injury case, there are some important areas that need to be focused on and documented. The examination should include a full assessment of neurological function, aiming to clarify findings from the history. This will include a detailed neurological examination of the upper and lower limbs, cerebellar examination and a full cranial nerve examination with documentation of pupil size and reaction to light. A unilateral pupillary dilatation may indicate local orbital injury or raised intracranial pressure. Glasgow Coma Score should again be formally assessed, as should the possibility of cervical spine injury.

Additionally, signs of a skull fracture or penetrating head injury should be sought including[1]:

• Obvious skull deformity or a deformity to cause concern to the doctor.
• Evidence of basal skull fracture:
- CSF from ear or nose;
- bleeding from ear or haemo-tympanum (blood behind eardrum);
- new deafness;
- battle's sign: bruising around mastoid. This may take 24 hours to appear;
- panda eye's: bruising limited to orbits indicates blood tracking from behind the eyes.

There is little history available as it is predominantly taken from his drunken girlfriend. The patient had drunk about 10 pints of lager and 1 hour ago was involved in a brawl. He was hit across the head with a glass bottle and he fell onto the carpeted floor in the pub. He did not appear to lose consciousness. He has not vomited and there has been no seizure. He has no significant medical history. He takes no medication and has no known allergies.

His observations remain stable. His GCS remains 12 (E3, M5, V4). His eyes are both reactive. His blood glucose (BM) is 5mmol/L.

What will you do with the patient now?

Once the clinical picture has become clearer, a decision needs to be taken on further management. The first question to ask is whether this patient needs an immediate CT scan to rule out an intracranial bleed (extradural haemorrhage, subdural haemorrhage, contusion or traumatic subarachnoid haemorrhage)? NICE guidelines (see below) give clear recommendations when a scan is indicated[1]. The indications for urgent CT scan (within 1 hour of injury) are:

• GCS less than 13 on initial assessment in the A&E department.
• GCS less than 15 at 2 hours after the injury on assessment in the A&E department.
• Suspected open or depressed skull fracture.
• Any sign of basal skull fracture.
• Post-traumatic seizure.
• Focal neurological deficit.
• More than one episode of vomiting.
• Amnesia for events more than 30 minutes before impact.

Additional indications for immediate CT head include age ≥65, coagulopathy or dangerous mechanism of injury where there is loss of consciousness or amnesia since the injury.

If a CT is not required then the next consideration is whether this patient can be safely discharged? If a patient is low risk without a dangerous mechanism of injury, they have a GCS score of 15/15 and no other factors that require hospital admission, they can be safely discharged to the care of a suitable adult with verbal and written head injury advice. If there is reason to have any clinical concern or the assessment is difficult due to drug or alcohol intoxication, the patient should be admitted for observation. In this case, this man's partner is also intoxicated and would be an inappropriate chaperone.

A patient should not be discharged home until their GCS has returned to 15/15[1].

Even in the presence of normal imaging, a patient should be admitted until their GCS is 15. If, after 24 hours, GCS score has not returned to 15/15, further imaging, such as CT or MRI, should be considered.

Decreased conscious level should be ascribed to intoxication only after a significant brain injury has been excluded[1].

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This patient fulfils the criteria for immediate CT head (and neck) but on discussion with the radiologist, the CT scanner is currently not working. You are told it should be fixed in the next few hours. You discuss this with a senior colleague who suggests that you admit the patient for neuro-observations until the CT scanner is available.

Is this acceptable?

NICE guidelines state that in the event of equipment failure, a patient with a GCS of 15 may be admitted for observation and transferred to a CT scanning facility in the event of clinical deterioration. This patient, however, should ideally be transferred to another CT scanning facility now because his GCS is only 12 out of 15.

If the patient is not co-operative, a judgement must be made regarding the need for general anaesthetic to facilitate CT scanning. The alternative is to admit the patient until CT scanning is available and the patient cooperative.

The patient is admitted to the ward for neuro-observations. He is put on half-hourly observations. Over the next 2 hours his GCS improves to 14 as he is able to open his eyes to voice (eyes 3), obey commands (motor 6) and is orientated (verbal 5). His other observations remain stable so the frequency of neuro-observations is reduced to hourly. Over the next hour, he becomes more confused and agitated, but continues to obey commands. His GCS is therefore 13/15. An hour later, his GCS has fallen further. He now opens his eyes to voice, localises to pain and says inappropriate words. His GCS is now 12 (E3,M5,V3). You are called to see him and confirm the findings. His pupils are both reactive although you wonder if the left pupil moves less briskly.

What should have been done and what should you do now?

Observations for the head-injured patient should include: GCS; pupil size and reactivity; limb movements; respiratory rate; heart rate; temperature; and oxygen saturations.

Having been admitted to the ward, it is right to perform half-hourly observations initially but this should be continued until the GCS is 15 and for a further 2 hours after this. If the GCS and observations are stable, they can then be reduced to 1-hourly for 4 hours, then 2-hourly thereafter. This observation was therefore lengthened too rapidly.

Any new agitation should cause alarm and requires a medical review. This patient's GCS may have improved as he "sobered up" and his blood alcohol levels fell. Any deterioration must be presumed to be due to the head injury until proven otherwise. Any drop of 1 point in the GCS that remains low after 30 minutes is significant. A drop in the motor score of 1 point is of even greater concern. Any evolving neurology signs including pupil signs require medical assessment. The accuracy of pupil signs in particular can be doubtful and subject to inter-observer variability. However, one should always err on the side of caution and not assume that changes in pupil size are insignificant. A CT scan should be performed urgently.

You are concerned that this patient's deterioration may be due to a significant head injury so contact your Surgical Registrar who is in theatre. He agrees it is of concern and promises to review him as soon as he is finished in theatre. He should be there in the next 30 minutes or so. He asks you to ensure his neuro-observations are continued half-hourly and to let him know if they drop further.

Is this appropriate?

This introduces further delay. The patient's falling GCS and new pupillary signs require an urgent CT scan to be performed. Even if the registrar is unable to assess the patient immediately, arrangements should be being made for the scan to be performed. Any additional delay to diagnosis and treatment is likely to worsen the patient's ultimate outcome.

Over the next 30 minutes, the patient's GCS drops further. His eyes remain closed, he withdraws from pain and he makes no sound. His GCS is now 6/15 (E1,M4,V1). His pupil is obviously dilated on the left. You contact your registrar who is en route. When your registrar arrives, the patient starts fitting.

What are the priorities now?

The priorities are to manage his **Airway, Breathing and Circulation** and to stop the fitting. This patient requires sedation, intubation and ventilation to prevent secondary brain injury. This ensures optimal oxygenation and cerebral blood flow and minimises the metabolic requirements of the injured brain.

A CT scan should be done as soon as possible to guide further management. If this is not immediately available, he needs an immediate transfer to a unit with CT scanning and neurosurgical facilities available.

This patient's CT scan demonstrates a large right-sided extradural haematoma (see Figure 1). This requires urgent surgical decompression. A call should be made to the on-call regional neurosurgical registrar and arrangements made to prepare the patient for transfer to the nearest neurosurgical unit for urgent surgery.

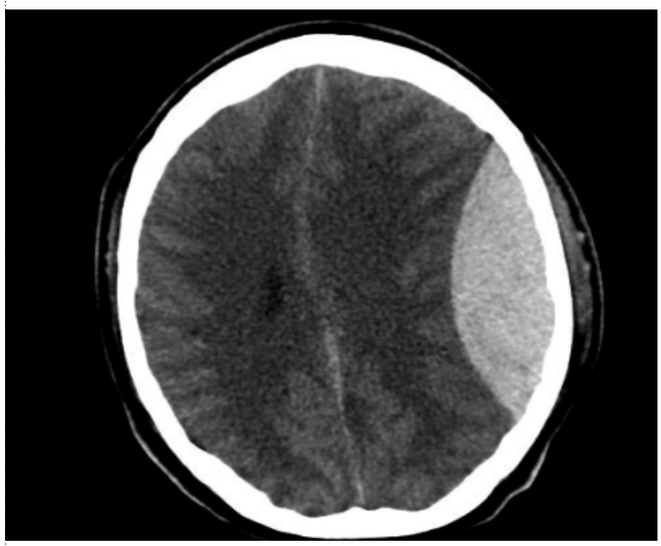

Figure 1: CT scan of the head.

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Monitoring and invasive lines should be sufficient to allow the safe transfer of the sedated patient and will normally include pulse oximetry, ECG, invasive blood pressure monitoring and expired carbon dioxide monitoring. However, delays to definitive surgery should not occur for routine X-rays or central line access unless this is clinically justified.

Delays to surgery must be minimised[3, 4]. The neurosurgeon will require a short amount of time to review the CT scans, confirm the diagnosis and arrange emergency theatre. If after a short delay, such as 15 minutes, you have not heard of the definitive plan, it is appropriate to again contact the neurosurgeon to clarify the This patient is transferred directly to a Neurosurgical theatre and undergoes craniectomy (removal of skull flap) and evacuation of haematoma.

It is likely he will remain sedated post-operatively with continuous intracranial pressure monitoring in the intensive care unit. It is possible that cerebral oedema will worsen over the next few days and some patients will die from uncontrolled cerebral oedema. However, other patients, despite a delay to surgery, do make a slow and steady neurological improvement and, with rehabilitation, can return to an independent lifestyle.

Summary and learning points

The patient described above is a typical presentation of an extradural haematoma. This case demonstrates the extremes of outcome that occur between the optimally-managed and the poorly-managed head-injured patient, and the contribution that Foundation doctors can make to patient outcome.

The patient with an extradural haematoma experiences a so-called "lucid period". This is because the extent of the primary brain injury, the damage caused by the mechanical impact, is often very minimal. If any loss of consciousness has occurred it is usually short-lived. The patient will often appear reasonably well. A GCS score of 15 during this lucid period demonstrates a healthy functioning brain. However, a skull fracture results in injury to the middle meningeal artery that runs over the outer surface of the dura. If this injury causes bleeding, it does so at arterial pressure. Therefore a progressively large haematoma develops within the rigid skull, reducing the space remaining for the healthy brain tissue. This causes a rise in intracranial pressure and a steady deterioration in brain function as it is compressed. The earlier this vicious cycle is stopped with surgery, the more likely the underlying healthy brain will maintain normal function. The longer that brain tissue is compressed and rendered ischaemic, the more likely it is that a poor outcome for the patient will result.

Prolonged delays cause the intracranial pressure to rise sufficiently high that brain shift occurs. Initially, herniation of the temporal lobe through the temporal hiatus results in traction on blood vessels supplying the midbrain and brainstem. Next, tonsillar herniation compresses the brainstem as the cerebellum pushes down through the foramen magnum. This is sometimes called "coning". Irreversible brain stem ischaemia progresses to brain stem death. Where brain stem death has occurred, the patient will die, usually from cardiovascular failure, over the next few days[5].

It is therefore essential that any head-injured patient is assumed to have a potentially serious head injury until proven otherwise. This requires a systematic approach as described above. It ensures that in the event of a multiply injured patient, management of the most life-threatening problems are managed first using the ABCDE approach.

A thorough history and examination will then help to determine the risk of serious head injury and focus the need for further investigations and management.

It must never be forgotten that a patient with an extradural haematoma can die if delays in treatment occur, but can fully recover with no long-term sequelae when prompt surgical evacuation takes place. Even when patients have suffered direct brain injury, such as acute subdural haematoma, intracerebral haematoma or contusion, the time to definitive treatment can have a significant impact in preventing secondary brain injury and improving outcome.

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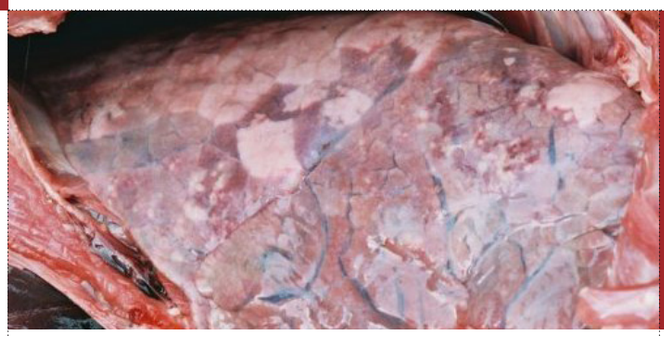
A COMMUNITY-ACQUIRED PNEUMONIA IN A PATIENT WITH CHRONIC LUNG DISEASE REQUIRING INTENSIVE CARE (LEVEL 3) ADMISSION

Jahnavi Dabali and Rob Townsend



We report a case of community-acquired pneumonia in a 73-year-old Caucasian lady with a background history of chronic airways disease (COPD) who was admitted in a state of septic shock.
Patient Management.

Abstract

We report a case of community-acquired pneumonia in a 73-year-old Caucasian lady with a background history of chronic airways disease (COPD) who was admitted in a state of septic shock and a CURB-65[1] score of 5 which needed level 3 (ITU) treatment and mechanical ventilation. Clinical examination and X-ray findings suggested bilateral consolidation complicated by a pleural effusion. Sputum and broncho-alveolar lavage (BAL) cultures grew S*treptococcus pneumoniae* and *Enterobacter cloacae*. She failed to respond to initial empirical antimicrobial treatment with co-amoxyclav and clarythromycin but responded well to meropenem.

Introduction

Case Presentation

A 73-year-old woman presented to the A&E department with increasing shortness of breath and confusion of 1-day duration. She was found in a state of collapse at home before admission.

Of significance from the past medical history, she had a past history of COPD with previous admissions for exacerbations. She was recovering from a recent stroke and had a past history of ischaemic heart disease (IHD). She underwent radiotherapy for laryngeal carcinoma 20 years ago.

On admission, she was assessed by the Foundation doctor who found her to be tachycardic (140/min), tachypnoeic (RR of 40/min) and hypotensive (Systolic BP: 70mmHg and Diastolic BP not recordable). Her SpO2 was 89% and the temperature was 36^{o}C (tympanic). On auscultation she had crackles and wheeze at both lung bases along with bronchial breathing in the left lung base. A provisional diagnosis of a chest infection was made and her blood gas analysis was as follows: pH 7.129, PaO_2 8.21kPa, PCO^2 11.3kPa indicative of type 2 respiratory failure. At this stage, a senior doctor was called to assess the patient in view of deteriorating respiratory function and a decision was made to intubate her. After intubation and resuscitation with IV fluids she was transferred to the ITU. Chest X-ray revealed an extensive consolidation in the left lower and right mid zones with moderate effusion on the left side. Her WCC 11.4 x 10^9/L, CRP 112mg/L and urea 11.7mmol/L. Sputum, urine, blood and broncho-alveolar lavage (BAL) samples were sent for microbiological investigations. She was empirically started on co-amoxyclav and clarithromycin for suspected severe community-acquired pneumonia (CAP) (CURB 65 score, 5). These were replaced by meropenem after 48 hours of treatment as she failed to improve clinically with worsening inflammatory markers (WCC 19.9 x 10^9/L and CRP 168mg/L).

The BAL and sputum samples grew *Streptococcus pneumoniae* and *Enterobacter cloacae* and the urine was negative for *Legionella* antigen (making a diagnosis of Legionella pneumonia less likely). There was a clinical improvement after 5 days of antibiotic treatment which was then stopped but she continued to be on a ventilator for another week before being weaned off it. This was also reflected by improved inflammatory markers (WCC 7.6 x 10^9/L and CRP 22mg/L). She had a prolonged hospital stay in view of her other co-morbidities.

Discussion

Here we presented a case of pneumonia in a COPD patient which illustrates that while the common respiratory pathogens, such as the pneumococci, are the key infecting organisms; gram-negative "coliforms", such as E*nterobacter sp.,* can also on occasions be pathogenic*.

Coliform organisms, such as the above, are often regarded as merely colonising flora. In the majority of cases where such organisms are seen in sputum samples, this is undoubtedly the case. But, occasionally in the context of those patients with chronic lung disease (or ventilator associated pneumonia patients) such organisms can be of clinical significance. In the case above, clinical improvement did not occur until antibiotic cover was altered to meropenem which is effective against such resistant coliforms as this.

Enterobacters belong to a group of coliforms known as AmpC producers. AmpC is a chromosomally encoded beta-lactamase enzyme found in certain organisms *(including Enterobacter, Citrobacter, Morganella, Serratia)*. The AmpC enzyme is an example of an inducible enzyme, so is only produced in the presence of its substrate and once present is capable of hydrolysing a range of beta-lactam antibiotics including penicillins, beta-lacatam/beta-lactamase inhibitor combinations (such as co-amoxyclav) and most of the cephalosporin class of antibiotics.

In the laboratory such "coliforms" may appear susceptible to a range of cephalosporin antibiotics unless the organism is either identified (i.e. knowing an organism is an Enterobacter species means you know it possesses AmpC) or a marker antibiotic is tested (such as cefoxitin) which is known to be a good in vitro inducer of the AmpC enzyme. Clinically while these organisms tend to test as susceptible to many cephalosporins and combination antibiotics (e.g. co-amoxyclav), such antibiotics should be avoided. This is because clinical failure can occur, either when the organism has switched its enzyme on or when the antibiotics have selected for a mutant strain which has lost the ability to repress the AmpC gene and so high level expression of AmpC now shows an organism which looks highly resistant on laboratory testing.

A COMMUNITY-ACQUIRED PNEUMONIA IN A PATIENT WITH CHRONIC LUNG DISEASE REQUIRING INTENSIVE CARE (LEVEL 3) ADMISSION

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Such organisms tend to be seen in patients who have already been exposed to antibiotics, and as such COPD patients who have frequently had antibiotic exposure and often quite broad spectrum antibiotics as well (co-amoxyclav, cephalosporins, etc.), are a group where such organisms are not uncommonly seen.

Again the case illustrates that while empiric antibiotics are usually correct choices as they are based on local prevalence data, in the context of a critical care setting or a patient that is not improving, we need to be willing to change antibiotics if we suspect clinical failure or the likely presence of resistant organisms.

In patients who we know have had or are likely to have resistant organisms, such as the one described, also not forgetting MRSA and ESBL (extended spectrum beta-lactamase coliforms), we may need to consider modifying our empiric choice to include such organisms as treatment failures in critical care have been shown to lead to higher morbidity, mortality and increased length of stays[2].

Multiple Choice Questions

1. Pneumonias are classified as community acquired and hospital acquired. The most common cause of community-acquired pneumonia (CAP) is:

a. *Haemophilus influenza.*
b. *S. pneumoniae.*
c. Influenza A and B.
d. *Mycoplasma* pneumonia.
e. *Legionella Spp.*

2. Causes of a poorly resolving pneumonia or recurrent pneumonia include all of the following except:

a. Aspiration of a foreign body.
b. Carcinoma of the lung.
c. Inappropriate antibiotic treatment.
d. Well-controlled insulin-dependent diabetes.
e. Congenital lobar abnormality.

3. The organism causing cavitational changes on a chest X-ray in a patient with pneumonia is most likely to be:

a. *L. pneumophila.*
b. *Haemophilus* influenza.
c. *Mycoplasma* pneumonia.
d. *Staphylococcus aureus.*
e. *C. psittaci.*

4. The CURB 65 score during initial hospital assessment is a good predictor of hospital mortality. What is the mortality rate if a patient has a CURB-65 score of 4 (out of 5):

a. 0.7%.
b. 3.2%.
c. 3%.
d. 17%.
e. 41.5%.

Answers

1. b.

Based on a study conducted in the UK, which involved 236 patients, the most common organism causing pneumonia in the community was Strep. *pneumoniae*. It accounted for 36% of cases of community-acquired pneumonia. Source: BTS guidelines for the management of community-acquired pneumonia in adults (2004 update).

Based on five studies conducted in the UK which involved 1,137 patients, the most common organism causing pneumonia in the hospital setting was Strep. *pneumoniae*. It accounted for 39% of all pneumonias in this setting.

