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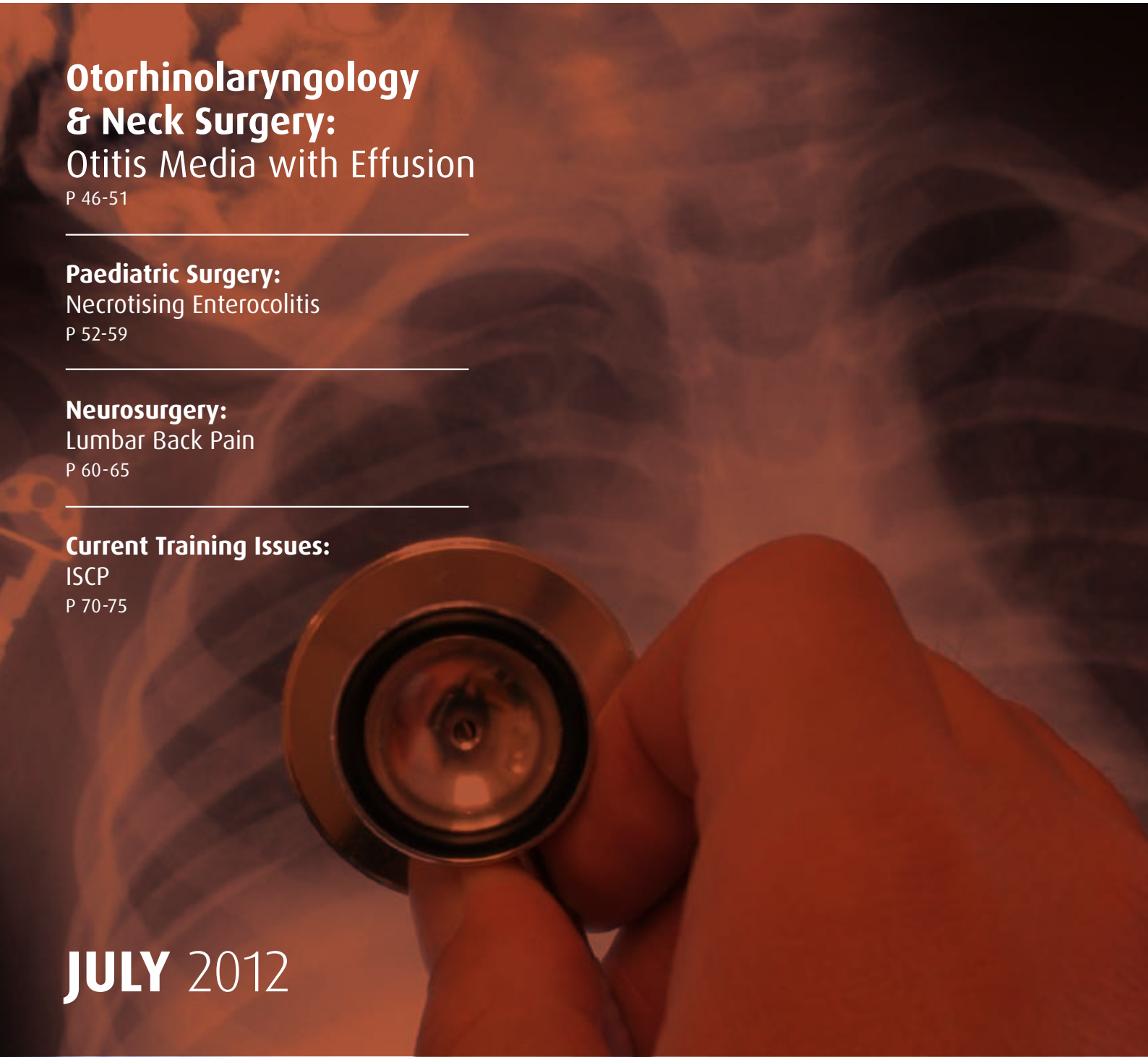
CORE SURGERY JOURNAL

Volume 2, Issue 4



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Dear Prospective Authors

Thank you for considering the submission of an article to 'Core Surgery'. This is a new journal aiming to educate and inform junior surgical trainees about relevant 'core' subject topics. Each issue will cover a topic from selected subspecialty fields; General Surgery, Orthopaedics and Trauma, Plastic Surgery, Ear Nose and Throat Surgery, Neurosurgery, Urology, Paediatric Surgery and Intensive Care Medicine. Articles will be required to be broad enough to help with preparation for the intercollegiate MRCS examination but also focus on key hints and tips on becoming a higher surgical trainee. A list of core topics in each subspecialty has therefore been agreed by the editors based on a selection of key topics in the MRCS curriculum. Authors are advised to agree a topic with the editors before writing an article.

Types of Article

Manuscripts are considered under the following sections:

1) Case based discussions
2) Practical procedures
3) Audit
4) Review articles
5) Course reviews
6) Research papers

Submission of Manuscript

Submissions will only be accepted via email and must be accompanied by a covering letter. Please submit your article to **coresurgery@123doc.com.** The covering letter must include a statement that all authors have contributed significantly and accept joint responsibility for the content of the article. In addition any financial or other conflict of interest must be declared.

Manuscript Style

Submissions should follow the style of the Vancouver agreement detailed in the International Committee of Medical Journal Editors' revised 'Uniform Requirements for Manuscripts Submitted to Biomedical Journals: Writing and Editing for Biomedical Publication', as found at **http://www.ICMJE.org/**

References

All articles must be referenced appropriately. The Vancouver system of referencing should be used; details can be found at **https://workspace.imperial.ac.uk/library/Public/Vancouver_referencing.pdf.** References should be cited using numerals in brackets [eg. (1)], in the order in which they appear. The list of references should reflect this order and names of journals should be abbreviated in the style used in Index Medicus **ftp://nlmpubs.nlm.nih.gov/online/journals/ljiweb.pdf.**

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Guidelines for the format of respective article types are as follows. All articles must contain an abstract of 150-250 words for indexing purposes and 3-5 keywords.

Case Based Discussions

Guidelines for the format of respective article types are as follows. All articles must contain an abstract of 150-250 words for indexing purposes and **3-5 keywords.**

Case based discussions

Should be about 1000-1500 words long and should focus on clinical assessment, differential diagnosis or treatment. The basic structure should be as follows:

Abstract:	The salient points of the case and discussion.
Case history:	Including the initial presentation, clinical setting and problem, investigation and treatment.
Discussion:	Covering the critical aspects of the management and the treatment options.

Practical Procedures

Should be about 1000-1500 words long. Although not essential it is highly advantageous if pictures and diagrams are supplied to illustrate the most salient points. Articles should be set out as follows:

- Abstract (Essential) – A summary of the article structure and salient features.
- History and pathology
- Indications and contraindications
- Gaining informed consent /explaining procedure to patient
- Equipment required
- Draping / sterile field preparation
- Patient positioning and relevant anaesthetic points
- Documentation of procedure
- Recording of complications and management of such

Audit

Articles should be 1000-1500 words long and of high quality. Each article must contain an abstract. Completed audit cycles are strongly preferred as are audits which have led to guideline development.

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Review Articles

The topic should be relevant to core surgical trainees, and a maximum of 2500 words long. The review should include an abstract, and a clinical vignette of a case relevant to the topic. The aim of including a clinical case is to provide a focus for discussion, and to ensure that the review is relevant and useful to our readership.