Source: BTS guidelines for the management of community acquired pneumonia in adults (2004 update).

2. d.

Well-controlled diabetes mellitus is not a predisposing factor for poorly resolving or recurrent pneumonias, although they may be at increased susceptibility to develop infections. The causes for this increased susceptibility is multifactorial and includes decreased neutrophil functions, impaired cytokine production by macrophages and vascular compromise.

3. d.

Chest X-ray findings in a patient with pneumonia often give a clue to the likely organisms. Lobar or segmental opacification is a finding in bacterial pneumonias and in the majority of atypical infections. Lower lobes are most commonly affected in all types of pneumonia and a quarter of patients show pleural effusions. Cavitation is an uncommon but classic feature of *Staphylococcus aureus* and *Strep. pneumoniae* serotype 3 infections.

4. e.

CURB-65 scores.

A 6-point score, 1 point for each of **Confusion, Urea** >7 mmol/L, **Respiratory** rate >= 30/min, **Blood pressure** (2 points) low systolic (<90mmHg) or diastolic (<= 60 mmHg), age >= **65 years** (CURB-65 score) based on information available at initial hospital assessment, enables patients to be stratified according to increasing risk of mortality or need for intensive care admission.

CURB-65 score	0	1	2	3	4	5
Risk of mortality/need for intensive care admission	0.7%	3.2%	13%	17%	41.5%	57%

Significance of CURB-65 score:

Scores 0–1: likely suitable for home treatment.
Scores 2: consider for supervised hospital treatment.
Scores 3 or more: manage in hospital as severe pneumonia.

A similar pattern of increasing disease severity was reported when only clinical parameters were considered (CRB-65) giving a 5–point score:

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CURB-65 score	0	1	2	3	4	5
Risk of mortality	1.2 %	5.3 %	12.2 %	32.9 %	18.2 %	57%

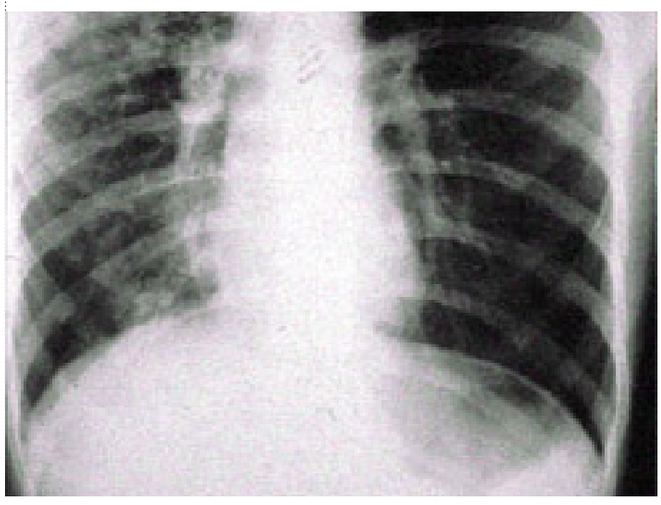
Significance of CRB score:

Scores 0: likely suitable for home treatment.
Scores 1 or 2: consider hospital referral.
Scores 3 or more: Urgent hospital admission.

Cell wall active agents	(Transpeptidase Inhibitors)
A) Betalactams	
Penicillins	Penicillin G/Penicillin V
Penicillinase resistant penicillins	Flucloxacillin/Temocillin
Broad spectrum	Ampicillin Amoxicillin Co amoxiclavamoxyclav Co fluampicil
Anti-pseudomonal penicillins	Piperacillin (+ Tazobactam) Ticarcillin (+ Clavulanic acid)
Mecillinams	Pivmecillinam
Cephalosporins	
1st Generation	Cephalexin
	Cefadroxil
	Cefazolin
2nd Generation	Cefaclor
	Cefprozil
	Cefuroxime
3rd Generation	Cefotaxime
	Cefoperazone
	Cefixime
	Ceftazidime
	Ceftriaxone
4th Generation	Cefepime
Monobactams	Aztreonam
Carbapenems	Imipenem Meropenem Ertapenem
	Transglycosylase Inhibitors
Transglycosylase Inhibitors	Vancomycin Teicoplanin
Cell Membrane agents	
Lipopeptides	Daptomycin
Lipopeptides	Colistin
Ribosome active agents	
Tetracyclines and Glycyl-cyclines	Tetracycline/Oxytetracycline Demeclocyline Lymecycline Doxycycline Minocycline Tigecycline
Macrolides	Erythromycin Clarithromycin Azithromycin
Lincosamides	Clindamycin

Figure 1: Site of action of commonly-used antibiotics.

	Common	Uncommon
Bacterial	*Streptococcus pneumoniae*	*Actinomyces and Arachnia spp*
	Haemophilus influenzae	*Moraxella catarrhalis*
	Staphylococcus aureus	*Francisella tularensis*
	Mixed anaerobic bacteria (aspiration)	*Acinetobacter var. anitratus*
	Enterobacteriaceae *Escherichia coli* *Klebsiella pneumoniae* *Enterobacter spp* *Serratia spp*	*Bacillus spp.*
	Pseudomonas aeruginosa	
	Legionella spp. (including L. pneumophila and L. micdadei)	
Fungal	Aspergillus spp	
	Candida spp.	
	Cryptococcus neoformans	
	Coccidioides immitis	
Rickettsial	*Coxiella burnetii*	
	Rickettsia rickettsiae	
Mycoplasma	*Mycoplasma pneumoniae*	*Chlamydia trachomatis*
	Chlamydophila psittaci	
Mycobacterim	*Mycobacterium tuberculosis*	Nontuberculous mycobacteria
Parasitic	*Pneumocystis carinii*	*Ascaris lumbricoides*
		Toxoplasma gondii
Viral		
Children	Respiratory syncytial virus	Adeno Vir types 1, 2, 3, 5
	Parainfluenza virus types 1, 2, 3	Rhinovirus
	Influenza A virus	Influenza B virus
		Coxsackievirus
		Measles virus
Adults	Influenza A and B	Rhinovirus
	Adenovirus types 4 and 7	Adeno Vir types 1, 2, 3, 5



A COMMUNITY-ACQUIRED PNEUMONIA IN A PATIENT WITH CHRONIC LUNG DISEASE REQUIRING INTENSIVE CARE (LEVEL 3) ADMISSION

Jahnavi Dabali and Rob Townsend

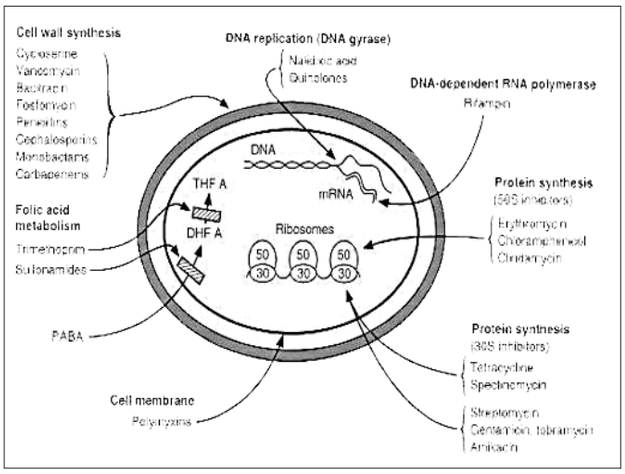


Figure 1: Site of action of commonly-used antibiotics.

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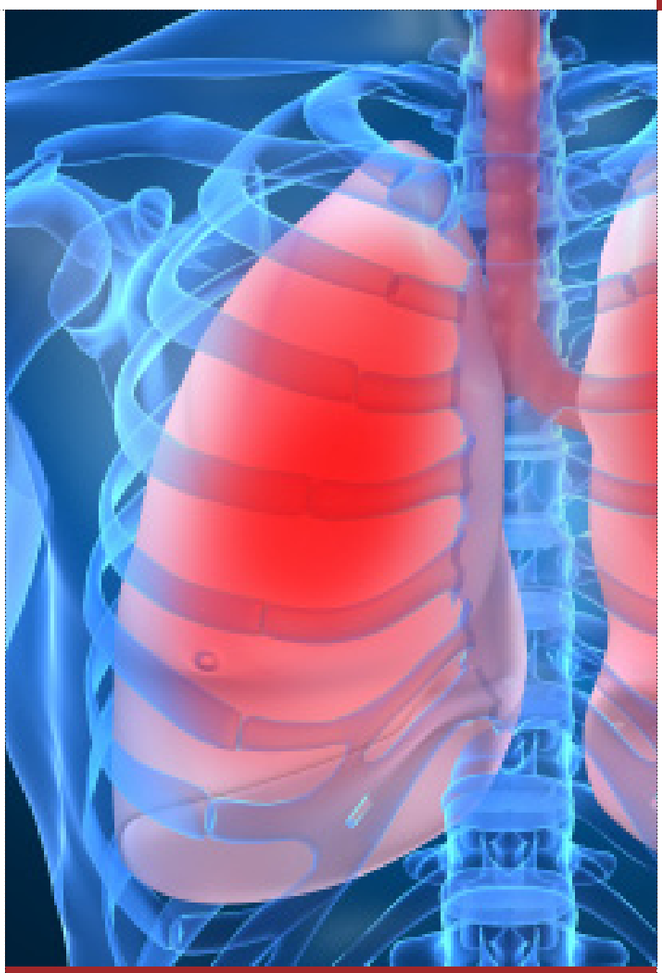

A community-acquired pneumonia in a patient with chronic lung disease. Patient Management.

NUTRITIONAL SUPPORT IN HOSPITAL

Joanne Butterworth

Malnutrition is both a cause and a consequence of ill health. Good Clinical Care.

Introduction

Malnutrition is both a cause and a consequence of ill health. Unfortunately it is common in the UK, especially in those who are unwell. In 2006–2007 almost 140,000 people in England and Wales were malnourished when they left hospital. Surveys consistently find that about 20% of patients in general hospitals are malnourished. Yet in up to 70% of these people, the fact they are malnourished is not diagnosed and therefore not properly addressed[1]. Even people who are well nourished, eat and drink less if they are ill or injured. Many older people and patients with long-term medical or psychosocial problems are chronically underweight. This can impair recovery or precipitate other medical conditions. Malnutrition is associated with increased length of hospital stay and increased likelihood of readmission. Foundation doctors need to be able to identify patients who have malnutrition, or are at risk of developing it, and refer them for appropriate treatment. Nutrition support is an important part of medical treatment and when considering whether to withhold it, or in failing to provide it, it is regarded in law as being equal to drug treatment.

The causes of malnutrition

These can be divided into four main headings.

1. Impaired intake due to:

a. Poor appetite because of illness, pain/nausea when eating, depression/anxiety, food aversion, medication or drug addiction.
b. Inability to eat because of diminished consciousness, weakness or arthritis in the hands or arms, dysphagia, vomiting, painful mouth conditions, poor oral hygiene or dentition, restrictions imposed by surgery or investigations.
c. Lack of food because of poverty, poor quality diet at home, in care homes or in hospital, problems with shopping and cooking.

2. Impaired digestion and/or absorption

Due to medical and surgical problems affecting the stomach, intestine, pancreas and liver.

3. Altered metabolic requirements

Related to illness, surgery, organ dysfunction or treatment.

4. Excessive nutrient losses:

a. GI Losses from vomiting, diarrhoea, fistulae, stomas, nasogastric tube losses, drain losses.
b. Other losses (e.g. skin exudates from burns).

Physical factors usually associated with illness are the main cause of malnutrition in adults in the UK, and individual patients may have more than one cause of malnutrition. Psychosocial issues have significant effects in some groups, such as the socially isolated, recently bereaved, low income groups and some older people.

The consequences of malnutrition

Malnutrition detrimentally affects physical function, psychosocial well-being and the outcome of disease. It can affect every system and tissue of the body with wide reaching clinical consequences.

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Adverse Effect	Consequence
Impaired immune responses	Predisposes to infection and impairs recovery when infected
Impaired wound healing	Surgical wound dehiscence, *anastomotic breakdowns, development of post-surgical fistulae,* failure of fistulae to close, increased risk of wound infection and non-united fractures. All can then lead to prolonged recovery from illness, increased length of hospital stay and delayed return to work
Reduced muscle strength and fatigue	Inactivity, inability to work effectively, and poor self-care. Abnormal muscle (or neuromuscular) function may also predispose to falls or other accidents
Reduced respiratory muscle strength	Poor cough pressure, predisposing to and delaying recovery from chest infection, difficulty weaning malnourished patients from ventilators
Inactivity, especially in bed-bound patients	Predisposes to pressure sores, thromboembolism and muscle wasting
Water and electrolyte disturbances	Malnourished individuals are usually depleted in whole body potassium, magnesium and phosphate, while simultaneously overloaded in whole body sodium and water. They also have reduced renal capacity to excrete a sodium and water load. This leads to vulnerability to refeeding syndrome and iatrogenic sodium, and water overload
Impaired thermoregulation	Hypothermia and falls, especially in older people
Vitamin and other deficiencies	Specific vitamin deficiency states, for example, scurvy and vitamin-related refeeding risks (e.g. Wernike-Korsakoff syndrome). Mineral deficiencies include iron deficiency anaemia and magnesium deficiency, which can cause tetany. A lack of trace elements can also be a cause of a range of problems
Menstrual irregularities/amenorrhoea	Infertility and osteoporosis
Impaired psychosocial function	Even when uncomplicated by disease, patients who are malnourished may experience apathy, depression, self-neglect, hypochondriasis, lack of self-esteem, poor body image, possible confusion about slow recovery, lack of interest in food, loss of libido and deterioration in social interactions. Malnutrition may also affect behaviour and attitude

Table 1: Showing adverse effects and their consequences[2].

In addition, malnutrition is associated with more frequent GP consultations and more frequent and longer hospital admissions. Surgical patients with malnutrition have around three times as many complications and four times greater risk of death than well-nourished patients[2].

If malnutrition persists for weeks, it can be life-threatening in itself.

Identifying malnourished patients

Clearly if malnutrition is the cause of so many problems, it is imperative that patients who are or are at risk of being malnourished are identified so that appropriate treatment can be started. Many different criteria have been used historically to assess malnutrition. The NICE guideline on nutritional support in adults[2] recommends the use of the "Malnutrition Universal Screening Tool" (MUST)[3]. This incorporates both current weight status and unintentional weight loss.

• Malnourished patients are defined as those with any ONE of the following:

1. BMI <18.5 (BMI = weight in Kg divided by height in metres squared).
2. Unintentional weight loss >10% within last 3–6 months.
3. BMI <20 **plus** unintentional weight loss >5% within last 3–6 months.

• Patients at risk of being malnourished are defined as those who:
- have eaten little or nothing for more than 5 days;
- are likely to eat little or nothing for the next 5 days or longer;
- have a poor absorptive capacity;
- have high nutrient losses;

NICE guidelines recommend:
- have high nutritional needs from causes, such as catabolism.

• There should be screening for malnutrition or the risk of malnutrition on:
- all hospital inpatients on admission;
- all outpatients at their first clinic appointment;
- all people in care homes on admission;
- all people on registration at GP surgeries;
- upon clinical concern*.

• Screening should be repeated:
- weekly for inpatients;
- upon clinical concern* for outpatients including patients in care homes.

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• Screening should be considered at other opportunities (e.g. health checks, flu injections).

Clinical concern includes
Unintentional weight loss
Apathy
Altered taste sensation
Loose fitting clothes
Fragile skin
Wasted muscles
Impaired swallowing
Prolonged intercurrent illness
Poor wound healing
Poor appetite
Altered bowel habit

Providing nutrition support

Having identified a patient who is malnourished, or at risk, the next step is to determine how best to provide nutritional support. All medical graduates should have a basic understanding of human nutrition. In *"Tomorrow's Doctors"*[4], the GMC states that "they must know about and understand the role that lifestyle, including *diet and nutrition* can play in promoting health and preventing disease". In addition, The Royal College of Physicians report "Nutrition and patients: a doctors responsibility"[5] states that "the clinical importance of nutrition is often overlooked by doctors...all doctors should be aware of nutritional problems and how to manage them".

There are some *clinical risks* associated with providing nutrition support. For example, oral supplementation can cause pneumonia in dysphagic patients. Enteral and parenteral feeding can cause GI problems, infections, metabolic upset and trauma.

There are also *ethical issues* relating to the provision, or not, of nutritional support. The risks and benefits of providing such support need to be discussed with patients and their consent obtained. If patients have capacity, difficult clinical and ethical issues can arise if the patient does not wish to artificially prolong their life. A competent adult can decline treatment including nutritional support even if this refusal is likely to result in their death. If patients do not have capacity, clinicians need to act in the best interests of the patient. In considering this, doctors should consult with family and carers, and take their views into account in the decision-making process. If there is any doubt as to a patient's capacity or what is in their best interest, legal advice should be sought.

The key decisions when planning nutritional support relate to what to provide and how to provide it.

What to provide

In people who are not severely ill or injured and not at risk of refeeding problems, the usual provision is[6]:

- 25–35 kcal/Kg/day total energy;
- 0.8–1.5g protein (0.13–0.24g nitrogen)/Kg/day;
- 30–35ml fluid/Kg/day;
- Adequate electrolytes, minerals, micronutrients and fibre.

These requirements may vary according to the patient's activity level and the underlying clinical condition (e.g. catabolism or pyrexia).

Particular care needs to be taken when using preparations which tend to supplement energy and/or protein without adequate micronutrients (all essential vitamins and trace elements) or minerals; or when using pre-mixed TPN bags that have not had tailored additions from pharmacy.

How to provide nutritional support

This may be:

1. Oral – providing fortified food, extra snacks and special drinks.
2. Enteral – delivering a complete feed directly into the gut via a tube.
3. Parenteral – giving nutrition intravenously.

1. Oral nutritional support

Where possible, the oral route is preferred. However, this is not possible in patients who have dysphagia. There are many indicators of dysphagia, some more obvious than others. Obvious indicators of dysphagia

Obvious indicators of dysphagia
Difficult, painful chewing or swallowing
Regurgitation of undigested food
Difficulty controlling food or liquid in the mouth
Drooling
Hoarse voice
Coughing or choking before, during or after swallowing
Globus sensation (pharyngis)
Nasal regurgitation
Feeling of obstruction
Unintentional weight loss e.g. people with dementia
Less obvious indicators of dysphagia
Change in respiratory pattern
Unexplained temperature spikes
Wet voice quality
Tongue fasciculation (may indicate motor neurone disease)
Xerostomia
Heartburn
Change in eating habits e.g. eating slowly, or avoiding social occasions
Frequent throat clearing
Recurrent chest infections
Atypical chest pain

Tables 2 and 3: Obvious and less obvious indicators of dysphagia[12].

NUTRITIONAL SUPPORT IN HOSPITAL

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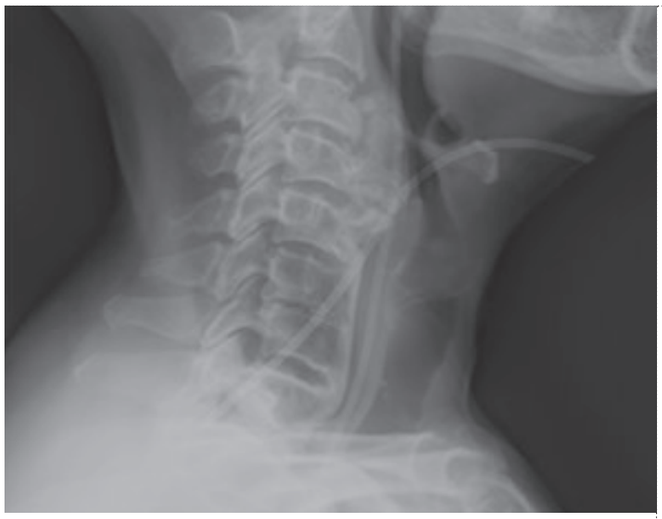

Any patient suspected of having dysphagia should be referred to health care professionals with skills in the diagnosis, assessment and management of swallowing disorders (e.g. Speech and Language Therapy, SALT). These patients must not receive nutritional support via the oral route.

Patients who **are** safe to swallow should receive adequate food and fluid in an environment conducive to eating. Providing normal food and drink, plus physical help to eat if needed may be enough to prevent malnutrition occurring. Government initiatives, such as "Better Hospital Food"[7] and "Protected Mealtimes"[8] are aimed at addressing the problems of poor catering standards, inappropriate or interrupted mealtimes, incorrect food consistencies and inappropriate eating aids and/or staff to help patients eat and drink for themselves.

Sip feeds can be used as supplements for patients who are unable to obtain their full nutritional requirements from ordinary foods alone. There are some nutritionally complete sip feeds which can be used as a sole source of nutrition. The Advisory Committee on Borderline Substances (ACBS) has identified approved conditions for which sip feeds may be prescribed. These are listed in Appendix 7 of the British National Formulary (BNF)[9] and include preoperative preparation of patients who are malnourished, proven inflammatory bowel disease and total gastrectomy. Dietary supplements are useful for patients with increased energy or protein requirements, or a small appetite. A full range of the available sip feeds and other nutritional supplements is also listed in Appendix 7 of the BNF[9]. These feeds should be prescribed on the drug chart to ensure that they are given at regular and appropriate times. The overall nutrient intake should contain a balanced mixture of protein, energy, fibre, electrolytes, vitamins and minerals.

For malnourished surgical patients, consideration should be given to perioperative oral nutrition support. Post-operative oral intake can generally be started within 24 hours of surgery.

Once adequate oral intake can be achieved from normal food, then oral nutritional support should be stopped.

2. Enteral nutritional support

In the acute setting, patients with unsafe or inadequate oral intake should be considered for a 2–4-week trial of nasogastric enteral tube feeding. For longer-term enteral tube feeding, a gastrostomy tube is often used. These are usually inserted percutaneously (Percutaneous Endoscopic Gastrostomy or PEG tubes) and may be used 4 hours after insertion. If there is dysfunction or inaccessibility of the upper gastrointestinal tract then post pyloric (i.e. duodenal or jejunal feeding) may be used instead.

Nasogastric tube position should be checked after insertion and before each use with aspiration of gastric contents, and testing with pH graded paper. The initial placement of post pyloric tubes should be checked with an abdominal X-ray, unless they were placed radiologically.

A wide range of enteral feeds are available. They vary in the amounts of protein, energy, sodium, volume, etc., which they provide. This allows feeding to be tailored to the individual patient and their clinical condition. These feeds are also listed in Appendix 7 of the BNF[9].

Enteral tube feeding may be delivered continuously or as boluses:

- In intensive care, feeding is generally continuous over 16–24 hours.
- If the patient requires insulin, it is preferable to feed over 24 hours to avoid any swings in glycaemic control.
- Ambulant patients may prefer overnight feeding, allowing them to be disconnected from the feed and to mobilise more freely during the day.

There should be a review of medication with expert advice from a pharmacist to ensure that formulations and timings are appropriate for nasogastric tube administration. When a tablet is crushed, it disintegrates immediately in the stomach and the drug is rapidly absorbed. If it was designed to disintegrate and be absorbed quickly, crushing makes little difference. However, with tablets designed for slow release, crushing can significantly alter absorption times and blood levels[10]. Also, some drugs require an empty stomach for absorption and so feed may need to be discontinued for a short time to allow this.

Some patients with delayed gastric emptying will respond to the use of a prokinetic (e.g. metoclopramide or erythromycin). Sometimes, feeding may be so severely limited by delayed gastric emptying, that post pyloric feeding or TPN have to be used instead.

Preoperative enteral tube feeding is sometimes used in surgical patients due to undergo major abdominal procedures. However, it should be reserved for those who are malnourished with inadequate or unsafe oral intake.

Patients can have enteral tube feeding in the community. There needs to be an individualised care plan with input from dieticians, district or care home nurses, GPs, pharmacists and sometimes also speech and language therapists.

Enteral tube feeding should be stopped when the patient is established on adequate oral intake.

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Total Parenteral Nutrition (TPN) should be reserved for patients who are malnourished. Good Clinical Care.

3. Parenteral nutrition

Total Parenteral Nutrition (TPN) should be reserved for patients who are malnourished or at risk of being so, cannot eat safely or adequately and have a non-functional, inaccessible or perforated GI tract.

For short-term (<14 days) feeding, a peripheral venous catheter can be used in patients who have no other need for central access. If feeding for more than 14 days, a Peripherally Inserted Central Catheter (PICC) can be used; or a dedicated lumen in a multi-lumen central line.

If feeding for more than 30 days, a tunnelled subclavian line is recommended.

Severely ill patients requiring TPN should receive continuous administration of feed. Delivery may be changed from continuous to cyclical in patients receiving TPN for more than 2 weeks.

In order to minimise refeeding problems, TPN should be commenced at no more than 50% of estimated energy and protein needs, building up to meet full needs over the first 24–48 hours. It should be increased progressively and the patient monitored closely.

There are various proprietary infusion fluids for parenteral feeding which differ in the amounts of energy, nitrogen and electrolytes which they provide. They are listed in Chapter 9 of the BNF[11].

TPN may be provided in the community. As with enteral tube feeding, it requires multidisciplinary team involvement to deliver an individualised care plan for each patient.

If adequate oral and/or enteral nutritional support can be established, TPN should be stopped using a step wise approach with daily review of the patient's progress.

Monitoring nutritional support

Hospital inpatients receiving nutritional support should be reviewed daily initially and then twice weekly with regard to:

• Nutritional factors:
- total nutrient intake;
- actual volume of feed delivered;
- fluid balance charts.

• Anthropometric factors:
- weight;
- BMI;
- mid-arm circumference;
- triceps skin fold thickness.

• GI function:
- nausea and vomiting;
- diarrhoea;
- constipation;
- abdominal distension.

Further checks should be carried out according to the method of feeding used. For patients who are enterally fed via a nasogastric tube, gastric tube position should be checked before each feed. There should be daily checks for nasal erosion, of tube fixation and that the tube is working properly.

For patients fed via a gastrostomy or jejunostomy, there should be daily checks of stoma site and tube position. Patients receiving TPN should have daily checks of their catheter entry site whether peripheral or central.

All patients should have their general clinical condition reviewed daily and their temperature and blood pressure checked at least daily initially, and then as needed. Drug therapy should be reviewed daily at first and then reduced to monthly to ensure drugs are prepared appropriately in order to reduce the incidence of tube blockage and to minimise any drug/nutrient interactions.

There should also be reviews to ensure that feeding goals are being met, and also that those goals are still appropriate to the patient.

In addition to clinical monitoring, there should be laboratory monitoring for all patients receiving nutritional support.

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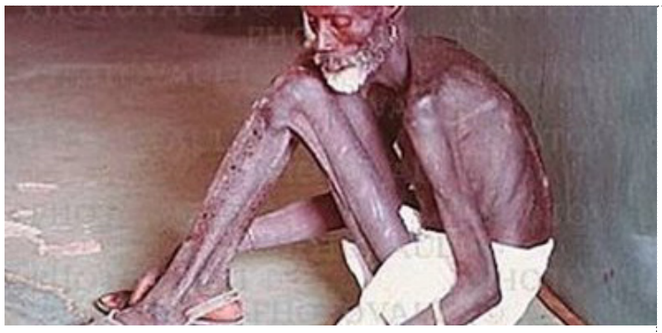

Refeeding problems

These can occur with oral, enteral or parenteral feeding in any severely malnourished individual. However, they are less likely with oral feeding because starvation is usually associated with a loss of appetite so refeeding does not occur too rapidly. Care must still be taken with the prescription of oral nutrition supplements, especially in patients with eating disorders. Enteral tube or parenteral nutrition feeding are more likely to cause refeeding problems because it is much easier to over feed. The problem may be made worse by using feed which does not include adequate vitamins, phosphate or electrolytes.

Refeeding problems happen because starvation causes reductions in cellular activity and organ function accompanied by micronutrient, mineral and electrolyte deficiencies. Giving fluid and nutrients to malnourished individuals will reverse these changes, but causes an increased demand for electrolytes and micronutrients. Feeding also results in a shift of sodium and water out of cells. If feeding is over rapid or unbalanced, it can precipitate:

- Fluid overload or depletion.
- Disturbances of organ function (e.g. cardiac failure, pulmonary oedema).
- Metabolic disturbances.
- Electrolyte imbalances including hypophosphataemia, hypokalaemia, hypomagnesaemia, hypocalcaemia and hyperglycaemia.
- Life-threatening acute micronutrient deficiencies.

Refeeding syndrome can occur when nutritional support is started in any patient who has had very little food intake for more than 5 days. In these patients, feeding should be introduced at a maximum of 50% of requirements for the first 2 days. Close monitoring is essential. If this reveals no refeeding problems, then after 2 days, the rate of feeding can be increased to meet full needs.

Some patients with very severe malnutrition are at even greater risk of developing refeeding problems. They include those with any one of the following:

- BMI <16 Kg/m^2.
- Unintentional weight loss of >15% within the last 3–6 months.
- Very little or no nutrient intake for >10 days.
- Low levels of potassium, phosphate or magnesium prior to any feeding.

Patients with two or more of the following lesser criteria are also at high refeeding risk:

- BMI <18.5 Kg/m^2.
- Unintentional weight loss >10% within last 3–6 months.
- Very little or no intake for >5 days.
- A history of alcohol abuse.
- The use of some drugs including insulin, chemotherapy, antacids or diuretics.

These patients should have feeding commenced at very low levels of energy and protein, with very gradual increases and careful monitoring throughout. This is best achieved with expert multidisciplinary team involvement to provide an appropriate regime.

All high-risk patients should commence feeding at very low levels of energy and protein, for example, a maximum of 10 kcal/kg/day, increasing levels slowly to meet or exceed full needs by 7 to 10 days. In extreme cases, use only 5 kcal/kg/day[2].

Circulating volume should be restored with fluid balance and overall clinical status monitored closely. Most of these patients will also need generous electrolyte supplementation even if pre-feeding levels are normal. If levels are already high (e.g. in renal impairment) supplementation may need to be more cautious, but even in these patients, total body levels of these electrolytes can be low and will drop further as refeeding progresses and renal function improves. A multivitamin and trace element supplement should also be provided.

Feeding levels can be increased slowly from their very low baseline level over the next few days only if careful clinical and biochemical monitoring reveals no problems.

The Wernicke-Korsakoff syndrome

Acute thiamine deficiency can occur in alcoholics and any chronic vomiting condition where refeeding causes an increased thiamine demand. It results in acute neurological abnormalities known as the Wernicke-Korsakoff syndrome. This requires expert management involving intravenous vitamin supplementation in addition to feeding, as starving cells switch back to carbohydrate metabolism. It is seen particularly in alcoholics who may have lower liver stores of thiamine. However, it can occur in any patient with chronic vomiting including those with gastric outlet obstruction and hyperemesis gravidarum.

The clinical syndrome consists of acute neurological abnormalities including:

- apathy and disorientation;
- nystagmus, ophthalmoplegia or other eye movement disorders;
- ataxia;
- severe impairment of short-term memory, often with confabulation.

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Patients should be managed as for refeeding syndrome with particularly high doses of daily thiamine and other B vitamins intravenously for 3 days.
Good Clinical Care.

Patients should be managed as for refeeding syndrome with particularly high doses of daily thiamine and other B vitamins intravenously for 3 days. It is not necessary to correct electrolyte disturbances *prior* to starting feeding. The eye signs and impairment of consciousness usually resolve, but the short-term memory loss may be permanent.

Benefits of providing nutritional support

For most patients, the provision of nutritional support is beneficial and without complications. Malnourished patients feel better when they are fed and are more able to cope with ill health; recuperation times are reduced and patients are less susceptible to further problems. Rapid functional benefits will occur with adequate feeding even before the weight loss has been regained. Supporting patients who are unable to eat enough on their own can keep them alive long enough for specific medical or surgical interventions to take effect.

Nutrition – true of false questions

1. General

a. Approximately 50% of hospital inpatients are malnourished.
b. Up to 70% of malnourished hospital inpatients are not identified as malnourished.
c. Hospital length of stay is not affected by a patients nutritional status.
d. All doctors should be aware of nutritional problems.
e. Nutritional support should be given to patients even if they do not really want it.

2. Causes of malnutrition

a. Poor dentition can cause malnutrition.
b. Surgical inpatients who are being kept "nil by mouth" can develop malnutrition.
c. Burns patients have excessive nutrient losses from skin exudates.
d. Diarrhoea and vomiting alone do not cause malnutrition.
e. Patients who have been recently bereaved are at risk of malnutrition.

3. Consequences of malnutrition

a. Malnourished surgical patients are four times more likely to die than comparable but well-nourished patients.
b. Malnourished patients are usually depleted in whole body sodium, potassium and magnesium.
c. Infertility can result from malnutrition.
d. Malnourished patients on intensive care are not more difficult to wean from a ventilator.
e. Phosphate deficiency can cause tetany.

4. Identifying and treating malnourished patients

a. Anyone with a BMI below 19 is malnourished.
b. Screening for malnutrition requires admission to hospital.
c. Nutritional support is the concern of nurses and pharmacists only.
d. Typical energy requirements are 50kcal/Kg/day.
e. Micronutrients are too small to worry about.

5. Oral and enteral nutritional support

a. Patients suspected of having dysphagia should be encouraged to eat and drink as much as possible.
b. Sip feeds are only suitable for patients with certain conditions.
c. Most surgical patients should be kept "nil by mouth" for a week post-operatively.
d. Gastrostomy feeding should be employed for anyone requiring more than 1 week of enteral support.
e. Medication which is crushed and put down an n.g. tube will be rapidly absorbed.

6. TPN and refeeding problems

a. Patients with an inaccessible or dysfunctional upper GI will have to have TPN.
b. TPN can be given peripherally.
c. TPN should be started at a maximum of 50% of estimated energy and protein.
d. Refeeding problems can occur with any method of feeding.
e. Cardiac failure is a recognised consequence of refeeding problems.

Answers

1. General

a. False. b. True. c. False. d. True. e. False.

Teaching notes: 20% of hospital inpatients are found to be malnourished, which affects outcome from hospital admission. All members of the care team, including Foundation doctors need to consider nutritional status in every patient. Artificial nutritional support via the enteral or intravenous route is classed as treatment, and competent patients should not be subjected to treatments they do not consent to.

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2. Causes of malnutrition

a. True. b. True. c. True. d. False. e. True.

Teaching notes: poor dentition, surgery and recent bereavement are all causes of impaired intake due to physical or psychological factors. Diarrhoea and vomiting affect digestion, absorption and increase nutritional losses, while burns patients have altered metabolic needs.

3. Consequences of malnutrition

a. True. b. False. c. True. d. False. e. False.

Teaching notes: the consequences of malnutrition are referred to in the table in the text. Patients have a sodium overload in malnutrition. Low calcium can cause tetany.

4. Identifying and treating malnourished patients

a. False. b. False. c. False. d. False. e. False.

Teaching notes: NICE guidance is discussed in the article. Malnutrition is a BMI below 18.5, patients do not need to come into hospital to be assessed. Consideration of malnutrition is the responsibility of all the members of the multidisciplinary team and appropriate calories of 25–35 kCal/kg/day plus micronutrients should be provided.

5. Oral and enteral nutritional support

a. False. b. True. c. False. d. False. e. True.

Teaching notes: dysphagia makes the oral feeding route difficult or contraindicated, and sip feeds are best used for identified groups of patients (e.g. those needing additional dietary support preoperatively. Gastrostomy is considered after 2–4 weeks of enteral feeding and crushing may speed up the absorption rate in slow release preparations.

6. TPN and refeeding problems

a. False. b. True. c. True. d. True. e. True.

Teaching notes: upper GI dysfunction does not require TPN as some patients may be suitable for other enteral feeding (e.g. gastrostomy/jejunostomy). Although TPN can be given peripherally it is better given via a dedicated central line to reduce infectious complications and phlebitis. Patients that have been malnourished should be given reduced calorie and protein for the first 1–2 days to reduce the risk of refeeding syndrome and consequent heart failure.

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EMERGENCY OXYGEN USE IN ADULTS

Sarah Heikal

Introduction

Oxygen is perhaps the most commonly used and under recognised drug in patients presenting with medical emergencies. Most trainees who look after acutely unwell patients will have some experience of patients suffering the adverse effects of over or under use of oxygen, yet few of us ever prescribe it. Why then are we so poor at prescribing this particular drug, when we recognise the correct prescription of other therapeutic agents as one of "the essentials" of medical care? Despite its importance, the physiology of oxygen delivery (and consequently the pathophysiology of impaired oxygen delivery) is not always fully understood by many medical staff. As junior doctors we tend to receive conflicting advice from more senior colleagues (especially from differing specialties), who often have very strong ideas about the administration of oxygen, despite a relative lack of randomised controlled trials on which to base them.

The resulting confusion often leads to oxygen being given for indications for which there is no evidence (breathlessness in the absence of hypoxia – for example, in patients with chronic airways disease (COPD)), or sometimes even despite evidence to the contrary (high concentration oxygen being given in uncomplicated myocardial infarction or stroke). Similarly, life-saving oxygen is often withheld for fear of carbon dioxide retention. The British Thoracic Society has sought to address these issues by publishing a comprehensive set of guidelines for the use of emergency oxygen use in adults[1]. This is largely based on a combination of observational studies and expert opinion – randomised controlled trials could be potentially very harmful to untreated patients – but the methodology is clear and well explained.

The guideline is a full review of the theory of acute oxygen therapy and covers not only mask types, prescribing, monitoring and the management of individual conditions, but also the underlying respiratory physiology behind it all. It is an extensive document and well worth reading. This article summarises the key points made, and provides some examples from cases to illustrate them.

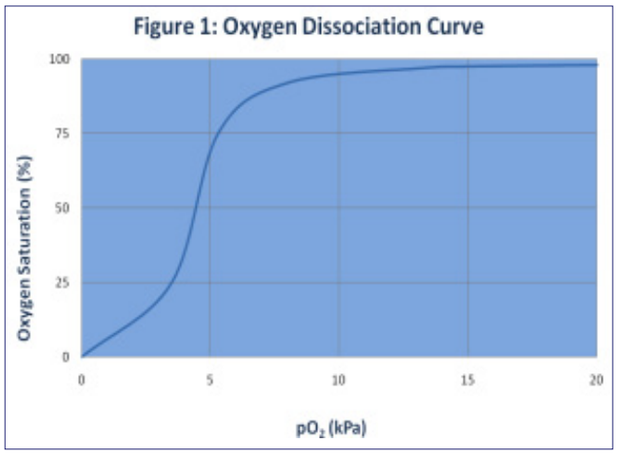

Figure 1: Oxygen dissociation curve.

Targeting saturations

The guidelines recommend targeting oxygen saturations (SaO_2), and adjusting the amount of oxygen required to achieve them accordingly. For the majority of patients this means aiming for saturations of 94–98%, the expected SaO_2 of normal healthy adults at sea level. The oxygen dissociation curve demonstrates the reason for this (see Figure 1). An increase from 98% to 100% saturation – shown by the flat part of the curve – has little effect on the amount of oxygen made available to the tissues (see Table 1). Below this, however, at an SaO_2 of ~92% (pO_2 ~8kPa), the curve becomes much steeper and any fall below this results is a significant reduction in oxygen carriage. Patients presenting with acute medical emergencies who are neither grossly hypoxic nor critically ill can therefore be given oxygen via a face mask or nasal cannulae, titrated according to their saturations.