Course Reviews

Should be a maximum of 1000 words and review a course which is either mandatory or desirable for core trainees and junior higher surgical trainees. An abstract is required summarising the article contents and salient conclusions.

Research Papers

Although the publication of research articles is not a core aim of the journal, Core Surgery welcomes research submissions if thought to be of interest to the readership. Articles should be written using the following headings (title page, abstract, introduction, methods, results, discussion, references). They should be a maximum of 2500 words of text including abstract, 30 references, 3 illustrations or figures. The abstract should be a maximum of 250 words and use the following headings (introduction, methods, results, conclusion). The title page should contain the title of the paper, the full names of the authors, the addresses of the institutions at which the research was carried out and the full postal address, email address and telephone number of the corresponding author.

MCQs / EMQs (All Articles)

Please note that all articles should be submitted **with five multiple choice questions (MCQs) or extended matching questions (EMQs)** attached, in the style of the Member of the Royal College of Surgeons (MRCS) 'Part A' examination. These questions should have answers and brief teaching notes/discussion included. Examples of the requirements for question style can be found here: **http://www.intercollegiatemrcs.org.uk/old/pdf/samplequestions_MCQ.pdf**

Summary

Articles considered for publication will be sent for review by our panel of consultants and junior surgical trainees. We wish you every success with your submission. Please contact the editorial team with any questions.

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NUTRITION IN THE SURGICAL PATIENT

D Kamali & R Shafei

Abstract

Optimal nutrition is dependant upon adequate intake and an intact functioning gut. There are many surgical conditions and operations that affect all three aspects leaving patients at high risk of malnourishment. Accurate assessment of the patient's condition and management will predict nutritional deficiencies. There are various forms of supplemental nutrition available to the sick surgical patient and the factors involved in preventing malnutrition in specific surgical conditions are discussed.

Keywords: Nutrition, surgery, re-feeding.

The maintenance of adequate nutrition requires three main criteria to be fulfilled: adequate intake; an intact GI tract and the ability to absorb nutrients.[1] Energy requirements should be fulfilled with carbohydrates (50%), fat (35%) and protein (15%) together with vitamins, minerals and trace elements.[2] Total energy requirements vary widely between individuals due to variations in BMR and physical activity, but average values can be calculated according to age (Table 1).

Nutrient	Adults 19-50 years	Adults >50 years
Carbohydrate	185g	176g
Fat	128g	123g
Protein	55g	53g
Iron	15mg	9mg
Zinc	9.5mg	9.5mg
Salt[3]	6g	6g

Table 1: The daily requirements and composition according to age.

Water is the most abundant constituent of the human body, making up approximately 60% body weight.[2] Daily fluid requirements vary according to age, environment, diet, levels of physical activity and illness, but average at 1.5L to 2L.

Nutritional Assessment and the Basal Metabolic Rate

Basal metabolic rate (BMR) is the energy expenditure by the body when awake and at rest in the supine position, 12 hours after a meal, following eight hours of physical rest and being in an environment that does not evoke shivering or sweating.[3,4] BMR can be measured only under laboratory conditions. Increased body weight, illness, younger age and male gender all increase the BMR[4] and therefore energy requirements.

Nutritional assessment should be an essential part of routine examination for every surgical patient in order to plan for surgical procedures. There are novel ways of assessing nutritional status but the most commonly used is body weight. An assessment of the Body Mass Index (BMI) is important together with recent weight loss. High risk patients are those with BMI <18.5 and recent weight loss of greater than 10%. Anthropometric measures (e.g. skinfold thickness, mid arm circumference) and functional tests (e.g. hand grip strength) may predict muscle mass loss but are unreliable in a clinical environment.

Boichemical investigations such as electrolytes, albumin, magnesium, calcium and phosphate levels are more commonly used as post operative markers of unwell patients and response to treatment. Close observation of unwell patients and involvement of dieticians are key to predicting malnutrition and planning optimal nutrition.[5]

Malnutrition in the surgical patient

Malnutrition is common. It occurs in 30% of patients with gastrointestinal disease. In patients with post-operative complications the figure is as high as 60%.[6]

Surgical conditions that predispose to intestinal failure include pancreatitis, peptic ulcer disease, mesenteric ischaemia, malignancy, diverticular disease and inflammatory bowel disease. Surgical patients are unlike any other patients in the hospital as in addition to their reason for theatre which may have rendered them malnourished in the first instance, they are starved further before their operation. If enteral feeding is not established to normal limits soon after surgery, a problem frequently encountered in emergency surgery, then multiple adverse outcomes can arise. (Table 2)

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Adverse Effect	Consequence
Impaired immune responses	Predisposes to infection & 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 fracture malunion. All can lead to prolonged recovery from illness, increased length of hospital stay and a delayed return to work.
Reduced muscle strength	Inactivity, inability to work effectively, and poor self-care. Abnormal muscle function may also predispose to falls or other accidents.
Reduced respiratory muscle	Poor cough pressure, predisposing to and delaying recovery from chest infection. Difficulty weaning malnourished patients from ventilators.
Inactivity	Predisposes to pressure sores and muscle wasting

Table 2: Adverse outcomes associated with malnutrition post operatively[7]

The metabolic response to starvation

When a patient has been fasting for twelve hours, most food from the last meal will have been absorbed. This results in an initial fall in plasma insulin and a rise in glucagon with subsequent glycolysis. The liver is now the source of energy in starving conditions. Glycogen is stored primarily in muscle but needs to be first broken down and converted into lactate. Lactate is then converted to glucose which is utilised for the metabolic needs of the organs, a process known as the Cori cycle. If fasting persists, the glycogen stores become depleted necessitating the use of alternate energy stores which leads to the utilisation of proteins and then fats.[6]

Studies of malnourished surgical patients show rapid functional benefits when adequate feeding is provided. These changes can occur well before weight has been regained, for example, malnourished patients, who often have low collagen deposition rates in surgical wounds, show improved deposition within days of receiving nutrition support.[8]

How can nutritional status be optimised pre-operatively?

Four major directions of research have emerged following the recent research on peri-operative care principles in digestive tract surgery. This is otherwise known as enhanced recovery after surgery (ERAS) and covers: detecting malnutrition, peri-operative hyperglycaemia/insulin resistance, enteral/parenteral nutrition and immunonutrition.[9]

Traditionally, patients have been fasted from midnight on the night before elective surgery. With the advent of the ERAS programme, there has been a major change in established practice. Patients are now able to continue with clear fluids up to two hours prior to surgery and solids up to six hours prior to surgery. This may change again in the near future however, as further research is now emerging to suggest that nutritional supplementation in the immediate pre-operative period may enhance post-operative recovery.[10]

Studies in colorectal patients using a carbohydrate loading drink prior to surgery is associated with a reduced hospital stay and a quicker return to normal gastrointestinal function.[11]

Methods of delivering nutrition

Enteral nutrition (EN) is defined as the delivery of a nutritionally complete feed (containing protein, carbohydrate, fat, water, minerals and vitamins) directly into the gut.[12] This is the preferred route of nutrition in all patients wherever possible. In most patients EN is achieved orally however, in some patients this is supplemented or given completely through either a nasogastric (NG) or nasojejunal (NJ) feed. NG or NJ is commonly used in patients with an impaired swallow or absent gag reflex where the risk for aspiration is high (e.g. in neurological disease).[3] If these risks are long term then a more permanent method of enteral feed, such as a percutaneous endoscopic gastrostomy (PEG) is commonly used.