System involved	Effects	Risks
Metabolic system	Increased 2,3- DPG production Haldane effect (increased CO_2 carriage)	Metabolic acidosis
Respiratory system	Increased ventilation and pulmonary vasoconstriction	Pulmonary hypertension
Cardiovascular system	Coronary vasodilatation Tachycardia Reduction in systemic vascular resistance (transient only) Increased cardiac output	Ischaemia/infarction of myocardium/other organs Hypotension Arrhythmias
Neurological system	Cerebral vasodilatation	Confusion, delirium, coma
Renal system	Activation of renin-angiotensin axis	Acute tubular necrosis

Table 1: Shows the physiological effects of hypoxia and its consequences.

In some situations the target saturations suggested by the guidelines are different:

- In patients who are at risk of hypercapnia (patients with moderate/severe COPD, chest wall deformities, morbid obesity) target saturations are lower at 88–92%.

- In specific conditions, such as carbon monoxide poisoning, where raised or "supranormal" oxygen levels are beneficial. Carboxyhaemoglobin has a half-life of 5 hours in patients breathing room air, but this is reduced to just 90 minutes when high-flow oxygen is administered via a reservoir (or non-re-breather mask). In carbon monoxide poisoning, saturation monitors are unreliable, as they are unable to differentiate between oxyhaemoglobin and carboxyhaemoglobin. Blood gases will also show a normal PaO_2, even in the presence of tissue hypoxia. It is therefore important to measure carboxyhaemoglobin levels (which can be as high as 50% in acute poisoning) and continue with high-flow oxygen until they return to "normal" levels (<3% in non-smokers, <10% in patients who are smokers).

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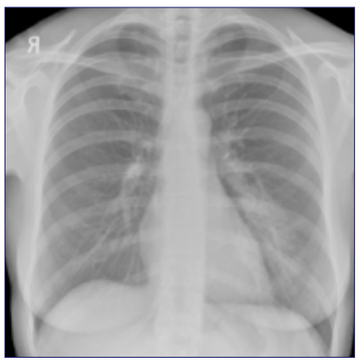
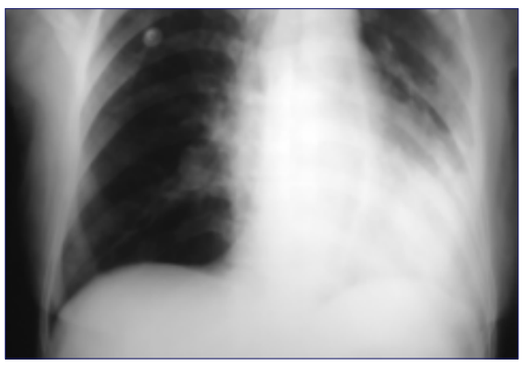


Figures 2 and 3: Chest X-ray of a woman with left lower lobe consolidation, on initial presentation and a repeat film taken 24 hours later.



Oxygen Use in Acute illness

Some serious illnesses are associated with hypoxia (e.g. pulmonary embolus, asthma) and may require significant levels of supplemental oxygen to correct this. If the patient is hypoxic, oxygen should therefore be given as well as attempting to correct the underlying cause where possible (e.g. drainage of pleural effusions, anticoagulation). Unfortunately, despite treatment, patients may deteriorate (see Figures 2 and 3), and require high-flow oxygen and even respiratory support in the form of ventilation. In such cases reassessment of the patient and senior help are both required.

There are traditionally a number of conditions, such as stroke and acute coronary syndromes, where supplemental oxygen has been given to all patients in an attempt to increase oxygen delivery to the brain or heart. However, there is no evidence that this is beneficial in the absence of hypoxaemia[2]. There is even some evidence to suggest that it may be harmful. For example, in acute coronary syndromes supranormal oxygen levels may result in vasoconstriction and increased systemic vascular resistance, thereby reducing myocardial blood supply[3]. Similarly, hyperoxia can result in cerebrovascular vasoconstriction, and recent studies have shown increased mortality in patients with mild to moderate strokes who are not hypoxic and are given supplemental oxygen[4].

By keeping a patient's pO_2 within normal ranges the damaging effects of acute hypoxia are prevented, while also avoiding any potential harm caused by hyperoxia. Oxygen should therefore be prescribed according to the patient's target saturations rather than a specific flow rate or inspired concentration (FiO_2).

It is worth noting that in some patients – such as the elderly, those with chronic lung disease, congenital cyanotic heart disease or chronic neuromuscular conditions – a lower SaO_2 may be normal. A sudden fall of >3%, even if saturations are within the target range for that patient, requires further assessment as it may be the first indication of an acute illness.

Oxygen use in hypercapnic patients

The most significant effect of excess oxygen on the respiratory system is hypercapnic respiratory failure, resulting in acidosis. There are thought to be several different mechanisms responsible for this, and the exact mechanisms are still subject to debate[5-8].

However, it is important to note that this phenomenon does not occur in patients in the absence of significant pulmonary disease (most commonly COPD) or musculoskeletal disease which affects the thorax. Also, not all patients with pulmonary disease will suffer with hypercapnic (type II) respiratory failure. Even among those COPD patients who are chronically hypercapnic, not all will develop a further rise in carbon dioxide during an acute exacerbation. Hypercapnia can occur while oxygen levels remain within normal limits, and even in the presence of mild hypoxia.

Although pulmonary disease may result in chronic hypoxia, it is the sudden reduction in available oxygen associated with acute exacerbations that will result in the same damaging effects as hypoxia in a patient with no underlying lung disease (see Table 1). However, oxygen is often withheld for fear of oxygen poisoning.

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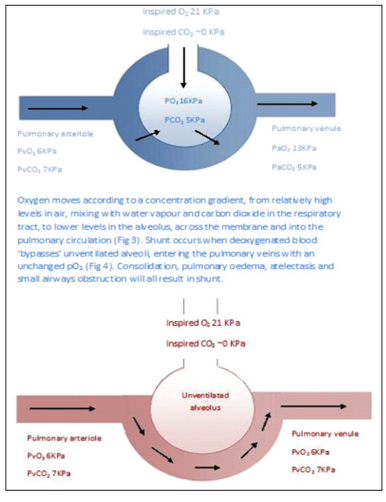
The BTS guidelines[1] provide some helpful guidance. In critical illness, initial treatment for all patients should be with high-flow oxygen, which can then be adjusted accordingly (see below). In patients with serious, but not critical illness, initial target saturations should be 88–92%, and controlled oxygen given (if needed) to achieve these. If an arterial blood gas then shows pCO_2 to be normal (and there is no history of the patient requiring ventilation) oxygen can then be increased, aiming for a range of 94–98%. Blood gases should then be remeasured after 30–60 minutes. Some patients are prone to repeated episodes of type II respiratory failure. In these patients supplemental oxygen levels should, based on previous blood gas estimations taken during previous acute illness.

Box 1: A patient is admitted with sepsis and oxygen saturations of 90%. A full blood count is taken and it shows his haemoglobin to be 5g/dl. Increasing his inspired oxygen improves his oxygen saturations to 100%, and his total haemoglobin oxygen content also increases by 10%. Giving the patient a 2 unit blood transfusion would increase his haemoglobin oxygen content by 40%, significantly improving oxygen delivery to the tissues.

If hypercapnic respiratory failure does result following high concentration oxygen therapy, patients are at risk of further damage from rebound hypoxia. This can occur if oxygen is withdrawn too quickly, in an attempt to correct the results of excessive oxygen therapy, and again this can be more dangerous than the hypercapnic failure itself. Sudden reduction in inspired oxygen leads to a rapid fall in oxygen tension to below the tension that was present prior to the commencement of supplementary oxygen therapy (the BTS guidelines provides helpful worked examples using the alveolar gas equation to explain this). Oxygen therapy should therefore be reduced gradually, through a range of venturi devices, with continuous saturation monitoring.



Figures 4 and 5: Show the effect of shunt on gas exchange in the alveoli (adapted BTS guidelines1).

Oxygen use in critically ill patients

There are a number of medical emergencies in which patients are very likely to suffer from hypoxia. These include sepsis, shock, trauma, massive pulmonary embolus and anaphylaxis. In these instances all patients – including those at risk of hypercapnia – should be started on high-flow oxygen (15L/min) via a reservoir or "non-re-breathing" mask. Once stable this can be titrated down to achieve saturations of 94–98%. In patients with COPD and other risk factors for hypercapnia initial target saturations should still be 94–98% until blood gas results are available, after which it may be possible to give controlled oxygen therapy. However, if they show the patient to be severely hypoxic and/or hypercapnic with a respiratory acidosis then the patient is likely to require supported ventilation and needs senior review and possible referral to critical care.

Increasing oxygen delivery in critically ill patients has been shown to reduce organ failure and length of ICU stay, and improves mortality (although there is still no evidence to support "supranormal" oxygen levels)[9, 10]. However, supplemental oxygen is only part of increasing oxygen delivery to tissues. As well as increasing the amount of oxygen inspired there are some simple but vital things that can be done:

- maintain and support an adequate airway;
- treat airflow obstruction (e.g. use of bronchodilators, chest physiotherapy to clear sputum); and
- reverse any respiratory depressants (e.g. opiates with naloxone).

Treating pulmonary oedema and consolidation increases the number of alveoli available to take part in gas exchange, as does reducing atelectasis by positioning of the patient, chest physiotherapy and use of positive airways pressure, for example, using CPAP (Continuous Positive Airways Pressure).

Adequate oxygen delivery to the tissues is dependent upon oxygen carriage and cardiac output. Nearly all oxygen is carried as oxyhaemoglobin and therefore correcting anaemia will significantly improve the delivery of oxygen to the end organs (see Box 1). Cardiac output is dependent upon an adequate circulating volume and venous return, as well as optimal myocardial function, so attention should be paid to the patient's fluid status (in sepsis in particular, large volumes of crystalloids may be required to ensure good fluid resuscitation). Severely shocked patients may require treatment with vasopressors and/or inotropes to improve systemic vascular resistance and maximise cardiac output. Treatment with inotropes requires invasive monitoring (e.g. central lines to monitor central venous pressure), and therefore critical care help should be sought sooner rather than later if this is the case.

Final thoughts

Knowing how much oxygen to give an acutely unwell patient can seem difficult and there are several pitfalls to avoid. However, using a systematic approach for each patient will help you determine whether oxygen is required, and if so how much should be given. The BTS guidelines provide a good source for further reading, in particular should you wish to refresh your knowledge of oxygen physiology. Here are a few points to remember:

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A 26-year-old woman is brought to the A&E department short of breath. Her partner tells you she is known to be asthmatic.
Good Clinical Care.

- Approach each patient as an individual, aim for saturations that are normal for them (see Box 2).
- Do not withhold oxygen from acutely hypoxic patients. Some patients are at risk of hypercapnic failure – try to identify these patients early and adjust target saturations accordingly.
- All critically ill or peri-arrest patients should be commenced on high-flow oxygen via a reservoir mask, including those at risk of hypercapnia. Once an ABG has been obtained, controlled oxygen therapy or supported ventilation may be necessary, depending on the results.
- Treat the underlying cause of hypoxia and try to improve oxygen delivery whenever possible.
- If uncertain, ask for senior help.

Consider the ABG below, taken from a patient on oxygen driven nebulisers:

pH 7.32
pO_2 9.7
pCO_2 6.5
HCO_3 24.9
BE -0.2

If the patient was, for example, 65 years old and known to suffer from COPD the gases would not be alarming. By reducing the patient's inspired oxygen (preferably controlled using a venturi device or similar) and continuing the nebulisers the mild respiratory acidosis would be corrected, and the patient would still be receiving adequate oxygen. However, if this gas was taken from a young asthmatic patient they would be alarming, as a raised or even a normal pCO_2 is indicative of very severe illness. The patient would require immediate and aggressive treatment as well as critical care involvement.

Box 2: Always consider the patient in front of you.

Case examples – self-test

Here are some example cases for you to consider. Read each one and consider how you would manage each patient – in particular the amount of oxygen you would give and the target saturations you would aim for (answers below).

Case 1

A 26-year-old woman is brought to the A&E department short of breath. Her partner tells you she is known to be asthmatic and has several previous hospital admissions, including one admission to ICU. She is alert but struggling to complete short sentences. Her observations are as follows: temp 36.5ºC, RR 28/min, SaO_2 95% on 15L O_2, HR 110/min, BP 110/67mmHg. Peak flow is 40% of the patient's best. On auscultation there is bilateral wheeze and reduced air entry throughout.

What is your immediate management and what else would you want to consider?

Case 2

A 79-year-old woman who is a life-long heavy smoker presents to the A&E department. Her daughter reports the patient has recently been diagnosed with "asthma" by her GP and started on inhalers. She has been increasingly short of breath over the past 6 months and her exercise tolerance is now limited to 1 flight of stairs. She started with a productive cough 4 days ago and her family say she was short of breath at rest last night but refused to come to hospital. They found her short of breath, cyanosed and confused this morning, and called an ambulance. Her observations are as follows: HR 110/min, BP 110/54mmHg, RR 36/min, SaO_2 84% on 15L O_2, temp 37·5ºC. She is very drowsy and difficult to rouse. On auscultation you hear bilateral wheeze with very reduced air entry throughout and right basal crepitations. You perform an ABG while waiting for a portable chest X-ray:

- pH 7.21
- pCO_2 8.2kPa
- pO_2 6.3kPa
- HCO_3 24.9mmol/L
- BE -11.

What is your immediate management? Would you consider reducing the rate of inspired oxygen?

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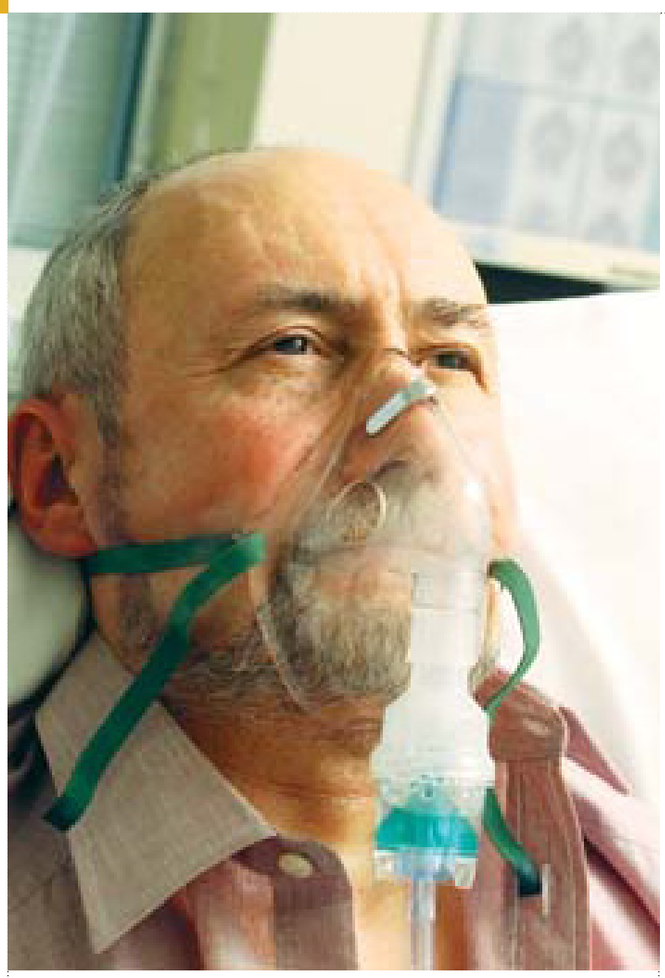

Case 3

An 82-year-old man is presents to the A&E department following an episode of chest pain and shortness of breath, which was relieved by sublingual GTN and oxygen. He is known to have multiple co-morbidities including COPD but lives alone. You are asked to see him by the nursing staff who are concerned that he has become drowsy and difficult to rouse. His observations are as follows: temp 36·2ºC, blood glucose (BM) 6.7mmol/L, HR 55/min, BP 132/89mmHg, RR 12/min, SaO_2 99% on 15L O_2 (high-flow oxygen via a reservoir mask was commenced by the ambulance crew and this has not been altered).

A keen medical student has taken an ABG for you:

- pH 7.30
- pCO_2 11.1kPa
- pO_2 10.5kPa
- HCO_3 37mmol/L
- BE -3.

What is the likely cause of the patient's decreased conscious level? How would you manage this patient?

Case 4

A 68-year-old woman with a history of hypertension presents with sudden onset of right-sided arm weakness and facial droop 1 hour ago, witnessed by her husband. She is otherwise fit and well. She is alert and orientated and her observations are as follows: temp 36·9ºC, HR 90/min, BP 165/92mmHg, RR 17/min, SaO_2 94% on air, BM 5.7mmol/L

What investigations would you request for this patient? Would you commence her on oxygen?

Case 5

A 67-year-old man with a history of an inferior STEMI and LVF 2 months ago. He underwent a successful Percutaneous Coronary Intervention (PCI) but required 2 weeks on CCU for treatment of pulmonary oedema. He has been short of breath on exertion since discharge but has represented following an acute onset of worsening breathlessness. The patient also has a history of COPD for which he takes regular inhalers. He is short of breath at rest, and was found to be cyanosed (SaO_2 76% on air) by the ambulance crew, who administered 15L O_2 via a reservoir mask. On arrival he was seen by the A&E department team where he received a dose of 40mg furosemide IV. On examination you find the patient alert but making considerable effort to breathe. He has bilateral pitting oedema and his JVP is raised to the earlobes. Auscultation reveals widespread crackles bilaterally and a gallop rhythm. Observations are as follows: temp 35·9ºC, HR 120/min, BP 120/82mmHg, RR 33/min, SaO_2 92% on 15L O_2. An ABG was been taken by the A&E doctor, who was particularly concerned that the patient is at risk of hypercapnia:

- pH 7.30
- pCO_2 5.2kPa
- pO_2 8.0kPa
- HCO_3 20mmol/L
- BE -4.

The nursing staff want to know if they should reduce the rate of inspired oxygen. Would you alter the patient's oxygen therapy at this stage? What are your next steps in the treatment of this patient?

Answers

Case 1

This is severe acute asthma. Oxygen-driven nebulisers, "back to back" if needed, and corticosteroids (either oral prednisolone 40mg, or 200mg hydrocortisone IV) should be given. Oxygen should be continued in between nebulisers, with target saturations of 94–98%. Magnesium (1.2–2g IV) can be given in patients with severe asthma who respond poorly to initial inhaled bronchodilator therapy, and in those with life-threatening or near-fatal asthma. IV aminophylline is no longer part of initial therapy (but can be considered in patients with near-fatal asthma and in those patients with life-threatening asthma who respond poorly to initial therapy). Bloods, including electrolytes, should be sent – some patients with asthma require rehydration and correction of electrolyte imbalance (in particular hypokalaemia may result from bronchodilator therapy).
Chest X-ray should not be routinely performed in the absence of suspected

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pneumothorax or consolidation, failure to respond to treatment, life-threatening asthma and/or the need for mechanical ventilation.

Arterial blood gases should be performed if SaO_2 is <92% at any point (whether on oxygen or air) or if there are any other features of life-threatening asthma present.

This patient has been admitted to ICU previously. Patients with acute severe life-threatening asthma who are not responding to therapy should be referred to ICU. This includes patients with evidence of hypercapnia, ↓pH or ↑H^+, persisting or worsening hypoxia, drowsiness, exhaustion, feeble respiration and a silent chest.

Case 2

This patient has acute (on chronic) hypercapnic respiratory failure. The "asthma" her family report is in fact likely to be COPD. Even in this context she is significantly hypoxic despite high-flow oxygen. The rate of inspired oxygen should not be reduced, despite the respiratory acidosis. The patient needs treatment with bronchodilators – nebulised salbutamol and ipatropium bromide, IV aminophylline – and corticosteroids, but may also need non-invasive ventilation (BiPAP). She should therefore be discussed urgently with senior staff. Target saturations should be between 88–92%. In some patients with COPD or other pulmonary disease it may be appropriate to aim for saturations lower than this, but this should be based on previous observations and ABGs. The history and examination findings are consistent with an infective exacerbation of COPD, and perhaps even pneumonia (the chest X-ray may confirm this), therefore empirical antibiotics should be given also according to your local hospital policy.