If none of these routes are appropriate, total parenteral nutrition (TPN) is administered which provides all nutritional requirements through the intravenous route, completely bypassing the gastrointestinal tract. The most common indications for TPN are: massive resection of the small intestine; intestinal fistulae or prolonged intestinal failure for other reasons. In other words, when energy and protein needs cannot be met via enteral administration.[6]

Both methods have their associated complications. EN has long been thought of as superior to TPN. Extensive research has repeatedly shown that clinical outcomes are more favourable with EN than TPN, including reduced risk of sepsis, multi-organ failure, haemoglobin concentrations and improved intestinal barrier function.[12,13,14] Table 3 summarises EN and TPN.

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	Enteral Nutrition	Total Parenteral Nutrition
Indication	• Moderate to severe malnutrition • Dysphagia • Distal enterocutaneous fistula • Major trauma & sepsis • Prolonged recovery • Inflammatory bowel disease	• Small/Large bowel obstruction • Short bowel syndrome • Proximal intestinal fistulae • Refractory inflammatory bowel disease
Advantages	• Maintains gut physiological • Increase gut blood flow • No central venous access • Protects from stress ulceration • Cheaper than TPN	• Does not require a functioning gut • All nutrition enters systemic circulation
Disadvantages	• Requires functioning gut • Gastro-oesophageal reflux • Gut upset (nausea, vomiting, diarrhoea, bloating, distension) • Feeding tube complications (nasal ulceration, displacement and blockage)	• Metabolic disturbances • Hepatic dysfunction • Expensive • Gut atrophy • Regular biochemistry monitoring • Complications of line insertion

Table 3: A comparison of EN and TPN[5,16]

Short bowel syndrome

Up to 50% of the small bowel can be resected without the need for long-term nutritional support. Patients develop malabsorption, malnutrition, diarrhoea, and electrolyte abnormalities. TPN is used in patients who have less than 150cm of small bowel. The main reason for this is as fluids and electrolytes advance along the digestive tract, the transit time slows allowing for increased reabsorption of essential elements. It is estimated that small bowel motility is three times slower in the ileum than the jejunum, with the colon the site of greatest water reabsorption. There are higher absorptive qualities of the ileum compared with the jejunum. Once adequate fluid resuscitation has been achieved, an assessment can be made of nutritional absorption and estimating parental supplementation. Patients, who continue with a degree of enteral nutrition, may suffer with gastric hypersecretion and persistent loose motions. These may be managed with proton pump inhibitors, loperamide, codeine and somatostatin analogues but still suffer with symptoms and require all nutrition from TPN.[17]

Re-feeding syndromes

Re-feeding problems can be life threatening. They encompass acute micronutrient deficiencies, fluid and electrolyte imbalance, and disturbances of organ function that results from over-rapid or unbalanced re-feeding. They may occur in any malnourished individual but those particularly at risk are patients who have had very little or no food intake, including overweight patients.[12]

Starvation causes adaptive reductions in cellular activity and therefore organ function accompanied by deficiencies of vitamins, magnesium, phosphate and whole body depletion of intracellular potassium.[18] Adaptive changes in starvation can be reversed by giving nutrients and fluid but doing so leads to an increased electrolyte and micronutrient demand, and simultaneously shifts sodium and water out of cells. Over-rapid or unbalanced nutritional support can therefore precipitate dangerous changes in fluid balance and deficiencies in phosphate, magnesium, potassium, and calcium.[7] These may lead to organ function disturbance in the form of dysrhythmias, cardiac failure, pulmonary oedema and circulatory fluid overload or depletion.

The problems of re-feeding are less likely to arise with oral feeding since starvation is accompanied by reduced appetite.[12] However, excessive feeding can be easily achieved with EN or TPN so care should be taken in the prescription of oral nutrition supplements. To reduce risk for these patients, nutritional support should be introduced at 50% of requirements for the first two days with generous thiamine provision, along with a multi-vitamin and trace element supplementation. Increasing to meet full needs may only occur where clinical and biochemical monitoring exclude re-feeding problems.19 Even greater care is needed in some patients, particularly where the following apply;

- **BMI <16 kg/m2**
- **unintentional weight loss of >15% over the last 3 – 6 months**
- **very little or no nutrient intake for >10 days**
- **deficiencies in potassium, phosphate or magnesium prior to feeding (13,20)**

NUTRITION IN THE SURGICAL PATIENT

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An example of a malnourished patient:

Questions

1. The Cori cycle is important in the utilisation of alternate energy stores to produce glucose. True or false?

2. The use of carbohydrate loading in colorectal patients decreases the risk of re-feeding but increases the time to restore normal bowel function. True or false?

3. Which is the site of greatest water reabsorption?

a) Duodenum
b) Jejunum
c) Ileum
d) Colon

4. Short bowel syndrome occurs when more than 150cm of small bowel is resected. True or false?

5. Which of the following characterise re-feeding syndrome?

a) Dysrhythmias
b) Cardiac failure
c) Pulmonary oedema
d) Fluid overload or depletion
e) All of the above

Answers

1. True

2. False: Use of carbohydrate loading decreases the risk of re-feeding and decreases the time to restore normal bowel function.

3. d) Colon

4. False: Short bowel syndrome occurs in patients who have a 150cm or less of small bowel remaining.

5. e) All of the choices

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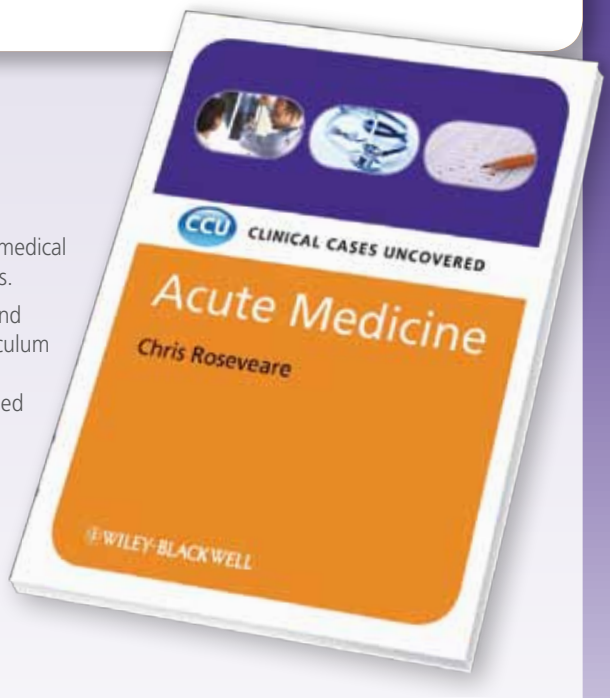
Acute Medicine

CLINICAL CASES UNCOVERED

Chris Roseveare,
Southampton General Hospital



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WILEY-BLACKWELL

SURGICAL TRAUMA CALLS - CLINICAL ASSESSMENT & MANAGEMENT PRINCIPLES OF BLUNT ABDOMINAL TRAUMA

C Fortescue

Surgical trauma calls - Clinical assessment & management principles of blunt abdominal trauma
General Surgery

Abstract

Initial approach to the trauma patient along the ATLS guidelines gives a thorough and systematic assessment and identifies life-threatening pathology early on. In blunt abdominal trauma identifying the point of injury can be difficult clinically, but clues can be taken from the history and examination findings. Careful investigation choices are needed to make a diagnosis and management strategy. Computed tomography is commonly used and treatment is either observation, endovascular therapy, or open surgical repair. The liver and spleen are the most commonly damaged organs but even when organ damage is identified not all injuries require surgery and many can be managed conservatively. The decision whether or not a laparotomy is indicated is not usually left solely to the SHO. If abdominal injury is suspected get senior support early.