Case 3

This patient is likely narcosed' as a result of the hypercapnia shown on the gas (although it is important to consider other possible causes). Although the bicarbonate suggests that there is a marked chronic component to this, the pH and base excess indicate that there has been an acute rise, which has arisen due to excess oxygen therapy. The pO_2 is probably higher than "normal" for this patient as he is drowsy. The rate of inspired oxygen should therefore be titrated down according to the patient's saturations, using a series of venturi devices to control the flow rate. Target saturations should be between 88–92% until previous ABG results for this patient become available. The patient should also be sat upright if drowsiness improves to enable maximum ventilation, and nebulised bronchodilators given "back to back" if required. A repeat ABG should then be taken at 30–60 minutes and in the interim he should be discussed with seniors for review and consideration for non-invasive ventilation.

Case 4

This patient has an acute neurological deficit, which is likely due to a left partial anterior circulation infarct. The time of onset (within 3 hours) means that she is potentially a candidate for acute stroke thrombolysis, if this available. She should therefore have an urgent CT head, to exclude a haemorrhagic stroke, and be discussed with the neurology team on call with the results. Bloods, including a coagulation screen, should also be sent. Her saturations are within the target range of 94–98% and therefore these should continue to be monitored. She does not require additional oxygen. If her saturations fall outside of this range, or by more than 3%, she should be reassessed as to the cause of this – in particular stroke patients are at increased risk of aspiration pneumonia due to poor swallow.

Case 5

The history and examination findings are consistent with cardiogenic pulmonary oedema. Although the ABG shows an acidosis, it is metabolic in origin and not due to hypercapnia (the pCO_2 is within normal limits) – tissue hypoxia is the likely cause and this will improve as oxygen delivery to the tissues improves. The patient is in respiratory distress and requiring high-flow oxygen to maintain his saturations. This should not be reduced at this stage.

The patient should be sat up and supported comfortably. A GTN infusion should be commenced and titrated according to the patient's blood pressure (keep the systolic BP >90mmHg). IV opioids can be given to patients complaining of chest pain or who are distressed. However, it should be given in only very small increments and should not be used in patients who are drowsy, confused or exhausted as it can precipitate a respiratory arrest.

The patient's response to this treatment, including his saturations should be monitored closely. Venodilatation and reduction in preload from the GTN and opiates may result in rapid improvement, and the rate of oxygen can be gradually reduced while keeping saturations between 88–92%. However, if the patient does not improve he should be discussed with senior staff as he will likely require critical care involvement and CPAP.

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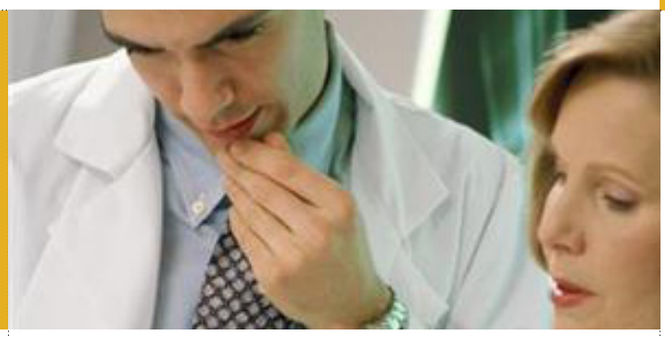
Thank you to Dr Danny Bryden
Consultant in Intensive Care Medicine, Northern General Hospital, Sheffield

Emergency Oxygen Tank.
Good Clinical Care.

MANAGING MEDICATIONS IN THE PERIOPERATIVE PERIOD

Alexander Kojro and Marie-Christine Law Min

It is the first Wednesday in August, your bleep goes, a nurse informs you that a patient has taken all his regular medications and is due to go to theatre on the afternoon list. Good Clinical Care.



It is the first Wednesday in August, your bleep goes, a nurse informs you that a patient has taken all his regular medications and is due to go to theatre on the afternoon list.

What do you, the new Foundation doctor, do?

Many patients admitted to hospital for surgery will be taking medicines which affect or are affected by perioperative medications, anaesthetics and even the surgery itself. Common dilemmas experienced by Foundation doctors are:

- To stop or continue routine drugs?
- What are the issues with withholding or continuing long-term therapies?
- Are there alternative drugs or routes of administration?
- Who to consult for advice?

In this review, the management of a number of common drugs and the issues regarding their continuation and discontinuation during the perioperative period are discussed. Areas of drug therapy examined consist of drugs affecting the cardiovascular, central nervous and endocrine system. An overview of the management of some common herbal medicines is also included.

Cardiovascular medications

A 65-year-old gentleman with stable angina, chronic cardiac failure and a history of myocardial infarction (MI) is admitted to the ward. His list of current medications includes aspirin, clopidogrel, frusemide, bisoprolol and ramipril. A detailed preoperative assessment should include a cardiovascular risk assessment. Cardiovascular conditions should be well controlled before surgery and patients with cardiovascular disease need to be carefully monitored as they are at an increased risk of MI, and in-hospital mortality, of around 30%. Below is a discussion of commonly prescribed cardiovascular medications and their possible implications for surgery[1].

Digoxin

A cardiac glycoside which increases myocardial contraction and reduces conductivity within the atrioventricular node. It is used in heart failure and supraventricular arrhythmias (i.e. atrial fibrillation). Omission can lead to recurrence of atrial fibrillation which carries an associated increased risk of thromboembolism, hypotension, tachycardia and myocardial ischaemia. There are preparations available if the patient is unable to take oral medications or has difficulty swallowing, although a dose adjustment may need to be made when changing preparation.

It is important to continue this medication up to and including the day of surgery, if possible. However, it is not without its potential for complications. The anaesthetist will need to be aware that the patient is taking digoxin as suxamethonium can precipitate cardiac arrhythmias when given concurrently, thus there may need to be a change of anaesthetic technique[1, 2]. Measurement of the digoxin level may be useful if any signs of toxicity are present or therapeutic efficacy is in doubt. It may also help determine the dose in patients with perioperative renal impairment.

Diuretics

Thiazide and loop diuretics are usually continued preoperatively. They may be withheld on the day of surgery to reduce the added complications experienced with hypovolaemia and hypotension especially in fasted patients[1].

However, potassium-sparing diuretics, such as spironolactone, should be withheld on the day of surgery as tissue damage and reduced kidney perfusion in the immediate post-operative period can lead to reflex hyperkalaemia[3].

Anti-arrhythmic medications

Amiodarone, a class III anti-arrhythmic drug has been associated with perioperative bradycardia (resistant to atropine), profound vasodilatation, low cardiac output and even death. However, this drug is often continued as withdrawal can lead to the recurrence of rhythm abnormalities and it would need to be stopped months before surgery due to its long half-life. Intravenous (IV) amiodarone is an option when oral or enteral therapy is not possible post-operatively, although cardiac monitoring facilities must be available throughout IV administration.

Some anti-arrhythmic medications (disopyramide, procainamide, quinidine) can prolong the muscle relaxation effects of non-depolarising neuromuscular blockers. However, they are often continued as this problem may be avoided by using smaller doses of the neuromuscular blockers and the consequences of a recurrence of abnormal heart rhythm far outweighs the complications of remaining on the medication.

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ß-Blockers

The continued use of these drugs preoperatively is recommended as 12–72 hours after stopping ß-blockade withdrawal like symptoms including nervousness, tachycardia, headache, exacerbation of myocardial ischaemia, myocardial infarction and even sudden death may develop. In addition, ß-blockade has been shown to reduce cardiovascular morbidity and mortality associated with the preoperative increased release of catecholamines, either through antagonism of the sympathetic effects of stress hormones or control of ventricular rate[4].

ACE Inhibitors

A commonly used class of drugs with proven efficacy in the treatment of a large and diverse group of medical conditions including heart failure, hypertension, diabetic nephropathy and retinopathy, and the prophylaxis of cardiovascular events. A large proportion of patients requiring surgery will be on one of these medications[2]. However, this class of drug is routinely stopped before surgery.

It has been found that continuation of ACE inhibitors preoperatively is not associated with improved blood pressure or heart rate control and that conversely in patients chronically treated with ACE inhibitors there is often a severe clinically relevant hypotension. These hypotensive episodes are more likely to require treatment with vasopressive agents[5]. ACE inhibitors also carry the risk of causing or worsening renal impairment post-operatively in vulnerable patients. Because of these problems, it is common for patients to be asked to stop ACE inhibitors 48 hours before surgery.

ACE inhibitors may be restarted when blood pressure and electrolytes normalise post-surgery. A good urine output and normal renal function must be ensured before restarting.

Anticoagulation therapy

Major surgery poses an increased risk of haemorrhagic complications as well as being a risk factor for thrombus formation. It is widely accepted that some form of anticoagulation therapy should continue for most of the preoperative period. As a result, most hospitals will have their own policy and it is beneficial to make yourself aware of this as soon as you start your surgical job.

Patients are often taking oral anticoagulation for a number of reasons including:

- atrial fibrillation (AF);
- venous thromboembolic disease (e.g. DVT, PE);
- arterial thromboembolic disease;
- prosthetic heart valves.

The British Society for Haematology recommends that unless there is a very high risk of thromboembolism, anticoagulation should be discontinued in preparation for surgery[6]. However, there are no definitive guidelines and the risk of further thromboembolic events and procedure related haemorrhage must be balanced.

Warfarin

Is a medication many patients are taking in the community as it is the anticoagulant of choice for DVT/PE, prosthetic valves and AF. How safe it is to stop and whether there is a need for heparin as alternative anticoagulation in the perioperative period often depends on local guidelines and may even require seeking expert advice. As an example the guidelines at The Northern General Hospital, Sheffield are illustrated in Table 1 below.

If warfarin is to be discontinued, then this should be done 4 days before surgery with the aim to allow the INR to fall below 1.5, which is a level considered safe for most types of surgery to take place. As warfarin is a vitamin K antagonist, vitamin K can be used to reverse the effects of warfarin if there is sufficient time.

AF/ PE/DVT	Pre-op: stop warfarin 4 days preoperatively, admit 2 days before operation, check INR and start subcutaneous enoxaparin 40mg once daily when INR <2.0. Post-op: continue enoxaparin and restart warfarin on the day after surgery provided haemostasis is secure. When INR >2.0 on 2 consecutive days stop enoxaparin
Prosthetic valve	Pre-op: stop warfarin 4 days preoperatively, admit 2 days before surgery, check INR and APTT. Start IV heparin when INR ≤2.5, with target APTT range of 1.8–2.7. Stop heparin 6 hours before surgery and check INR + APTT 2 hours preoperatively. Post-op: restart IV heparin 6 hours post-op (no loading dose). Restart warfarin on the day following surgery provided haemostasis is secure[6]. Stop heparin when INR >2.5 on 2 consecutive days.

Table 1: Showing anticoagulation advice as taken from local guidelines at Northern General Hospital, Sheffield, (INR: international normalised ratio, APTT: activated prothrombin time).

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Aspirin.
Good Clinical Care.

Antiplatelet agents

Aspirin

Causes an irreversible inhibition of platelet cyclo-oxygenase which lasts for the life time of the platelet (7–10 days). There is no definitive consensus regarding the use of aspirin in the preoperative setting. It is a balance between the risk of haemorrhage and predisposing the patient to an increased risk of thromboembolic complications. In procedures with a higher risk of bleeding or where post-operative haemorrhage could have serious implications, such as spinal and neurosurgery, it would be acceptable to stop aspirin. If aspirin is stopped, this should happen 7–10 days before surgery to enable recovery of sufficient platelet function. Post-operatively, aspirin may be restarted when the risk of bleeding is no longer significant.

Clopidogrel

Is a thienopyridine which acts by irreversibly binding to receptors on the platelet, inhibiting platelet aggregation for the life span of the cell. Studies have found that the concurrent use of this medication with aspirin gave a further 10% relative risk reduction in patients with non-ST elevation acute coronary syndrome (ACS) or unstable angina.

Clopidogrel has been referred to as the "surgeon's headache" because of its propensity to cause bleeding. Research has found an increase in bleeding over placebo of 1% with clopidogrel alone and if used in addition to aspirin this rises to 50%. The risk of haemorrhage must be weighed against the risk of predisposing the patient to a thromboembolic complication in order to decide whether or not clopidogrel should be continued perioperatively. Where clopidogrel is used for patients with drug-eluting stents, continuation is advised. In such patients, any decision to stop clopidogrel should be discussed with a cardiologist.

Full return of platelet function would require complete platelet replacement, however, homeostasis does not require 100% of all circulating platelets to be functional. Clopidogrel can be discontinued 5–7 days before surgery with no increase in haemorrhagic complications. This has been adopted as the standard for the majority of surgery[8]. If stopped preoperatively, clopidogrel can be restarted when the bleeding risk is no longer significant post-surgery.

Diabetes

These patients are prone to acute metabolic problems, complications associated with renal and vascular manifestations of their diabetes and delayed wound healing. They also have an increased risk of post-operative infections. These inherent risks are exacerbated by the stress response caused by surgery leading to both glycogenolysis and gluconeogenesis, resulting in a hyperglycaemic state with the possibility of ketoacidosis, electrolyte disturbances and protein catabolism[1, 9].

This is also an area where hospitals will have their own local guidelines and it is important to familiarise yourself with these at the earliest opportunity. In this example, the guidelines from the Northern General Hospital, Sheffield were used. It may be necessary to seek expert help from the diabetic team to prevent delay and inappropriate diabetic management.

The first consideration should be regarding the circumstances surrounding the patient's need for surgery. For an elective operation, good preoperative diabetes control is important, typically assessed by a patient's Hb A1c (target <9%). However, in the following circumstances poor control should not preclude surgery:

- urgent/emergency cases;
- cases of possible or proven malignancy;
- where delay could cause the patient significant morbidity.

It is also important to be vigilant for the undiagnosed diabetic patient: untreated perioperative diabetes carries a higher mortality risk than either non-diabetic or treated diabetic patients. If practical, it would be of use to do a random or preferably a fasting blood glucose level on the following groups of patients: age >60 or >40 if Asian/African Caribbean; ischaemic heart disease; claudication; hypertension; obesity; abscess; fungal infection; history of recurrent sepsis; or a family history of diabetes.

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Insulin-treated diabetes

Patients with type 1 diabetes should be placed on a morning list and ideally be eating at lunchtime where possible. Depending on the type of surgery and glucose control, patients may be managed differently. For instance, some patients may need to reduce their bedtime insulin dose before surgery, omit their insulin on the morning of surgery and restart their usual insulin post-operatively once eating and drinking normally. Others may need to omit their usual insulin on the day of surgery and start on an intravenous insulin/dextrose infusion until normal diet is resumed. Patients who have been on an insulin sliding scale regime for a considerable period of time should be referred to the diabetes team. If the operation is in the afternoon, a light breakfast may sometimes be allowed. In this case, the diabetes team may be required to advise on the patient's morning insulin dose[10].

From an anaesthetic view point the use of regional anaesthesia and dual prophylactic antiemetic therapy should be considered to allow diet to be reintroduced a soon as possible. Consideration should be given to the choice of perioperative fluids and the avoidance of nitrous oxide.

Diet/tablet-controlled diabetes

Oral hypoglycaemic therapy should be omitted on the morning of the surgery and the operation carried out as early as possible, with regular blood sugar checks. Some patients may require the use of an intravenous insulin/dextrose infusion to ensure better blood glucose control. Once the patient is eating and drinking properly they can restart their normal medication. Again, the use of regional anaesthesia and dual antiemetic therapy should be considered to speed up the return to good glycaemic control[10].

Metformin is stopped 2 days before major surgery because of the risk of lactic acidosis if a patient were to develop renal impairment post-operatively. Before restarting metformin post-operatively, it must be ensured that the patient's renal function has returned to normal or baseline. Other oral anti-diabetic medications, such as the short-acting sulfonylureas, are withheld on the morning of surgery.

Central Nervous System (CNS) medications

These consist of a widely used group of medications which have major medical and social importance and are used to treat conditions as diverse as epilepsy, anxiety, depression, schizophrenia and the neurodegenerative disorders of Parkinson's and Alzheimer's. Despite this their mechanism of action is for the most part not understood although modification of synaptic transmission is implicated.

The major drug groups used routinely are:

Benzodiazepines

These suppress the function of the CNS by potentiating the effects of GABA, an important inhibitory neurotransmitter, by increasing the receptor's affinity for the GABA molecule. All these compounds have hypnotic, anxiolytic, anticonvulsant, muscle relaxant, sedative and amnesic properties in varying degrees. Commonly used examples include temazepam, lorazepam and diazepam. Benzodiazepines are associated with tolerance and dependence. They are, however, relatively safe perioperatively, and for this reason and the danger of sudden withdrawal syndrome leading to an excitatory state associated with hypertension, agitation, delirium and seizures, they are usually continued[11].

Anti-epileptics

The pathophysiology of epilepsy is poorly understood: seizure involves the initiation of an abnormal impulse followed by propagation to neighbouring neurones. This disordered excitability is thought to be due to an imbalance between excitatory (glutamate) and inhibitory (GABA) neurotransmitter activity.

The mechanism of action for these compounds falls into one of three categories:

- enhancement of inhibitory (GABA) transmission;
- inhibition of excitatory (glutamate) transmission;
- stabilisation of neuronal membrane excitability.

The most commonly used anti-epileptics include phenytoin, carbamazepine and sodium valproate, all of which stabilise the neuronal membrane. The anti-epileptic properties of other CNS medications, especially barbiturates and benzodiazepines, can be attributed to their modification of central inhibitory and excitatory transmission[11].

Anticonvulsants should be taken on the day of surgery to reduce the likelihood of a seizure. Prolonged muscle contractions associated with seizure can affect the respiratory pattern, increase cellular oxygen demand and create a hypoxic state that can lead to an increased risk of cerebral dysfunction and damage. Other complications of seizures include rhabdomyolysis, precipitated renal failure, metabolic acidosis and increased extracellular potassium concentrations. These all reduce seizure thresholds and affect the likelihood and safety of surgery. Anticonvulsant therapy should be reinstated as soon as possible post-operatively[12]. If the patient is unable to take oral medications following surgery, advice may be sought from the pharmacist regarding alternative routes and doses of administration.

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Antidepressants

The monoamine theory of depression suggests that it is a functional deficit of monoamine (in particular noradrenaline and 5-HT) transmission in the CNS that leads to depression, thus common antidepressants work to address this imbalance. There are three different major groups of antidepressants:

- Tricyclic antidepressants non-selectively inhibit the neuronal reuptake of noradrenaline and 5-HT e.g. amitriptyline and clomipramine.
- Selective serotonin reuptake inhibitors (SSRIs) (e.g. fluoxetine and paroxetine).
- Monoamine oxidase inhibitors (MAOIs) prevent the breakdown of monoamine neurotransmitters increasing their availability (e.g. phenelzine)[11].

SSRIs are very safe in the preoperative setting and can be continued without concern. Tricyclic antidepressants may be continued but with caution. They inhibit the uptake of norepinephrine and serotonin and therefore may enhance the action of sympathomimetic drugs given during anaesthesia. Concomitant use of SSRIs or tricyclic antidepressants and other serotonergic agents, such as tramadol and pethidine, should be avoided if possible to avoid the risks of serotonin syndrome. MAOIs carry a potential risk of hypertensive crisis and a large number of associated drug interactions. Serotonin syndrome or respiratory depression may occur with concurrent pethidine and a hypertensive crisis with sympathomimetic drugs. The fact that MAOI inhibition lasts for 2 weeks means that withdrawal of the MAOI must occur at least 2 weeks prior to surgery, which increases the risk of psychiatric relapse. Therefore, advice from the psychiatric team should be sought. Alternatively, MAOIs may be continued with careful selection of anaesthetic agents.