Keywords: Trauma, ATLS, laparotomy.

Trauma calls can be panic-inducing-hives-of-activity, but following the Advanced Trauma Life Support (ATLS) algorithm is a straight-forward way to assess and initially manage any trauma patient until you can start putting the pieces together for form a differential diagnosis and management plan.[1] This begins with the ABCDE approach of a primary survey and stabilising the patient, followed by a detailed secondary survey and further investigations as necessary, see Figure 1.

A is for AIRWAY	a) Patent, obstructed or partially obstructed? b) Look in the mouth for any foreign objects, vomit or loose teeth. Remove an object under direct vision, but do not blindly finger sweep the oral cavity. c) 'A' also includes C-Spine immobilisation and oxygen. Check hard collar is correctly fitted and sized. All patients should be given high-flow oxygen with a non-rebreathe mask.
B is for BREATHING	• Equal, bilateral chest movement? Paradoxical movement? Central trachea? • Percussion – dull suggests fluid/blood and hyper resonance suggests air in chest cavity • Auscultation • Respiratory rate and oxygen saturations.
C is for CIRCULATION	• Heart rate and blood pressure. • 2x wide-bore cannulae, IV fluids, O-negative blood on stand-by. • Signs of haemorrhage – "4 & the floor" – thorax, abdomen, pelvis & long-bones/limbs. • Apply dressings to any wound/lacertions and splint and broken/bleeding limbs
D is for DISABILITY	• Glasgow Coma Score (GCS) – Head injury • Blood glucose
E if for EXPOSURE	If you have reached 'E' and patient is stable, a top-to-toe examination is required looking for any other injuries o Scalp – lacerations, bony tenderness, 'boggy' swellings. o Face – Lacerations/bruising/bony tenderness. Check mandible, jaw closure and teeth alignment. o C-Spine – log-roll the patient - palpate the whole spine from top to bottom to elicit any bony tenderness. Perform a PR at this stage to check anal tone and sensation if there is any suspicion of a spinal injury. o Chest – look for bruising. Palpate for any areas of tenderness suggestive of rib fracture(s). o Abdomen – look for bruising. Palpate and auscultate. o Pelvis – bruising, tenderness. Genital examination especially in males with a history of a deceleration injury. Urine dip to look for haematuria. o Limbs – Laceration/wounds? Palpate each bone in turn & move each joint – low threshold for imaging

Figure 1: The primary and secondary ATLS survey (1)

Case study

A 29 year old male had an RTA, driving along the road when he lost control, hitting a moving bus. He was estimated to be driving at 35mph. He was the only occupant of the car. He was wearing a seat-belt and the air-bags (steering wheel and side) were deployed on impact. He was cut out of the car by the fire service– delaying his transfer to hospital by roughly 40 minutes. He is transferred to the stretcher.

Particularly think about the mechanism of injury and what you may expect to find. This patient was in an RTA and was wearing a seat belt. The mechanism would likely be a sudden deceleration with a force applied to the abdomen and chest from the seat-belt. Rapid deceleration can also cause hyper-extension and hyper-flexion injuries to the c-spine, and cause intra-abdominal shearing forces.

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The patient was alert and orientated. He was able to speak (airway patent) and his C-spine had been triple-immobilised correctly by the paramedics. The chest moved normally with breathing. There were reduced breath sounds and hyper-resonance on percussion on the left side. The trachea was central. Respiratory rate was 24 and oxygen saturations were 99% on 15l via a non-rebreathe mask. Heart rate 95 and blood pressure 125/75. GCS was 15/15 and blood glucose was 5.5. There was no obvious source of bleeding.

On abdominal examination there was marked generalised tenderness with guarding, with maximal tenderness in the right flank, right iliac fossa and right hypocondrium. There was also noted to be bruising of the anterior abdominal wall in a horizontal distribution across the lower abdomen and over the right clavicle – consistent with a seatbelt pattern. A chest drain was sited on the left side. The C-spine was cleared after clinical and radiological examination. Focused Abdominal Sonography for Trauma (FAST) was performed which did not show any free fluid within the abdomen. The patient remained haemodynamically stable and a CT chest/abdo/pelvis was performed. This showed no injury other than the pneumothorax.

The abdomen is a notably difficult part of the body to assess clinically. The abdominal cavity contains many different organs and major blood vessels and damage to these organs may present in a very similar way. Key clinical signs are peritonitis, localised abdominal tenderness with guarding, shoulder tip pain (referred pain via phrenic nerve), persistent hypotension with no obvious cause for bleeding found, blood at the urethral meatus and bruising of the abdominal wall.

The history of the trauma scenario is key to predicting injuries;

- Mechanism of contact - which was moving and which was stationary – the object of impact or the body. (The body moving onto a stationary object can be considered a deceleration injury)
- The site if impact and what structures underlie it.
- The speed or force of contact.

Blunt abdominal trauma causes intra-abdominal injury by three main mechanisms;

- Deceleration injuries occur as a result of sheering forces between organs/structures of different mobility. Examples include the junctions between peritoneal and retroperitoneal structures, vascular pedicles and bowel mesentery. Shearing forces to vessels typically cause intimal tears, which may lead to thrombosis and vessel occlusion.

- Crush injuries result from a direct blow to the abdomen/truck, which results in the abdominal organs being crushed against the posterior abdominal wall (lower rib cage) or vertebral column. Solid organs such as liver, spleen and kidneys are most vulnerable to this mechanism as they are fixed. Where as most bowel and vasculature have some degree of mobility to escape the crushing force.

- Compression injuries are often similar to crush injuries in mechanism of action. However, instead of a force applied to a localised point of the abdomen they cause a generalised, rapid rise in intra-abdominal pressure. Unlike crush injuries it is the hollow organs, such as bowel and bladder, which are most at risk here resulting in rupture or perforation.

Assessment

The aim of the assessment is to identify patients with life-threatened abdominal injuries and proceed to emergency laparotomy, but to avoid unnecessary surgery in those who have more minor injuries and predict those injuries that are not immediately life threatening may still require surgical intervention at a later stage.

Ultrasonography

This is 'Focused Abdominal Sonography for Trauma' and involves ultrasound of the abdomen to look for the presence of free fluid. It is widely available in most A+E departments and is 60-94% sensitive and 83-98%.[2,3] A 'negative' FAST scan does completely not rule out intra-abdominal injury, as it is particularly poor at identifying retroperitoneal and hollow viscus injuries.[4] The accuracy of ultrasonography is highly operator dependant.[5]

Diagnostic Peritoneal Lavage (DLP)

Due to the now widespread availability of FAST scanning DPL has largely become obsolete in the trauma setting.[6] DLP continues to be taught on trauma courses and one deanery found that 62% of its surgical trainees had never even seen DLP being performed.[7] Several recent studies have shown it to be far superior to FAST, and even computed tomography (CT) at detecting intra-abdominal injury, where the only abnormality is mesenteric or bowel trauma.[8]

Computed tomography (CT)

CT is reserved for patients who are haemodynamically stable and have clinical signs or a high index of suspicion for intra-abdominal injury. Helical (spiral) CT with intravenous contrast is highly accurate at identifying solid organ injury to the liver and spleen, as well as arterial haemorrhage and retroperitoneal damage.[9] It is less sensitive in bowel, mesenteric and pancreatic injury, and is poor at detecting venous haemorrhage.[10] In patients with reduced conscious level or a suspicion of cranial, cervical spine or thoracic trauma 'full body' (head to pelvis) CT scan may be advocated (See figure 2).