Anti-mania drugs

While lithium may be continued for a minor surgery, it is recommended to discontinue lithium for 24 hours before any major operation[13]. Surgery-related electrolyte disturbances and reduced renal function may lead to lithium toxicity. Once electrolytes are normalised post-operatively, lithium should be reintroduced. Care should be taken with the use of diuretics so as not to cause dehydration. If possible, non-steroidal anti-inflammatory drugs (NSAID) should be avoided as they reduce the elimination of lithium. Serum lithium level may be monitored if necessary.

Neurodegenerative diseases

Diseases, such as Parkinson's and Alzheimer's, are characterised by a pathological and irreversible loss of CNS neurones. With the exception of Parkinson's there is very little, if any scope for pharmacological intervention. The degeneration and loss of inhibitory dopamine neurones lead to hyperactivity and thus the characteristic muscle rigidity, tremor, hypokinesia and postural instability of Parkinson's. Drug therapy aims to correct this imbalance by replacing dopamine (levodopa), direct activation of central dopamine receptors (bromocriptine), enhancing release of dopamine (amantadine) and reducing breakdown by inhibiting monamine oxidase (selegiline)[11]. Patients with significant Parkinson's disease who have major surgery will often need a multidisciplinary team consultation between pharmacy, neurology and anaesthesia as to a suitable plan for their drug treatment in the perioperative period. Anti-Parkinsonian medications are generally continued on the day of surgery. The half-life of levodopa is very short: autonomic dysfunction can result in intra-operative cardiovascular instability, and general anaesthesia can lead to increased post-operative confusion. Medications should be commenced as soon as possible after surgery. The neurologist and pharmacist may be able to advise on alternative drugs and routes of administration if the patient is unable to take oral medications. Rarely, in advanced disease, the use of apomorphine may be initiated under specialist supervision in patients who cannot tolerate oral or enteral medications. Medications to avoid include the antiemetic metoclopramide and the phenothiazines (e.g. prochlorperazine) which antagonise the effects of levodopa[12].

Hormones

Contraceptive pill

The spontaneous incidence of venous thromboembolism (VTE) in healthy, non-pregnant women not taking the oral contraceptive pill is 5:100,000. The risk is higher in women taking a combined oral contraceptive and depending on the hormonal components, the incidence rises to between 15 and 25:100,000. When this is combined with the effect surgery has on the promotion of VTE, by damaging veins, immobilisation and blood hypercoagulability, there is general advice that the hormone contraceptive pill should be discontinued 4 weeks before major surgery with appropriate advice regarding the need for additional contraceptive precautions. It should be restarted post-operatively at the first menses occurring at least 2 weeks after full mobilisation. In emergency surgery where it was not possible to discontinue the pill, thromboprophylaxis should be prescribed as per hospital policy. Recent evidence suggests that the progesterone-only pill need not be stopped[1, 15].

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Hormone Replacement Therapy (HRT)

For women undergoing major surgery who are at high risk of DVT or PE, the risks of continuing therapy may outweigh the benefits. In these cases, HRT should be discontinued four weeks prior to surgery. If this is not possible, thromboprophylaxis should be prescribed on admission.

Corticosteroids

Steroid therapy is another area which requires special consideration in the perioperative setting. Exogenous glucocorticoid administration leads to suppression and atrophy of the hypothalamic-pituitary-adrenal axis (HPA), and thus patients are unable to mount a sufficient physiological stress response. Hence, long-term steroid therapy should not be withdrawn abruptly. It has been shown that normal function returns rapidly in the majority of patients and the routine use of steroid cover is not required more than a few weeks after cessation of therapy. This is especially good considering several other concerns regarding the use of corticosteroids in surgical patients including adverse effects on wound healing, immunosupression, interaction with non-depolarising neuromuscular blocking agents and myopathy[16].

Many patients (e.g. those with asthma) are on long-term steroid treatment. Stress due to surgery is associated with an increased cortisol production. Therefore, patients at high risk of HPA suppression will have greater corticosteroid requirements. These include patients who have been taking ≥10mg daily of prednisolone (or equivalent) and patients undergoing bilateral adrenalectomy who will always require steroid cover, as well as patients with known adrenal insufficiency/hypopituitarism. Regimes for steroid replacement are often complex and with no national consensus document the Foundation doctor is advised to become familiar with any local guidelines where available. If concerned, the Foundation doctor can also seek advice from the endocrinology team.

Alternative and complementary medicines

In the UK studies have found that between 40–50% of surgical patients have used some form of complementary or alternative medicine with the overall majority having used a herbal medicine[17, 18]. As these are available over the counter without much advice or information, patients are less likely to view these as important enough to inform the doctor about them unless a specific history of herbal medication use is taken.

There are eight herbal medications available which have the potential to cause operative or post-operative complications and thus should be stopped in the preoperative period. Their effects are either due to their intrinsic pharmacological activity, pharmacodynamic or pharmacokinetic interactions[19], or their actions on platelets, the CNS, enzyme induction or immune modulation.

Echinacea

A member of the daisy family used for the prophylaxis and treatment of viral, bacterial and fungal infections. Studies have shown echinacea to have immunosuppressant effects. Expert opinion warns against concomitant use of echinacea in patients who require perioperative immunosupression (i.e. those awaiting organ transplantation). There is also a theoretical risk of poor wound healing and opportunistic infections because of this mechanism of action. There have also been reports of anaphylactic reactions to echinacea. The pharmacokinetics of echinacea are not very well known in the perioperative period, and it should be stopped as far in advance of surgery as possible (at least 2 weeks).

Ephedra (ma hung)

Derived from a shrub native to central Asia, it is used to promote weight loss, increase energy and also in the treatment respiratory conditions, such as asthma and bronchitis.

Ephedra has been found to cause a dose-dependent increase in both blood pressure and heart rate, caused by the active compound ephedrine, a non-catecholamine sympathomimetic agent present in ephedra. The sympathomimetic effects have been associated with fatal heart attacks, nervous system complications, vasoconstriction and vasospasm of coronary and cerebral arteries, and may cause MI or thrombotic stroke.

Ephedra has a short half-life in humans and thus stopping this medication can be left as late as 24 hours prior to surgery.

Garlic

Has the potential to modify the risk of atherosclerosis by reducing blood pressure, thrombus formation, and lowering serum lipid and cholesterol levels. As part of its mechanism of action garlic inhibits platelet aggregation in a dose-dependent fashion which is potentially irreversible. It may also potentiate the effects of conventional anti-platelet drugs. Due to its potential for causing irreversible inhibition of platelet function, it is recommended that garlic is discontinued 7 days prior to surgery.

Gingko

Is a derivative of the Ginkgo biloba leaf and is used for cognitive disorders. Studies suggest that ginkgo may stabilise or improve cognitive function in patients with Alzheimer's and multi-infarct dementia.

Gingko may increase the risk of bleeding perioperatively due to inhibition of platelet-activating factor. Although small clinical trials have not shown increased bleeding, there have been reports of spontaneous intracranial bleeding, spontaneous hyphema and one post-operative laparoscopic bleed.

Based on its associated risk of bleeding and its pharmacokinetic data, it is recommended that ginkgo is discontinued 36 hours prior to surgery.

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Asian Ginseng.
Good Clinical Care.

Ginseng

Both American and Asian ginseng are used for their "adaptogenic" effects. Ginseng is reputed to protect the body against stress and restore homeostasis. The many heterogeneous compounds act similarly to steroid hormones.

While it may be able to reduce blood pressure, it may also adversely lower blood glucose which may lead to hypoglycaemia, mainly in patients who have fasted. It may also cause a potential irreversible inhibition of platelet aggregation in vitro and in vivo leading to prolonged coagulation time.

These concerns warrant the need for ginseng to be stopped 7 days before surgery.

Kava

Derived from the dried root of the pepper plant piper methysticum, kava has gained popularity as an anxiolytic and sedative. A trial has shown that it may be of benefit in the symptomatic treatment of anxiety. The active components have dose-dependent effects on the CNS including antiepileptic, neuroprotective and local anaesthetic effects. These effects are thought to be potentiated via GABA receptors, the same receptors through which barbiturates have their action. Therefore kava potentiates the sedative effect of barbiturates.

Kava should be discontinued at least 2 weeks prior to surgery. It should be tapered gradually over several weeks to prevent withdrawal.

St John's Wort

Also known as Hypericum perforatum. it has shown efficacy in the short-term treatment of mild-to-moderate depression in a number of trials. The pharmacologically active compounds inhibit serotonin, noradrenaline and dopamine reuptake which could create a syndrome of serotonin excess.

However, the most widely documented and significant effect of St John's Wort is its effect on drug metabolism. It causes induction of the cytochrome P450 enzyme $3A^4$, which in turn increases the metabolism of other drugs, such as cyclosporine, midazolam and lidocaine, and reduces their plasma concentration and clinical effect. Other P450 enzymes induced include the $2C^9$ which, for instance, may affect the anticoagulant effect of warfarin. St John's Wort also affects digoxin pharmacokinetics.

Due to its pharmacokinetic data, this medication should be stopped at least 5 days prior to surgery.

Valerian

Is a herb used as a sedative particularly in insomnia and virtually all herbal sleep aids contain valerian. It produces its sedative and hypnotic effects in a dose- dependent manner, mediated through modulation of GABA neurotransmission. As previously discussed with kava, barbiturate sleep time is increased and thus valerian is expected to potentiate the sedative effects of anaesthetics.

The pharmacokinetics of valerian have not been studied but its effects are thought to be short-lived. Caution should be taken with abrupt discontinuation due to the risk of a benzodiazepine-like withdrawal. Thus, it may be prudent to taper the dose slowly over several weeks before surgery.

Other routes of administration

It is important to remember that many medications, for instance, digoxin and phenytoin, are available in preparations suitable for routes of administration other than the normal oral route. If it is essential that a patient continues on a certain medication either pre-, intra- or post-operatively, it would be of benefit to consult the BNF, hospital pharmacist or medicines information for advice on available preparations, suitable routes of administration, drug dosing and monitoring. Recommendations on alternative preparations and dosage adjustments of some specific medications belonging to the groups of drugs discussed in this article are covered to a limited degree in Table 2.

Conclusion

The advice given in this article draws on local guidelines and as new information is available and protocols change within hospitals, it is important for the Foundation doctor to be familiar with the local guidelines which apply to each different place of work.

In conclusion, the risk of losing disease control on stopping long-term therapies before surgery must be balanced with the risk posed by continuing them during surgery. It is vital that the anaesthetist knows all the drugs that the patient is taking, including over the counter medications. A Foundation doctor can contribute to good patient care and team working by ensuring that a careful medication history is taken, reviewing the routine medicines prior to surgery, raising any drug-related concerns and consulting other specialists when necessary for advice.

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Medication	Discussion	Recommendation
Cardiac		
Digoxin	Withdrawal can lead to fast AF and its associated increased risk of clots, MI and hypotension. Concurrent use with suxamethonium can precipitate arrhythmias.	Continue. Liquid or IV preparation available. Dose reduction of 33% is recommended when changing from oral to IV route. Although in theory a dose reduction is required when switching from tablet to liquid preparation, in practice it is unlikely to be clinically important. Monitor digoxin level (at least 6-hours post-dose) following any administration changes or if digoxin toxicity suspected or therapeutic efficacy is in doubt.
Amiodarone	Omitting may be associated with recurrence of rhythm abnormality.	Continue. IV amiodarone may be necessary if oral/enteral therapy is not possible for a significant period of time (e.g. 1 week). Cardiac monitoring facilities must be available during intravenous administration.
_-blockers	Stopping treatment can lead to withdrawal effects, such as nervousness, tachycardia and exacerbation of myocardial ischaemia, even sudden death. There is good evidence these medications reduce perioperative morbidity. Increased hypotensive effect when given with general anaesthetics.	Continue. Restart when blood pressure (BP) and heart rate normalise post-operatively. If oral/enteral administration not possible post-operatively, IV preparation (e.g. IV atenolol) may be necessary. Cardiac monitoring is required during IV administration.
ACE Inhibitors (ACEIs	Can cause profound hypotension on induction of anaesthesia and reduced tolerance of hypotension. Carries risk of causing or worsening renal impairment post-operatively in vulnerable patients.	Stop 48 hours before surgery. Re-introduce when BP and U&E's normalise post-surgery. Ensure good urine output and renal function before reinitiating. May require restarting at lower dose and titrating dose according to BP if patient has missed their ACEI for a prolonged period of time
Thiazide/loop diuretics	May cause electrolyte abnormalities. Enhanced hypotensive effect when given with general anaesthetics.	Generally continued. Correct any electrolyte imbalances prior to surgery
Potassium sparing diuretics (e.g. spironolactone, amiloride)	Should be omitted on the morning of surgery on the basis that tissue damage and reduced kidney perfusion post-operatively can lead to hyperkalaemia. Enhanced hypotensive effect when given with general anaesthetics.	Omit on morning of surgery. Monitor fluid balance, urine output and U&E's.
Antiplatelets and anticoagulants		
Aspirin	Causes irreversible platelet inhibition. No absolute consensus about whether or not aspirin should be continued perioperatively, Assess the risk of haemorrhage versus the risk of thromboembolism.	Continue or stop 7 to 10 days preoperatively depending on the type of surgery to be undertaken and the risks of haemorrhage versus risks of thromboembolism. If stopped preoperatively, restart when risk of bleeding no longer significant. If not allowed oral/enteral administration post-operatively, aspirin suppositories may be considered. Note that rectal absorption is less reliable.
Clopidogrel	Irreversible platelet inhibition that has been shown to increase the risk of haemorrhage by 50% if given with aspirin.	Stop 5 to 7 days before surgery. In patients with cardiac stents, it may be indicated to continue. Decision to stop clopidogrel should be discussed with cardiologist. If stopped preoperatively, restart when risk of bleeding no longer significant.
Warfarin	Increased risk of bleeding needs to be balanced against the underlying reason for treatment and increased risk of a thromboembolic event. Some patients will require replacement of warfarin with either prophylactic doses of a LMWH or unfractionated heparin.	Stop 4 days before surgery. Post-operatively, restart only once haemostasis is secure and further surgical procedures (such as wound debridement, epidural catheter, chest drain removal) are not imminent.
Diabetic		
Insulin	Depending on the type of surgery and glucose control, patients may be managed differently.	Refer to local guidelines. Seek advice from diabetes team if required.
Metformin	Carries a risk of lactic acidosis if patients develop renal problems.	Stop 2 days before surgery. Restart post-operatively when patient is eating and drinking normally and renal function normal.
CNS medications		
Anti-Parkinsonian drugs	All medications should be continued to reduce autonomic dysfunction.	Continue. Enteral administration – Madopar dispersible tablets available; Sinemet tablets can be dispersed in water. For patients on modified-release preparations, convert to dispersible tablets and increase dosing frequency. Consult pharmacist if unsure about dose conversion. Monitor for change in clinical effect. Cabergoline and entacapone may be dispersed in water for enteral administration. Selegiline liquid preparation available. Rarely, in severe cases when patient is unable to take medications orally/enterally, IV apomorphine may be indicated under specialist recommendation and care.
Anti-epileptics	Should not be stopped to reduce the likelihood of seizure and the added complications this causes. It is important intra-operatively to avoid hypocapnia and pro-convulsant drugs, such as ketamine.	Continue. Enteral administration – use liquid preparation where possible. Phenytoin, carbamazepine, sodium valproate liquid preparations available. Suspension of phenytoin 90mg in 15ml approximately equivalent in therapeutic effect to tablets or capsules containing phenytoin sodium 100mg, but care is needed in making changes. Modified-release preparations NOT suitable for enteral administration, therefore convert to liquid or crushable tablets and give in divided doses. When oral administration not possible; Phenytoin – IV preparation available (IV = oral dose) Give in divided doses. Monitor plasma phenytoin level. Carbamazepine – convert to rectal preparation (suppositories 125mg = tablets 100mg) Monitor response and plasma carbamazepine level. Sodium Valproate – IV preparation available (IV = oral dose)
MAOIs	Carry the risk of precipitating a hypertensive crisis and a large number of drug–drug interactions, such as with sympathomimetic agents.	Seek advice from psychiatric team whether to continue or not. If need to discontinue, withdraw at least 2-weeks preoperatively.
SSRIs	Are very safe. Cessation may result in withdrawal syndrome particularly of short-acting SSRIs, such as paroxetine.	Continue.
Tricyclic antidepressants	Inhibit uptake of norepinephrine and serotonin, thus may enhance the action of sympathomimetics.	Continue with caution.
Lithium	Surgery-related electrolyte disturbances and reduced renal function can lead to lithium toxicity.	Depends on the type of surgery: - Minor surgery – continue. - Major surgery - stop 24 hours before surgery. Post-operatively, restart once electrolytes normalise. Enteral administration – convert from lithium carbonate (tablets) to lithium citrate (liquid). Dose conversion: lithium carbonate 200mg = lithium citrate 509mg

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Hormones		
Steroids	Some patients require steroid cover or an increase in their steroid therapy perioperatively for disease maintenance and post-operative requirements. These include patients who undergo adrenalectomy, have adrenal insufficiency and are at risk of hypothalamo-pituitary-adrenal axis suppression. See advice from endocrinology if necessary.	Discussion with relevant team required.
Combined oral Contraceptive pill	Increased risk of VTE of between 15–25:100,000.	Minor surgery – continue. Major surgery – stop 4 weeks prior to surgery and advise on alternative contraception methods. Restart post-operatively at first menses occurring at least 2 weeks after full mobilisation. If this is not possible, thromboprophylaxis should be prescribed on admission.
Progesterone-only pill	No increased risk of VTE.	Continue.
HRT	Women undergoing major surgery who are at high risk of DVT or PE, the risks of continuing therapy may outweigh the benefits.	Minor surgery – continue. Major surgery – stop 4 weeks prior to surgery and advise on alternative contraception methods. Restart post-operatively at first menses occurring at least 2 weeks after full mobilisation. If this is not possible, thromboprophylaxis should be prescribed on admission.
Herbal		
Echinacea	May cause immune suppression, thus a theoretical risk of poor wound healing and opportunistic infections.	Stop as far in advance of surgery as possible (at least 2 weeks).
Ephedra	Has a sympathomimetic effect. May cause vasoconstriction of coronary and cerebral vasculature. Risk of ventricular arrhythmias with halothane.	Discontinue 24 hours before surgery.
Garlic	Inhibits platelet aggregation in dose-dependent fashion, which may be irreversible.	Stop at least 7 days before surgery.
Gingko Biloba	Inhibition of platelet activating factor, hence increased risk of bleeding.	Discontinue at least 36 hours prior to surgery.
Ginseng	May contribute to hypoglycaemia especially in fasted patients. Possible irreversible inhibition of platelet aggregation.	Stop at least 7 days before surgery.
Kava	Potentiation of the sedative effects of anaesthetics.	Stop at least 2 weeks before surgery. It should be tapered gradually over several weeks to prevent withdrawal.
St John's Wort	Induction of cytochrome P450 enzymes 3A4 and 2C9. Therefore possibly interacts with drugs, such as warfarin, cyclosporine, lidocaine, midazolam, alfentanil.	Discontinue at least 5 days before surgery.
Valerian	Potentiates the sedative effects of anaesthetics. Abrupt withdrawal may cause withdrawal symptoms.	Taper dose slowly over several weeks before surgery.
Lithium	Surgery-related electrolyte disturbances and reduced renal function can lead to lithium toxicity.	Depends on the type of surgery: - Minor surgery – continue. - Major surgery – stop 24 hours before surgery. Post-operatively, restart once electrolytes normalise. Enteral administration – convert from lithium carbonate (tablets) to lithium citrate (liquid). Dose conversion: lithium carbonate 200mg = lithium citrate 509mg.