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Diagnostic modality	Advantages	Disadvantages
FAST scan	• Quick • No radiation • Readily available • Sensitive and specific for free fluid	• Rarely identifies site of bleeding • Poor for hollow viscus and retroperitoneal injury • Operator dependant
CT scan	• Good identification of solid organ injury • Can assess retroperitoneum • Can identify arteial haemorrhage	• Involves radiation • Patient must be haemodynamically stable • Poorly identifies bowel and mesenteric injury • Cannot identify venous haemorrhage
DPL	• Better than FAST and CT for identifying hollow viscus and mesenteric injury • Most sensitive than FAST and DPL	• Invasive • Doesn't identify site of bleeding

Figure 2: Comparing the main trauma imaging modalities.

Management

Management of abdominal trauma largely falls into 2 categories: surgical and non-surgical. All haemodynamically unstable patients proceed rapidly to laparotomy to control the bleeding. Investigation and observation determine the outcome for the remainder (See figure 3).

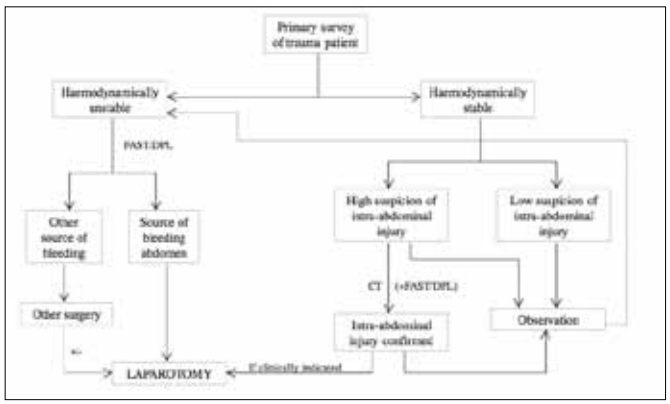

Figure 3: Basic algorithm for management of blunt abdominal trauma.

Indications for laparotomy

Any haemodynamically unstable patient with a strong suspicion that the abdomen is the source of bleeding should proceed for emergency surgery. 'Strong suspicion' may be due to free fluid on FAST scan, clinical signs, or from a process of elimination that no other source of bleeding has been identified and the patient remains haemodynamically unstable. In recent years more and more patients with confirmed intra-abdominal injury are being managed non-operatively and advances in endo-vascular procedures/therapeutic angiography mean that bleeding can often be controlled without surgery.

Other factors or associated injuries that may push your decision towards laparotomy include: associated chest trauma or pelvic fracture requiring surgery, base deficit/increased serum lactate (suggestive of ischaemic process), haematuria or reduced consciousness of the patient. Avoiding unnecessary laparotomy will spare patients the risk of developing post-operative atelectasis, pneumonia, pleural effusion, ileus or wound infection among others.

Emergency laparotomy

A laparotomy for trauma requires a large mid-line incision, typically from xiphisternum to pubic symphysis. On opening the abdomen any frank blood should be manually removed and systematic packing of the abdomen with large, dry abdominal swab begins. All 4 quadrants of the abdomen should be packed, but in particular packs should be placed:

- Around the liver

(lateral, inferior, anterior to the liver and superior at the right costal margin)

- Below the left hemi-diaphragm
- Lateral to the ascending and descending colons
- Pelvis
- Central abdomen

Each can them be removed in turn and the organ examined. Bleeding vessels are initially clamped with mosquito forceps and only tied off definitively once full haemostasis of abdominal bleeding has been achieved. The bowel is also examined and any gross rupture is closed with staples or sutures. A full and detailed inspection of the abdomen is only carried out once haemostastis is achieved and gross contamination has been limited. This may be during the same procedure, or the patient may return to theatre at a later time. If the patient is to return to theatre on a further occasion the abdomen is usually not closed definitively, but temporarily, often using a clear dressing.

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Types of Injury

Solid organ injury

The liver and spleen are the two most commonly damaged organs in blunt abdominal trauma. Both can undergo organ laceration/paranchymal injury and haematoma formation. Grading systems for describing the extent of damage on CT scan exist for both,[11] but largely speaking less severe injuries may be managed non-surgically and severe injuries usually surgically.[12]

Liver

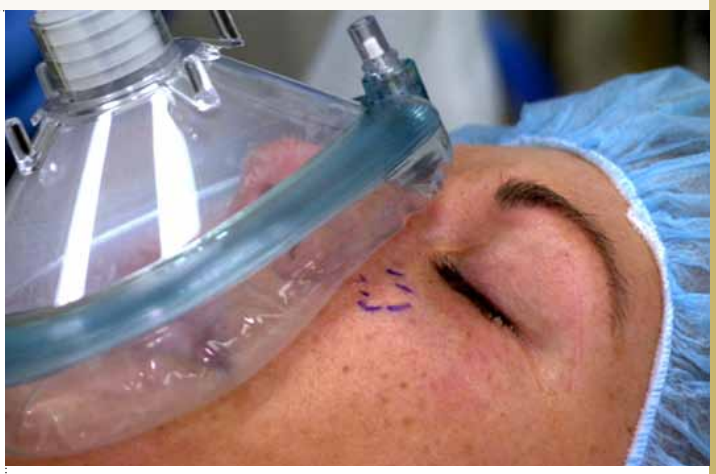
Hepatic injuries as a result of blunt trauma include contusions, subcapular and intra-parenchymal haematomas, lacerations (single or multiple) or fracture of the parenchyma. The latter of these may result in areas of devitalised tissue and hepatic necrosis. The severity of such injuries has been classified by the American Association for the Surgery of Trauma (AAST) into 5 grades (See figure 4).

Grade 1	Haematoma	Subscapular <1cm
	Laceration	Superficial parenchyma <1cm deep
Grade 2	Haematoma	Subcapsular or parenchymal 1-3cm width
	Laceration	1-3cm depth in parencyma
Grade 3	Haematoma	Subcapsular or parenchymal >3cm width
	Laceration	>3cm deep
Grade 4	Haematoma	>10cm
	Laceration(s)	Lobar destruction/devascularisation
Grade 5	Global destruction/devascularisation of liver	

Figure 4 - AAST grading of traumatic hepatic injuries (15)

The liver, unlike most other organs, has a surprising capacity to heal after quite major insult. A large majority of patients will undergo non-surgical treatment. One study found that grade I-III injuries faired well with non-surgical management, with failure in only 3% to 7.5% of cases. Whereas grades IV and V had failure of conservative management in 14% to 52% of cases.[13,14]

Where laparotomy is required to achieve haemostasis this is accomplished at first with packing around the liver to tamponade the bleeding. Individual vessels may then be cauterised or sutured, and larger lacerations can be held with small sutures, with or without an omental plug. In the rare instance that the blood supply to an area is fully disrupted, the devascularised segment should also be removed.