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POST-OPERATIVE COMPLICATIONS

Cavin Gray

Post-operative complications.
Patient Management.

Worldwide it is estimated that approximately 200 to 250 million surgical procedures are performed each year. Many of these patients will suffer from one or more post-operative complications. Definitions of what constitutes a post-operative complication vary but the term generally suggests an adverse occurrence affecting a patient's well-being, which may at the very least delay recovery and in the worst cases result in lasting disability or death. Because of the difficulties in both defining post-operative complications and in gathering data, it is difficult to provide an accurate incidence of their occurrence but some studies have suggested figures as high as 30% or more. Post-operative complications may arise as a direct consequence of the surgical procedure or anaesthesia, or be related to underlying medical conditions and while not all will be serious or life-threatening even those which are mild and self-limiting may, nonetheless, be distressing to patients and since they may require treatment and prolong hospital stay, they may have significant cost implications.

Classification

Post-operative complications may be classified in a number of ways. For example, this could be in relation to the nature of the problem, its severity or its timing. With regard to the timing of post-operative complications, they can be regarded as immediate, early or late.

Immediate	Early	Late
Primary haemorrhage	Secondary haemorrhage	Incisional hernia
Respiratory insufficiency	Respiratory infection	Failure of prostheses
Cardiovascular problems (e.g. infarction or arrhythmias)	Wound infection	Chronic infection
Inadequate fluid replacement	Confusion	Recurrence of original disease
	Nausea and vomiting	
	Pain	

Table 1: Post-operative complications.

Management

When faced with a post-operative complication its precise nature may be established by means of history taking, examination and the use of appropriate investigations, coupled to an awareness of particular problems likely to be encountered in such a setting. It is of particular importance that complications, which are life-threatening or may result in significant morbidity, are identified and treated early. This may particularly apply in cases of haemorrhage or cardiorespiratory insufficiency. As in many other acute settings this requires particular attention to the key areas of airway, breathing and circulation. Many hospitals now use scoring systems based on key clinical observations which are designed to detect such problems at an early stage and allow appropriate management to be initiated at an early enough point to minimise adverse outcomes. Some problems may be amenable to relatively simple treatment but in the case of serious complications further surgical intervention, critical care or other specialist support may be necessary. It may be best to illustrate some of these issues by means of some commonly occurring clinical scenarios.

Scenario 1

A 52-year-old man who has undergone a nephrectomy under general anaesthesia with epidural analgesia, is noted 30 minutes after arrival in the recovery ward to be hypotensive with a blood pressure of 85/50. He is tachycardic, with a heart rate of 110 and on questioning admits to feeling faint. The recovery nurse has given two 250ml boluses of 0.9% saline solution, which resulted in a modest but unsustained rise in blood pressure on each occasion.

There are a number of possible explanations for this patient's clinical condition. The combination of hypotension and tachycardia is strongly suggestive of hypovolaemia that may be due to inadequate volume replacement preoperatively or ongoing blood loss. It is possible that even though haemostasis appeared adequate at the time of surgery the return to a more normal blood pressure following recovery from anaesthesia may reveal further bleeding. However, hypotension is a common side effect of epidural analgesia, and a perioperative myocardial infarction or even an anaphylactic reaction could present a similar picture.

Initial management will require assessing the response to further volume resuscitation and where time permits measuring the patient's haemoglobin and performing a 12 lead ECG. Further clues as to the patient's volume status may be given by evidence of peripheral shutdown and reduced urine output. The brief, unsustained response to fluid boluses in this setting is strongly suggestive of ongoing blood loss and if the patient remains unstable with no other obvious explanation an urgent return to theatre for further surgical exploration and control of any persistent bleeding is indicated.

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Scenario 2

A 78-year-old woman who underwent total hip replacement surgery under general anaesthesia 4-days previously, is noted to have an oxygen saturation of 91% breathing air which rises to 95% breathing oxygen via nasal cannulae at 4 litres/ minute. She has a blood pressure of 115/60, a heart rate of 105/minute and a respiratory rate of 20/minute. Her temperature is 37.3^{0}C.

Likely causes of this clinical picture at this time include pulmonary embolism, respiratory infection and myocardial infarction. Features in the history which might give some indication of the cause include dyspnoea, pleuritic chest pain, productive cough, haemoptysis, cardiac type chest pain or calf pain. Although these features may give useful diagnostic clues, however, their absence does not rule out significant pathology. Clinical examination may yield further clues and should include auscultation of the heart and lung fields and examination of the legs to look for signs of possible deep venous thrombosis. Initial investigations should include chest X-ray, ECG, full blood count, assay of cardiac enzymes and arterial blood gas analysis. Where pulmonary embolism is suspected the British Thoracic Society guidelines recommend that a clinical probability scoring system be used in conjunction with a D-dimer assay[1]. If imaging is indicated the technique of choice where it is available is computed tomographic pulmonary angiography (CTPA). Other techniques which may give diagnostic information include a cardiac echo to demonstrate right heart strain in patients too unstable for a CTPA or ventilation/perfusion scanning and leg ultrasound to demonstrate the presence of deep venous thrombosis. Treatment of pulmonary embolism is by a combination of supportive treatment and anticoagulation. Supportive treatment is with oxygen therapy and support of the circulation with fluid and inotropes if required. Anticoagulant therapy usually involves heparin (low molecular weight or unfractionated) followed by oral therapy using warfarin. In cases of massive pulmonary embolism thrombolysis may be considered.

Scenario 3

An 87-year-old woman who underwent uncomplicated hemiarthroplasty surgery for a fractured neck of femur 24-hours ago under spinal anaesthesia with midazolam sedation, is becoming increasingly agitated. She appears disorientated in time and place and is reluctant to allow nursing staff to carry out basic care. She makes repeated attempts to remove intravenous cannulae and does not tolerate her oxygen mask. Her blood pressure is 120/70, heart rate 90, oxygen saturation 90% on air and temperature of 37.6^{0}C.

Post-operative cognitive dysfunction is a common and difficult problem. The precise aetiology remains unclear but there is a strong association with increasing age, with some studies suggesting that approximately one third of patients over the age of 80 who undergo a surgical procedure will demonstrate some degree of cognitive impairment in the post-operative period[2]. While usually temporary, a significant number of patients will still demonstrate some degree of impairment 1 year later. Other risk factors which have been postulated include perioperative hypoxaemia or hypotension and prolonged exposure to deep levels of anaesthesia. Although the cause is frequently unclear there are a number of important remediable causes which should be investigated and treated if necessary.

Infection may well present in this manner, and in this patient the possibility of respiratory tract, urinary tract or wound infection should be considered. An appropriate history should be taken and specimens of urine, sputum and wound swabs if indicated should be obtained for microbiological analysis. The patient's white blood cell count may assist in making a diagnosis and treatment may be initiated guided by local antibiotic policy.

Hypoxaemia may also contribute to confusion and pulse oximetry, possibly with arterial blood gas analysis in addition, should be employed, with oxygen therapy initiated if required. Electrolyte disturbances may also be implicated and serum urea and electrolytes should also be measured.

There is a considerable temptation to prescribe sedative drugs to these agitated patients but there is a strong possibility that they might actually exacerbate confusional states and careful thought should be given to any such decision, particularly if there is any suggestion that impaired respiratory dysfunction may be contributing to the confusional state since this is likely to be made worse by most common sedative drugs. In general it is preferable to treat the cause of the confusion where this can be established or to increase the level of nursing input, if necessary by such measures as moving the patient to a location near the nursing station.

Conclusion

While these scenarios cover only a few of the potential post-operative complications likely to be encountered, they demonstrate many of the principles involved in their management and lay particular emphasis on the importance of early detection and management of serious, potentially life-threatening complications using a systematic method of assessment combined with a knowledge of the likely differentials.

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SODIUM IMBALANCES FOR THE FOUNDATION DOCTOR

Thomas Cannon, Tracie Plant and Miguel Debono

One of the most common and sometimes complicated electrolyte abnormalities encountered in hospital patients are sodium imbalances.
Patient Management.

Introduction

One of the most common and sometimes complicated electrolyte abnormalities encountered in hospital patients are sodium imbalances. Unlike the management of other electrolyte imbalances, which are guided by specific well-established guidelines, sodium disorders are often not managed as well. This is because of the larger array of causes and aspects of management that can be employed. It is hoped that this article will go some way to aiding Foundation doctors in their understanding of abnormalities of serum sodium levels and how to approach their investigation and management.

Sodium regulation

The vast majority of sodium in the body is extracellular and is maintained within a range of 135–145mmol/L. A typical daily intake of sodium is between 100–300mmol, the majority of which is absorbed from the gastrointestinal tract. Sodium is excreted through the kidneys and by sweating.

Sodium, along with the main anions (chloride and bicarbonate) and glucose, maintain a serum osmolality of 285–295mosm/kg[1] and is calculated as: 2x[Na]mmol/L + [urea]mmol/L + [glucose]mmol/L[1].

A rise in the serum osmolality triggers the release of antidiuretic hormone (ADH) and the stimulation of thirst. A lowering of the osmolality causes larger amounts of dilute urine to be formed. Aldosterone also plays a role in the regulation of sodium balance through its effects in the distal convoluted tubule and the distal colon on the Na/K ATPase (sodium pump)[2], stimulating sodium absorption.

Hyponatraemia

Hyponatraemia can be classified as a serum sodium level of less than 135 mmol/L[3]. It is the most common electrolyte abnormality encountered in clinical practice, with a reported mild hyponatraemia (130–135mmol/L) incidence of 15–30% seen in hospitalised patients and of 1–4% for more severe hyponatraemia (<130mmol/L)[4].

The main causes we have encountered in hospital practice are contained in Table 1.

Main Hospital Encountered Causes Of Hyponatraemia
Incorrect intravenous fluid administration
Heart failure
Drug causes
Poor oral fluid intake
Gastrointestinal losses

Table 1: Main hospital encountered causes of hyponatraemia.

Generally, a patient with a serum sodium of 130–135mmol/L will have no symptoms, with symptoms increasing in severity as sodium falls further. Patients can remain asymptomatic with low sodium especially if this is chronic. An acute presentation of hyponatraemia is defined as hyponatraemia occurring within 48 hours and requires prompt treatment[1, 3]. Symptoms seen include anorexia, lethargy, headache, muscle cramps, agitation, reduced GCS and seizures[3]. Cerebral oedema can occur and if left untreated, hyponatraemia can be fatal.

History and examination

Once it has been established that a patient has low sodium, it is necessary to find the cause. The initial mainstay of this is through a thorough history and examination. Primarily in hospital patients, one should ask about gastrointestinal (GI) losses, such as diarrhoea and vomiting, symptoms of any current infection especially chest related, medication use, recent head injuries/surgery and symptoms indicating fluid overload, such as shortness of breath, orthopnoea and lower limb swelling. Eliciting symptoms that may point towards an undiagnosed malignancy can also go some way to explaining the hyponatraemia. A full systemic enquiry should be performed when the cause is not obvious.

The fluid status of the patient should be thoroughly examined. Signs of hypovolaemia include tachycardia, low blood pressure, postural drop, reduced skin turgor, reduced or absent jugular venous pressure (JVP), poor urine output and dry mucous membranes. A patient that is overloaded may appear oedematous with a raised JVP, have a gallop rhythm on cardiac auscultation, bilateral fine basal crepitations on chest examination and may appear short of breath. Congestive cardiac failure, liver disease and nephrotic syndrome may cause the patient to be fluid overloaded. The nephrotic syndrome is a triad of urinary protein loss, hypoalbuminaemia and oedema.

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Signs of underlying disease should be looked for in the examination, for example, stigmata of underlying liver pathology (such as spider naevi, jaundice, ascites and a palpable liver). Signs of hypothyroidism may include bradycardia, a palpable goitre, proximal muscle weakness and dry skin. Signs of Addison's disease, such as postural drop and increased skin pigmentation should also be looked for. Key points of note in the examination are summarised in Table 2.

The Examination	
Fluid status	Signs of underlying co-morbidities
Fluid balance chart	Congestive cardiac failure
Pulse and blood pressure	Stigmata of liver disease
Jugular venous pressure	Nephrotic syndrome
Skin turgor	Signs of hypothyroid
Capillary refill time	Signs of Addison's disease
Mucous membranes	Signs of malignant disease
Signs of oedema	

Table 2: The examination of a hyponatraemic patient[1].

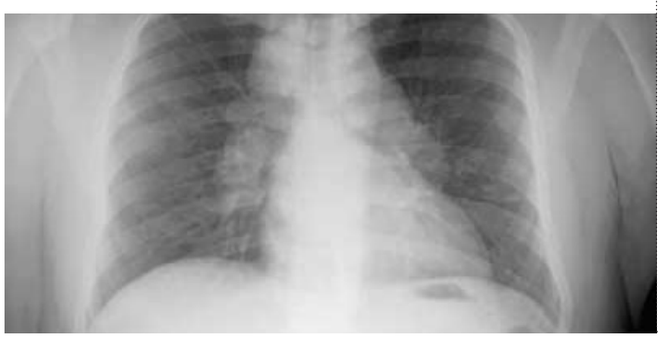
Investigation

There are a number of investigations necessary to undertake before diagnosing the cause of the hyponatraemia. The blood tests will not only establish the extent of the hyponatraemia but will give an indication of the hydration status and may identify a possible cause if there is an underlying endocrine abnormality.

When requesting biochemical tests on the urine, it is important that the urine is sent for examination on the same day and at the same time as the serum samples in order that the paired results can be interpreted accurately.

A simple chest radiograph may reveal a host of clinically relevant findings. Signs of consolidation may indicate an infective cause. An enlarged heart or signs of fluid overload may point towards congestive cardiac failure. An obvious mass may suggest a lung neoplasm. A simple urine dipstick may reveal signs of a urinary tract infection. A CT head may be indicated if there is evidence in the history to suggest a recent head injury or there are symptoms or signs of focal neurology.

The tests summarised in Table 3 are essential first line investigations.



Investigations for hyponatraemia		
Blood tests	Urine tests	Imaging tests
Urea and electrolytes	Urine osmolality	Chest X-ray
Liver function tests	Urine sodium	Consider CT head
Serum osmolality	Dipstick urine	
Thyroid function tests		
Random cortisol		
Serum glucose		

Table 3: The investigations of a hyponatraemic patient.

Diagnosis

The essential pieces of information necessary to determine the potential cause of the patient's hyponatraemia are their fluid status and their urinary sodium level. These can then be interpreted together in order to give a list of differential diagnoses. This is summarised in Table 4.

Fluid status Urine sodium	Underfilled	Normal	Overfilled
<30mmol/L	GI losses Insensible losses Pancreatitis Burns	Decreased sodium intake	Congestive cardiac failure Cirrhosis with ascites Nephrotic syndrome
>30mmol/L	Renal loss (e.g. diuretics, salt-wasting nephropathy)	Syndrome of inappropriate ADH* Hypothyroidism Post-transurethral resection syndrome	Acute and chronic renal failure

Table 4: Interpretation of the investigation results in order to establish the cause. * Syndrome of Inappropriate ADH (SIADH) is a diagnosis of exclusion[1].

Interpretation of the serum osmolality further assists with diagnosis. Hyponatraemia with a normal serum osmolality may be caused by pseudohyponatraemia, which is the presence of an increased number of large molecules relative to sodium in the serum. The post-transurethral resection syndrome is characterised by hyponatraemia, neurological deficits and cardiorespiratory compromise[5]. Hyponatraemia in the presence of a raised serum osmolality may be caused by high levels of glucose in the serum in conditions such as diabetic ketoacidosis and hyperosmolar non-ketotic coma[5]. A reduced plasma osmolality can be seen in both hypovolaemic and euvolaemic states.

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It is important not to forget the common drug causes of low sodium.
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It is important not to forget the common drug causes of low sodium. The main drugs commonly encountered that can cause hyponatraemia are thiazide and loop diuretics, proton pump inhibitors, selective serotonin reuptake inhibitors, mirtazapine, sulphonylureas, haloperidol, heparin and carbamazepine[3]. This is by no means an exhaustive list and product literature should be consulted for any drugs for which there is doubt.

Syndrome of Inappropriate ADH (SIADH) is a diagnosis of exclusion and is dependent upon the presence of a serum osmolality <270mosm/kg and a raised urinary sodium of >30mmol/L which will contribute to an inappropriately high urine osmolality >100mosm/kg. This diagnosis cannot be made until glucocorticoid deficiency and hypothyroidism have been excluded[1].

SIADH can be caused by all the previously listed drugs (except diuretics), head trauma, malignancies, pulmonary diseases, infections, human immunodeficiency virus (HIV) and post-operative pain[6]. Common malignancies that can cause SIADH through the ectopic production of ADH include small-cell lung cancers, duodenal cancers and pancreatic cancers[2]. It is estimated that up to 35% of patients with HIV have SIADH, with the most common causes being Pneumocystis carinii pneumonia, central nervous system infections and malignancies[2].

Management

The management of low sodium depends on the diagnosed cause and the duration of the hyponatraemia. Hyponatraemia in patients with a normal fluid status is that which is most commonly encountered in hospitalised patients[6, 7].

Hyponatraemia developing over a period <48 hours is defined as acute onset and carries the greatest risk for the development of cerebral oedema[1, 3]. For the patient with acute symptomatic hyponatraemia the initial mainstay of treatment involves supportive treatment and rapid termination of seizure activity[3]. It is appropriate in this setting to involve specialised staff at an early stage. Administration of any fluids to correct hyponatraemia should be carefully considered. Any hypotonic solutions administered will only exacerbate the degree of cerebral oedema. The rate of correction of hyponatraemia is a controversial subject, with a poor evidence base for the use of hypertonic saline[1, 6]. It is, however, advised that the total correction of serum sodium levels should not exceed 8mmol/L per day and that the aim should be to increase the level to a safe range and not to normalise serum sodium[4]. Infusion of 300ml of 3% or 600ml of 1.8% hypertonic saline over 6 hours is a good initial starting point for the treatment of acute hyponatraemia [3].

For acute symptomatic hyponatraemia, the sodium deficit (in mmol) can be calculated by multiplying the total body water (TBW) by the desired sodium concentration minus the actual sodium concentration as follows:

Sodium deficit = TBW x (desired sodium – actual sodium)[3]
(TBW = body weight x 0.6 for males or 0.5 for females)

This formula aids in the calculation of the replacement fluid to be given.

Hyponatraemia attributed to endocrine causes should be appropriately managed with hormone replacement therapy, which will require specialist input[5]. Drugs which are attributed to the hyponatraemia should be omitted if possible or substituted with suitable alternatives.

In contrast to acute hyponatraemia, patients with chronic hyponatraemia are generally asymptomatic[6]. Removal of the precipitant is essential in order to allow serum sodium levels to gradually correct. Fluid restriction is, however, advised should the patient become symptomatic. It is recommended that an initial fluid restriction of 1 litre/day for a serum sodium <130mmol/L is instituted[1]. There are, however, no hard or fast rules on fluid restriction; senior medical advice should be sought.

Hyponatraemia – case report

We have encountered in clinical practice an interesting case of hyponatraemia for which the cause remained unclear for a period of time.

An 82-year-old gentleman was noted to have mild hyponatraemia with a baseline serum sodium of between 130–135mmol/L. Repeat serum sodium showed that this value had dropped to 123mmol/L without any obvious precipitating cause. Paired with this were normal thyroid function tests and a normal random cortisol. His serum osmolality was normal at 288mosm/kg, urine osmolality was 289mosm/kg and urinary sodium was 57mmol/L. This is not technically SIADH, as the serum osmolality was normal. Clinical examination revealed no obvious signs of infection or fluid overload.

Examination of the medication list revealed omeprazole 20mg daily and furosemide 40mg daily as possible drug causes. Fluid restriction was trialled at 1 litre per day, along with omitting these possible contributory drugs.

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Repeat serum sodium levels showed no real improvement, with values remaining in the range of 123–124mmol/L. However, a further repeat a few days later showed a serum sodium of 115mmol/L, serum osmolality of 258mosm/kg, urine sodium of 15mmol/L and urine osmolality of 192mosm/kg. Endocrine advice was sought, and on further questioning of the patient, a history of chronic loose stools was elicited. It was also apparent that the patient was exceeding his fluid restriction. Examination of the medication list again highlighted a recent new drug as a possible contributing cause of the low sodium; donepezil 5mg once daily. Hyponatraemia is not a listed side effect in the British National Formulary (BNF) for donepezil, though diarrhoea is. There have, however, been published case reports of donepezil as a cause of hyponatraemia.

By removing donepezil and starting slow sodium initially 4 tablets per day (to compensate for possible chronic losses in the loose stools), a rise was seen in the serum sodium levels. The serum sodium returned to 126mmol/L in 2 days, returning to within normal limits within 2 weeks.

This case highlights the picture so often seen in hyponatraemic patients of multiple aetiologies potentially causing the drop in serum sodium. It is often only with careful thought and analysis combined with repeated reviews of the results of any changes that the abnormality can be rectified.

Hypernatraemia

Hypernatraemia is a much less common electrolyte imbalance than hyponatraemia[2]. Hyperosmolality occurs through either a gain in sodium or a net water loss[1]. In ill patients the protective thirst mechanism is often impaired or absent, thus such individuals are more susceptible to hypernatraemia. Symptoms are not usually evident until the serum osmolality rises above 158–160mmol/L[1]. The symptoms of significantly raised serum sodium are generally non-specific and are very similar to those seen in low sodium states. The initial symptoms are anorexia, muscle weakness, nausea and vomiting. Later the patient may develop life-threatening symptoms of altered GCS and coma; shrinkage of brain tissue may cause acute bleeding resulting in subarachnoid haemorrhages.

History and examination

This is much the same as for hyponatraemia and involves an accurate assessment of the patient's fluid status. The cause is often apparent from the history[1].

Hypernatraemia can be classified according to the fluid status of the patient. This is summarised in Table 3. Diabetes insipidus causes hypernatraemia with a hyperosmolar serum where the urine is inappropriately dilute and can be either nephrogenic or central in origin[7]. Patients with diabetes insipidus generally present with both polyuria and polydipsia[1]. Nephrogenic diabetes insipidus occurs through resistance of the renal tubules to ADH whereas central diabetes insipidus occurs because of a deficiency in the production of ADH in the hypothalamus. A water deprivation test can be used to differentiate between central and nephrogenic diabetes insipidus[1]. In most cases the thirst response attempts to correct the hyperosmolar state[7].

Classification of hypernatraemia		
Underfilled	**Normal**	**Overfilled**
Burns and sweating	Diabetes insipidus	Iatrogenic
GI losses	Fever	Hyperaldosteronism
Diuretic usage	Hyperventilation	
Renal failure		
Bowel obstruction		

Table 5: Classification of hypernatraemia in the context of the patient's fluid status[1].

Investigation

It is important to obtain measurements of both serum and urine osmolalities on the same day if diabetes insipidus is suspected. Serum osmolality tends to be above 295mosm/kg in central diabetes insipidus[2]. This may be followed with a water deprivation test and measurement of serum vasopressin[2]. Measurement of total urine output over 24 hours to confirm polyuria is also essential prior to starting any investigations. Furthermore, if central diabetes insipidus is queried after a water deprivation test, brain imaging to identify space-occupying hypothalamic lesions may be undertaken following specialist advice[7]. It is estimated that approximately 50% of cases of central diabetes insipidus are idiopathic in origin[2].

Management

If hypernatraemia develops over a relatively short period the correction of sodium balance can prevent neurological sequelae, such as seizures and/or coma. It is advocated that acute hypernatraemia is managed with the careful administration of 5% dextrose, whereas chronic hypernatraemia is managed with oral water. However, 0.45% saline should be used in patients unable to take oral fluids[1]. Calculation of the free water deficit can be used to estimate the fluid required to be infused over 48 hours for a patient that is fluid deplete[8]. The formula is shown below.

Free Water Deficit = Body Weight (kg)
X Percentage of Total Body Water (TBW)
X ([Serum [Na+] / 140] - 1)

TBW is as follows:
0.6 for a young man
0.5 for a young woman or old man
0.4 for an old woman

It is important to add estimated insensible losses to the free water deficit calculated in the above formula to obtain an accurate estimation of the volume of water to be replaced.

Table 6: The calculation of free water deficit[8].

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Central diabetes insipidus should be managed with desmopressin treatment and nephrogenic diabetes insipidus should be managed by the removal of precipitant drugs[1].
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Central diabetes insipidus should be managed with desmopressin treatment and nephrogenic diabetes insipidus should be managed by the removal of precipitant drugs[1]. Specialist advice should be sought in the further management of any patient with diabetes insipidus.

Where to turn for help

The initial ports of call for help are your seniors. The medical registrar on-call will be able to offer further advice and review the patient if necessary, as will the endocrine registrar on-call. There may also be published protocols where you work to help you begin to investigate the sodium abnormality. Do not forget to ask for advice and assistance from hospital pharmacy services and the internet can be a further source of information.

Useful websites include:

www.endocrinology.org
access to free review articles.

www.bmj.com
access to articles from the BMJ group.

www.acutemed.co.uk
free online guidance for a number of common medical conditions.

http://emedicine.medscape.com
comprehensive medical and surgical advice from America.

It is important to bear in mind that some internet sources may be inaccurate and provenance should be checked.

Summary

Disorders of sodium balance are common among hospital inpatients and it is generally the job of the Foundation doctor to identify and initiate treatment. Both hypo- and hypernatraemia can be poorly understood and perceived as difficult to manage, highlighting a deficit in knowledge of this topic. While this article is only a brief overview of the area, it is hoped that it will provide an easily accessible initial reference point.

Self-assessment

The following self-assessment questions are designed to help you understand the topic more fully. Not all of the answers are in the article, so some further reading may be necessary.

Multiple choice questions

1. Where in the kidney does most of the re-absorption of sodium take place?
a. The proximal tubule.
b. The loop of Henle.
c. The distal tubule.
d. The collecting system.

2. Which of the following medications can cause hyponatraemia as a side effect?
a. Furosemide.
b. Spironolactone.
c. Paracetamol.
d. Omeprazole.
e. Lithium.

3. In a typical male adult, what percentage of his total body weight is water?
a. 40%.
b. 50%.
c. 60%.
d. 70%.

4. Which one of the following scenarios would be consistent with a diagnosis of SIADH?
a. Urinary sodium of 15mmol/L, urine osmolality of 115mosm/kg, serum osmolality of 290mosm/kg.
b. Urinary sodium of 50mmol/L, urine osmolality of 210mosm/kg, serum osmolality of 265mosm/kg.
c. Urinary sodium of 35mmol/L, urine osmolality of 165mosm/kg, serum osmolality of 288mosm/kg.
d. Urinary sodium of 18mmol/L, urine osmolality of 120mosm/kg, serum osmolality of 270mosm/kg.

SODIUM IMBALANCES FOR THE FOUNDATION DOCTOR

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5. What should be your initial action in the assessment of a patient with a decreased level of consciousness that you feel is caused by high serum sodium?

a. Cannulate and give intravenous 5% dextrose fluid.
b. Assess their airway.
c. Assess their breathing.
d. Assess their circulation.
e. Assess their fluid status.
f. Organise a water deprivation test.

Answers

1. a.
The proximal tubule re-absorbs approximately 2/3 of sodium.

2. a. b. d.
All drugs should be examined in the British National Formulary for side effects affecting sodium balance. Furosemide, spironolactone and omeprazole have hyponatraemia as a reported side effect.

3. c.
Water is 60% of total body weight for men and 50% for women.

4. b.
In SIADH, urinary sodium is more than 30mmol/L and the patient has a serum osmolality <270mosm/kg and a high urine osmolality >100mosm/kg.

5. b.
All patient assessments should begin with "ABCDE" assessments to detect and deal with any life-threatening complications (e.g. the loss of airway caused by the unconsciousness).

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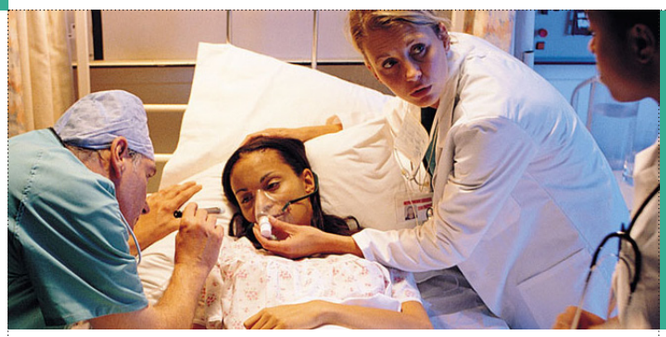
THE ACUTE CARE COMMON STEM: A ROUTE INTO TRAINING IN INTENSIVE CARE MEDICINE

Hasan Qayyum and Justin Squires



Both emergency medicine and acute medicine place great value on knowing when to refer a patient for admission or specialist input.
Teaching & Training.

Introduction

The Acute Care Common Stem (ACCS) was developed in response to an increasing demand for multi-competent junior doctors. It is designed to deliver medical staff skilled in recognising and managing sick patients; competent to define the nature of acute problems; and to deliver the specialist interventions required. In this article, we will review the content of the ACCS, why it might be the right path for you to take, what to look out for between different schemes and how to maximise your chances of getting a place.

What is ACCS?

ACCS is a 2-year programme which includes training in four specialities: acute medicine; emergency medicine; anaesthesia; and intensive care medicine. It promotes understanding about care of the full spectrum of patients and as such is a sound basis for a future in any of the four parent specialities. The overall programme[1] has been agreed with the relevant Royal Colleges and training boards and is now the standard entry point into emergency medicine training.

Emergency medicine

Trainees usually have a 6-month placement in their first year. The idea is to get a good mix of clinical knowledge and appropriate skills in the care of the ill or injured patient early on. The emergency department (ED) is a good place to gain a wide range of competencies and experiences very quickly. Depending on your career plans you may want to influence (if you can) the type of ED you work in: general physicians would not normally be involved in managing the care of children so you may be better in an adult only ED. If you are planning a career in emergency medicine a placement in a department that caters for both adults and children will give you valuable early experience of the many aspects of managing paediatric patients.

Acute medicine

This is a specialty rapidly developing its place in hospital medicine. In many hospitals acute medicine Foundation Year 2 doctors (FY2s) or Core Medical Trainees (CT1s) will take referrals from the ED or general practitioners for medical patients who need admission to hospital for investigation or treatment. These jobs usually alternate "take" 1 day and "post-take" the next. Support is from either the on-call medical registrar and consultant, or more often by acute medicine registrars and consultants. These are often busy but highly rewarding jobs as you are likely to present the patients you have seen to a consultant post-take ward round, providing immediate feedback (and multiple opportunities for completion of assessments).

Both emergency medicine and acute medicine place great value on knowing when to refer a patient for admission or specialist input. If you are planning to be a general physician, you need to appreciate the competing demands on the time of an emergency department doctor. Equally, if you are planning to be an emergency department doctor, you need know when to refer from the ED to acute medicine. These two placements ensure you make and receive such referrals many times and become able to make tricky judgement calls more confidently or know how to seek help and advice.

Anaesthesia

If you are planning a career in anaesthesia, the ACCS provides insight into medical management of sick patients and the initial management of trauma patients which you would otherwise not receive as part of the standard anaesthesia training programme. If you wanted some anaesthesia-related skills but did not want to be an anaesthetist, then previously they would have been hard to develop, as training in anaesthesia is often difficult to arrange for non-anaesthetic trainees. The ACCS programme now promotes the development of essential competencies in anaesthesia to allied emergency care trainees who are not necessarily planning a career in anaesthesia. Depending on local agreements with your school of anaesthesia, placements can be between 3 to 6 months.

Intensive care medicine

Recent literature[2] suggests a quarter of all intensive care unit admissions originate from the emergency department. Increasingly more ED doctors need more ICM competencies to bring critical care to the patient by initiating emergency treatment. For example, advanced airway management, non-invasive ventilation and intensive therapy for severe sepsis. This programme includes a placement in intensive care to develop expertise in these procedures and to develop an intensive care way of thinking.

Like anaesthesia, intensive care medicine has a high ratio of senior staff so there are many opportunities for learning. Ideally by the end of the CT2 year you should be able to accept patients to a critical care setting, demonstrating skills which will be valuable whichever path you plan to follow, even if only to immediately recognise patients who need critical care support.

You will be expected to complete at least the core competencies of the Intercollegiate Board for Training in Intensive Care Medicine (IBTICM) which will then count for the future should you wish to consider dual accreditation in ICM.

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How can I become an ACCS trainee?

Through competitive application under nationally agreed guidelines. The interview panel usually consists of representatives of all four specialities, so do not worry if you think your special interest in any of the four specialities will not get due credit. It is worth noting before committing to the ACCS programme, you are likely to be asked to specify the specialty you want to pursue a Certificate of Completion of Training (CCT) in.

Entry to the first year (CT1) of ACCS training is through direct entry from FY2 with no previous experience. SHOs with less than 12 months' previous experience at SHO level in anaesthesia, critical care, acute medicine or emergency medicine could also enter at CT1 level. The person specification and selection criteria are at:

http://www.mmc.nhs.uk/pdf/PS%202009%20CT1%20ACCS-AM_2.pdf

For entry at ST2 level, you will need at least 12 months experience at CT/SHO level in ACCS specialities, not including Foundation modules. See the criteria at:

http://www.mmc.nhs.uk/pdf/PS%202009%20CT2%20ACCS-EM_2.pdf

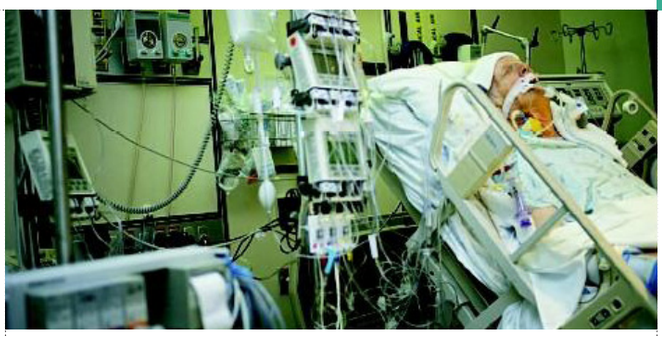
In essence, what the appointment panel is looking for is evidence of commitment to a specialty, whichever one you choose. Listed here are a few attributes we think will give you an edge:

1. Relevant experience helps. For example, a Foundation module in relevant medical (e.g. cardiology, respiratory medicine) or surgical (neurosurgery, urology, orthopaedics) specialties.

2. Audit and Research. Participation in audits is direct evidence of clinical governance and will support your application immensely. Try to complete a full audit cycle if possible. Find out who your department's audit lead is and get started; this is often the most difficult step. Some consultants will see your participation in research and audit as a good time management exercise. Research might sound daunting at Foundation level, but, believe us, it isn't. Evidence of contribution to departmental guidelines and e-learning modules all count.

3. Teaching Skills. Evidence of structured feedback for teaching, recommendation of instructor potential on a recent life support course and deanery-based teaching courses (e.g. teaching the teachers) are far more feasible with limited time than trying to obtain a professional qualification in medical education.

4. Exams. We all know how difficult it was to get study leave or even exam leave during our Foundation modules. Exams are not encouraged in this period as they do not form part of Foundation competencies, which is your main goal in those years. However, candidates with an exam pass (MCEM Part A, MRCP Part 1 or FRCA Primary MCQs) are considered to have an edge. Again, although exams may not count in the shortlisting process, they are certainly perceived as evidence of independent learning and commitment.



5. Personal Skills. There are key personal skills which the interviewing panel are looking for. Situational awareness (the ability to recognise a critically ill patient in a full waiting room or realising a simple general anaesthetic is not going well and getting help before it gets out of control), coping with pressure (can you increase activity without making mistakes when you need to), being a good team member (and the ability to take a leadership role in a safe environment) and good communication are just a few essential attributes looked for.

I am an ACCS trainee – what now?

Well done, but getting in is only half the job. The next 2 years will be filled with surprises and new challenges. Juggling the rest of your life with shifts looking after the sickest patients in the hospital can be difficult. Determination, perseverance and a sound clinical knowledge with loads of enthusiasm will help. So might some of the tips below:

1. Get an educational supervisor. You need one. Remember, you need to take the initiative, making sure you get appraisals and workplace-based assessments done. Your supervisor should help, support and advise you, focusing you towards necessary and achievable goals.

2. Workplace-based assessments. There are quite a few that need completing! The best way is to do them as you go and not leave them to a week before your last appraisal. Useful people to approach include senior trainees, staff grades and nurses for areas within their competency, as well as consultants. As part of your CT2 year, you will need to get basic competencies signed as well. In itself this is a big task requiring two consultants in anaesthetics/intensive care medicine to sign you off for each of the basic competencies. After a period of orientation in these placements, especially intensive care, it is sensible to try to get something signed off every shift, this also makes sure that you are getting the best educational value out of each post and that your learning is relevant to your syllabus.

K Think of logbooks as evidence of independent learning and they become your friend. There are excellent examples on the College of Emergency Medicine and the Royal College of Anaesthetists' websites. A simple format is also present in the ACCS training manual1. The key here is to start at literally day one and enter the information as you go. It is very complicated and time consuming to do at the end of a placement, as we have learned to our cost!

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4. e-learning. Another novel approach to aid your learning needs is completing online modules. The College of Emergency Medicine has its own free section on the **doctors.org.uk** website called **CEM ENLIGHTENme.** Similarly, to aid your anaesthetics/ICM needs, the website **e-la.org.uk** has numerous modules you could complete which cover a range of topics from basic anatomy of the airway to the pharmacology of muscle relaxants.

What happens after ACCS?

Hopefully, this should be clear from the start. Your next step will depend on which specialty CCT programme you are joining. Acute medicine and emergency medicine trainees will join their respective trainees at ST3 level. Anaesthetic trainees join at ST2 level and will have expected to pass the FRCA primary MCQ assessment prior to this. If a trainee does more than 3 months of ICM, they will be expected to have completed some of the intermediate competencies. Because there is no standalone CCT programme in ICM, trainees dual accrediting must complete one of the CCT programmes listed, then devote further time to specific ICM training. See the ICTBICM website for more detail.

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2. The Way Ahead 2008–2012. College of emergency medicine. December 2008. **www.collemergencymed.ac.uk/asp/document.asp?ID=4474**

Useful websites

www.accsuk.org.uk
Will give you a structure of your ACCS programme. A good source to download your assessment forms from.

www.e-la.org.uk
Designed to cater for the needs of trainee anaesthetists, this useful website can also be used by ACCS trainees free of cost for a limited period. The interactive lessons and self-assessment sessions follow the anaesthesia curriculum.

www.collemergencymed.ac.uk
The College of Emergency Medicine website. Information on exams, forthcoming conferences, training needs and membership, plus a useful section on clinical guidelines.

www.ep-ic.org
Emergency Physicians in Intensive Care (EPIC) information on intensive care training aimed at emergency medicine doctors.

www.rcoa.ac.uk/ibticm
The Intercollegiate Board for Training in Intensive Care Medicine. Contains all the info about intensive care training from basic (at ACCS level) right through to the award of a joint CCT.

www.ics.ac.uk
The Intensive Care Society. Helpful information on intensive care, also information on national "State of the art" meetings which are where you should aim to get your audit (if you do one in intensive care) presented.

www.acutemedicine.org.uk
The Society for Acute Medicine.

www.rcoa.ac.uk
The Royal College of Anaesthetists includes lots of relevant information and the important downloadable e-logbook.

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