Spleen

Traumatic splenic injuries also have an AAST classification – based on their appearance on CT scan (Figure 5). However, unlike the liver, the spleen may also undergo delayed rupture. Due to the post-operative risk of overwhelming post-splenectomy infection haemodynamically stable patients with splenic injury are managed non-surgically. Even those who demonstrate bleeding at the time of CT scan usually have gone on to haemostasis by the time laparotomy is performed.[16]

Grade 1	Haematoma	Subcapsular – less than 10% of surface area
	Laceration	Capsular tear <1cm
Grade 2	Haematoma	Subcapsular – 10 - 50% of surface area Intra-parenchymal – less than 5cm
	Laceration	1-3cm – not involving trabecular vessels
Grade 3	Haematoma	Subcapsular - >50% of surface area Expanding or ruptured haematoma Intra-parenchymal - >5cm or expanding
	Laceration	>3cm depth or involving trabecular vessels
Grade 4	Laceration	Involving segmental or hilar vessels Devascularisation >25%
Grade 5	Shattered spleen Vascular injury to hilum	

Figure 5. AAST grading of traumatic splenic injuries (15)

Identifying the patients who will fail conservative management continues to be a challenge. Of those with 'high grade' injuries on CT and for planned observation, up to 44% will go on to require surgery. In some instances therapeutic angiography, with transcatheter embolisation of a specific vessel has been shown to improve the outcomes for the non-surgical management group. Particularly in patients with splenic injury, either proximal or distal splenic artery embolisation has been shown to successfully control haemorrhage, while avoiding the need for laparotomy.[17]

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Kidneys

Renal injury occurs in less than 5% of blunt abdominal traumas.[18] These may be intrarenal or subcapsular haematomas, lacerations, segmental infarctions and pedicle injuries/avulsion. Of the lacerations, these can further be classified as to whether they communicate with the renal pelvis or not. Renal injuries can be graded as minor, intermediate and severe, or by the AAST classification (See Figure 6). Grade I and II (corresponding roughly to 'minor') are usually managed non-surgically. Grade V almost always require surgery and grades III and IV may under selective surgical exploration. Only 1.9% of blunt renal injuries will require surgical exploration[19] and 75% to 85% of renal injuries are considered 'minor'.[16] In most instances haematuria and urine extravasation will resolve spontaneously with no intervention. Persistent extravasation of urine is a relative indication for surgery, as is devitalised renal parenchyma. For unstable patients with suspected renal injury it has been suggested that 'on-table' intravenous urogram is performed as part of the laparotomy, prior to renal exploration.[13]

Grade 1	Contusion	Haematuria (micro or macroscopic). All imaging normal.
	Haematoma	Subcapsular
Grade 2	Haematoma	Confined to renal peritoneum
	Laceration	<1cm depth. No urinary extravasation (renal pelvis not involved)
Grade 3	Laceration	>1cm depth. No urinary extravasation. Renal pelvis not involved.
Grade 4	Vascular	Injury to renal vessels. Haematoma contained.
	Laceration	Extending through cortex, medulla and pelvis.
Grade 5	Vascular	Avulsion of renal hilum – devascularised kidney
	Laceration	Shattered kidney

Figure 6 – AAST grading of traumatic renal injury (20).

Pancreas

Blunt trauma may result in pancreatic injuries such as duct disruption or avulsion, pancreatic contusions, lacerations, fractures and acute pancreatitis. Although only 3% to 12% of patient will have a pancreatic injury, there is a surprisingly high associated mortality, of 16% to 20%.[13] Complications of pancreatic trauma include pseudocyst development, abscess formation, fistula formation and as mentioned, pancreatitis. Diagnosis is often on clinical suspicion, as biochemical changes, such as raised amylase and raised white cell count are often not evident for 24 to 48 hours after injury and initial CT findings are also often equivocal.[21]

Vascular injury

Vascular injury as a result of blunt abdominal trauma is substantially less frequent than solid organ injury. The portal vein is reported as the most common of these, with inferior vena cava, iliac artery and mesenteric root being less frequent. Of note, injury to the portal vein is almost always associated with complete pancreatic rupture.[17] Vascular injuries such as these often result in a haemodynamically unstable patient and proceed quickly to emergency laparotomy.

Hollow viscus injury

Damage to the urinary system and bowel as a result of blunt abdominal trauma is uncommon. Less than 5% of those presenting with blunt abdominal trauma will have a bowel injury.[22] Particularly at risk is the duodenum, as it is retroperitoneal for most of its course, so is fixed in position. This results in it being compressed against the spine, especially in road-traffic collisions by the car seat belt. Unfortunately bowel injuries are poorly visualised by both ultrasound and CT scan in comparison to other injuries, resulting is delay to diagnosis, surgery and complications such as abscess formation, peritonitis and sepsis. Bladder rupture is rare, but may be suspected if blunt trauma occurred when the bladder was full. Of those with initial haematuria most settle spontaneously within 48 hours and do not usually require further intervention.

Mesenteric injury

Even less common than hollow viscus injury is mesenteric injury, occurring in 1% to 5% of blunt abdominal traumas.[23] Damage to the bowel mesentery as a result of compression or shearing forces can rupture the blood vessels contained within it. The resulting haemoperitoneum may be evident on ultrasound scan, DPL and CT scan. However, identifying the source of the bleeding is difficult due to imaging limitations. In presence of a haemoperitomeum, but not obvious solid organ injury, a site of mesenteric injury should be sought. As blunt abdominal trauma so frequently results in damage to more than organ, mesenteric injury often goes undiagnosed unless laparotomy is undertaken for another reason.

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Questions

1. Which is the most appropriate method confirmation of intra-abdominal injury in a patient from a road traffic collision with BP 90/55, Pulse 110, RR 20, Sats 99% on 15L oxygen.

a) Abdominal ultrasound
b) Computed tomography
c) Diagnostic laparoscopy
d) Diagnostic peritoneal lavage

2. Which of these abnormalities would you discover and treat first during your primary survey.

a) Blood glucose of 3.1
b) Open fracture of forearm
c) Foreign material in the oral cavity
d) Tension pneumothorax
e) Blood pressure 70/35

3. Which of these is not usually a site of profound haemorrhage

a) Long bones
b) Pelvis
c) Thorax
d) Cranium
e) Abdomen

4. A patient sustains an isolated small bowel rupture due to blunt abdominal trauma. Which investigation is most likely to identify this?

a) CT scan
b) Angiography
c) Diagnostic peritoneal lavage
d) Ultrasound scan

5. Which of these is the least appropriate management for a stable patient with a low grade splenic injury on CT scan.

a) Emergency laparotomy
b) Observation
c) Therapeutic angiography and embolisation
d) Discharge from hospital
e) Repeat CT scan in 48 hours

Answers

1. a). In most UK centres FAST scanning is readily available and would quickly confirm the abdomen as the site of haemorrhage before proceeding to laparotomy. NB – in the absence of available ultrasound diagnostic peritoneal lavage may be used to confirm intra-abdominal injury.

2. c). Following the ABCDE algorithm of ATLS you would discover and remove foreign material in the oral cavity as part of your assessment of the airway.

3. d). Although intracranial haemorrhage can be catastrophic for other reasons, the volume of blood potentially contained within the cranial vault is not large enough to result in haemodynamic instability.

4. c). The presence of faecal matter in the aspirated fluid from DLP would indicate a bowel injury. CT scan may be able to identify this, but DLP is far more accurate.

5. d) All of the above except D may be indicated in this patient, depending on he circumstances, at this stage.

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NON ACCIDENTAL INJURY IN CHILDREN

M Petrie & TA Coughlin

Non Accidental Injury in Children
Trauma & Orthopaedic Surgery

Abstract

Non-Accidental injury forms part of the spectrum of child abuse, usually caused by the parent or caregiver. As a clinician, one must maintain a high index of suspicion and manage any injured child in a safe environment.

Keywords: fracture, non-accidental injury, paediatric

Case Vignette

A seven-week old boy presented to A&E with his mother complaining of a painful left arm. The history given was that the father was trying to put the boy's jumper on to go out when he heard a crack. The child did not cry and so nothing more was thought of it. His mother took him out and only later when they got home and he was crying inconsolably did the grandmother recommend seeking medical attention. In the Emergency Department radiographs taken revealed a spiral humeral fracture following which he was admitted. NAI was considered as a possible cause of injury. The fracture was treated conservatively. He was seen by the Orthopaedic Consultant who suggested investigation for osteogenesis imperfecta and osteopenia but concern persisted that this was NAI. After further questioning by the social workers, the story had changed enough to raise concern further. Bruising to the lower limbs and sacrum was noted. Opthalmoscopy was normal.

The concerning features in this case include; delayed presentation, change in history and a significant fracture with no appropriate mechanism of injury.

The paediatricians investigated the child further with head CT and MRI and a skeletal survey. The findings were; multiple healing rib fractures, metaphyseal fractures of the proximal and distal left tibia and the distal left femur. The decision was made to place the child with a foster family and a social services assessment of the family situation was instigated.

Introduction

Non accidental injury (NAI) is part of the spectrum of child abuse. It describes the physical abuse of an infant (aged < 1 year), child (aged 1-13 years) or young person (aged 13-17 years). It may occur on its own or in combination with emotional abuse, sexual abuse, neglect or fabricated or induced illness (Munchausen by proxy syndrome[1]). The perpetrator is often the child's caregiver and the diagnosis should be considered in any child with injuries for which there is no reasonable explanation.

It is difficult to obtain exact epidemiological data regarding NAI as it is both under-reported and under-diagnosed. A survey by the National Society for the Prevention of Cruelty to Children (NSPCC) found that; 7% of children had suffered serious physical abuse, 6% had suffered serious neglect, 6% emotional abuse, 11% sexual abuse from an unrelated but known person and 4% sexual abuse from within the family. Child abuse causes at least 1-2 deaths per week in England (2).

Recognising NAI is difficult but it is critical. Abuse is the second leading cause of mortality in infants and children. If a minor case of NAI is missed and the child is returned to the home, there is a subsequent risk of serious injury in 25% and death in 5%.

The definition of physical abuse when consideration is given to place a child on a Child Protection Register is, "Actual or likely physical injury to a child, or failure to prevent physical injury (or suffering) to a child, including deliberate poisoning, suffocation and Munchausen by proxy syndrome."

Risk factors for NAI are poverty, learning disability, single parents, substance abuse and a history of abuse to the caregiver. The age distribution of NAI is significantly weighted towards younger children with a significant percentage of victims being under the age of 3.

That said NAI can occur within any social circumstance and must be considered whenever the injury is not consistent with the history. Any injury sustained in a totally dependent, non-ambulatory child should be considered to be suspicious.

There is strong evidence of the harmful sequelae of NAI, both in the short and long-term, with health, development and wellbeing affected. These effects can last throughout adulthood and include anxiety, depression, substance misuse and behavioral disorders affecting future relationships, careers and parenting capacity.[3]

Once concerns are raised over possible NAI, steps must be taken to investigate the case and to prevent further injury. The appropriate clinical lead for safeguarding children or consultant paediatrician must be informed as per local hospital guidelines. The clinician who first raises concern regarding possible NAI has the ultimate responsibility to ensure that the case is escalated and that the appropriate care pathway is initiated and completed. There have been several high-profile cases in the media that highlight how a lack of continuity of care and clinical responsibility can end in tragic circumstances.

NON ACCIDENTAL INJURY IN CHILDREN

M Petrie & TA Coughlin

Initial Management

When managing a child suspected of suffering from NAI, the key is for the child to be in a safe supportive environment. Keep any adults with the child clearly informed of any concerns and more importantly, why suspicions have been raised. Remember that an injury suspected of being NAI may in fact have an innocent explanation or the adult with the child may not be the perpetrator and may be unaware any abuse has occurred.

History: Begin taking the history by asking open questions directed to the child, if they are able to answer. If not, then start taking a history from the carer with them. It is important to also take a history from the child alone, if possible, which requires parental consent. It is important to approach this in a tactful way with the parent, so as not to provoke an understandable emotional response to a potentially confrontational situation. If the parent refuses the interview this should raise suspicion of NAI further. Red flags in the history for abuse are outlined in Table 1. A family history of osteogenesis imperfecta,4 osteopenia or any other associated medical conditions should be excluded. One should also take note of the child's demeanor, appearance and the interaction between the child and parent or carer.

History	Incomplete or inconsistent history.
	Delayed presentation.
	Severity of injury out of proportion to the stated mechanism.
	Injury inconsistent with the developmental stage.
	Refusal of parent to allow questioning of the child alone.

Table 1: Red flags in the history for abuse.

Examination: The examination must be general as well as focal so no injury is missed. A body map should be used to provide accurate documentation of external injuries. The site, size, shape, colour and pattern of injuries should be documented. Throughout the examination it is critical that a chaperone is present.

- **Start with a general examination of the patient, including height and weight measurements (this will identify failure to thrive).**
- **Examine the whole body paying special attention to the scalp, mouth (look for a torn frenulum), gums, eyes and ears.**
- **Examine the external genitalia for trauma, including the perineum.**
- **Perform ophthalmological examination as retinal haemorrhage may be the only indicator of trauma.[5]**
- **If medical photography is available then it is a useful way of accurately documenting injuries. This can be important later on should a police investigation be necessary.6 It is best practice to gain consent from the parent/guardian though this is not essential if a child states they have been the victim of abuse.[7] Consultant consent can be used for patients who are not competent to sign on their own.**

When examining and documenting injuries it is important that descriptions follow the definitions below.

- **Abrasion: superficial scrape to the skin.**
- **Bruise: leak of blood from vessels causing skin discoloration.**
- **Incision: clean cut to the skin**
- **Laceration: tear or split in the skin**

Documentation: Accurate and detailed note-keeping is of the highest importance when NAI is suspected. Any concerns that you or other healthcare professionals have and the reason why, should be documented. Record keeping is also important in highlighting frequent, or unusual patterns of presentation. This is often overlooked in practice meaning cases can slip through the net.[8]

Signs of NAI

Presentation can be varied. The key is to remain vigilant and consider NAI in all injured children. There are types and patterns of injury which make NAI more likely and should raise suspicion further. Below we discuss the most common presenting injuries associated with NAI. The red flags in examination findings are outlined in Table 2.

Bruising: Bruising is the most common sign of NAI. However, on its own it is far from diagnostic. Accidental bruising in an ambulatory child is often seen on bony prominences such as the anterior tibia, knee, upper leg and forehead. However, bruising on the face, trunk, buttocks, lower back and ears is rarely accidental.9 Where bruising in suspicious sites is seen, only a consistent and plausible history should allay fears.

The number of bruises or bruises in clusters should be noted and suspicions raised when a large number is seen. One study showed that in a normal mobile child 20% will have more than 5 bruises, 4% over 10 and only 1% will have over 15.[10]

The age of bruises is difficult to clinically diagnose and so considering this is not usually helpful.[11] However, the morphology of bruises can be very useful. Unusually shaped bruises should elevate suspicion, especially if the resemble objects such as a hand, ligature, bite or implement (eg belt buckle, hairbrush).

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Burns: Burns are relatively uncommon in children. Up to 25% of those seen will be as a consequence of NAI. As with bruising the morphology and site of burns should be considered. Mild palmar burns may be self inflicted if the child touches something hot. Immersion burns to the hands, feet and buttocks may be caused intentionally or accidentally by immersion in hot water. Recognisable patterns include cigarettes, cutlery and curling irons causing a particular pattern of branding on the skin.[12] A specialist burns unit should be consulted in any patient with more than 5% full thickness or 10% partial thickness burns.

Fractures: Fractures are the third most commonly seen sign of NAI behind bruising and burns. They are disproportionately seen in younger children with 85% occurring in children under the age of 3. Red flags for suspected NAI include fractures of differing ages and radiographic evidence of occult fractures. A number of fractures have been shown to be highly specific for NAI:

Metaphyseal fractures (figure 1): these fractures are usually a consequence of NAI and were first described by Caffey in children with concurrent head injury consistent with abuse.13 There are two types; corner fractures and bucket handle fractures. They are caused by shear forces on the physis (growth plate).

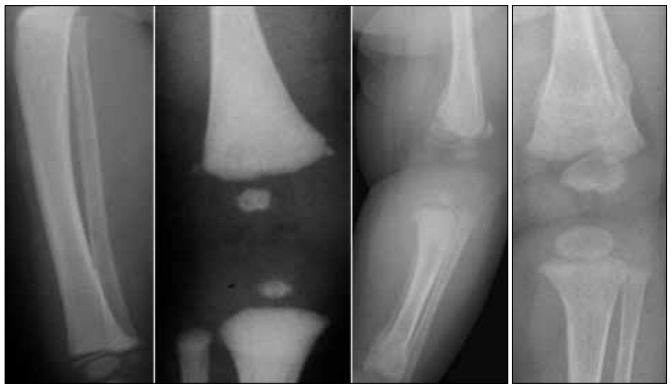

Figure 1: a) Bucket handle fracture of the distal tibia b) Metaphyseal corner fracture of distal femur in a 2 week old c) Metaphyseal fractures of distal femur and proximal and distal tibia of differing ages d) mature femoral fracture discovered on skeletal survey in a child presenting with a torn frenulum

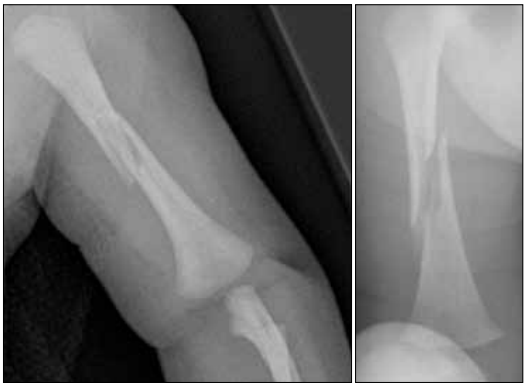

Figure 2: a) Humeral shaft fracture in a 2 week old; b) Spiral femoral shaft fracture in a 4 week old

Long bone fractures (figure 2): humeral shaft fractures under the age of three and femoral fractures under the age of one are unusual in accidental injury. Fractures of the digits in non-ambulatory children are also suspicious.

Rib fractures (figure 3): the ribs in children are more flexible than in adults and so fractures are rare (14). Injury to the ribs posteriorly is particularly suspicious and is commonly caused by squeezing the child's chest.

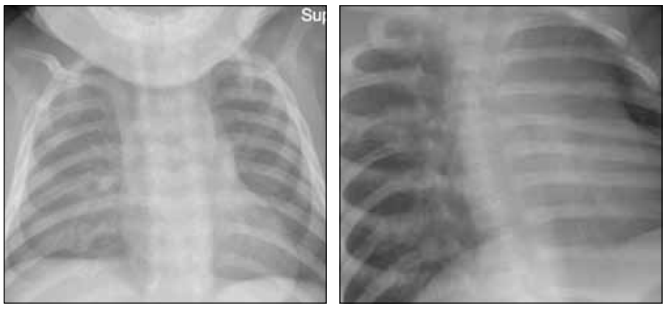

Figure 3: a) right-sided rib fractures on AP (anteroposterior) view b) posteromedial right sided rib fractures on oblique view.

Other fractures (figure 4 and 5) with a high specificity for abuse include those of the scapula, lateral end of the clavicle, vertebrae and complex skull fractures (non-parietal)[15]

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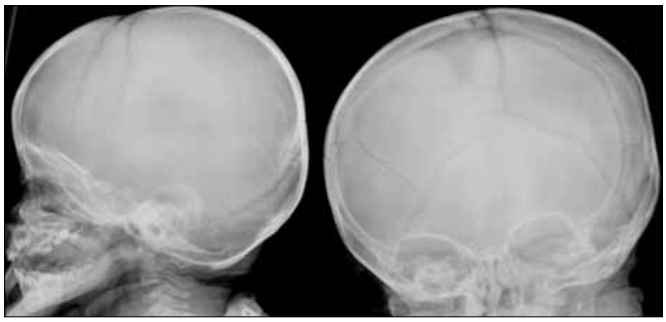

Figure 4: parietal skull fracture in a 3 month old baby (non-ambulatory).

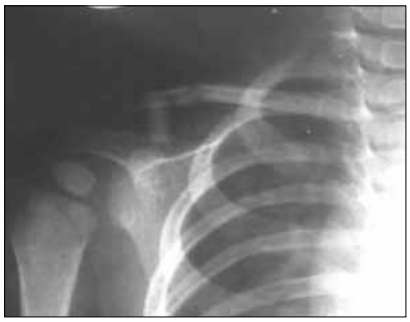

Figure 5: right-sided clavicle fracture.

Bites: Human bites leave a specific pattern on the skin. This pattern can be used to match the injury to a dental record in an investigation so photographic documentation is critical. Human bites usually cause crush injury and bruising rather than an actual break in the skin.

Binding Injuries: these occur when the hands or feet are tied together. Acutely this will present with abrasions, erythema and swelling. Healed injuries may still be seen as cutaneous pigment changes.

Internal Injuries: A violent attack may also cause internal injuries and these must be borne in mind when assessing the child. Abdominal trauma may cause rupture of the liver, spleen, kidney or bowel. Chest trauma may result in pneumothorax or cardiac injury such as haemopericardium. Any of these can be life threatening injuries and so must be identified and treated expeditiously.

Examination	Bruises	Site: non-bony prominences (face, trunk, buttocks, lower back and ears)
		Shape: (hand, ligature, teeth mark grip or implement)
		Multiple, clusters, differing ages
	Burns	Immersion burns (glove-stocking)
		Shape: (cigarette, iron, curling iron)
	Fractures	Differing ages and occult fractures
		Metaphyseal fractures
		Long-bone fractures
		Rib fractures
		Scapula, lateral end of the clavicle, vertebrae and complex skull fractures (non-parietal)
	Human Bites	Shape of bruising - photographic evidence
	Ligatures	Binding lacerations to ankles/wrists
	Internal Injuries	Rupture of liver, kidney, spleen, bowel
		Haemopericardium or pneumopericardium

Table 2: Examination findings that should raise suspicion for NAI.

Investigation

The investigation of NAI very much depends of the presenting condition of the child and the suspected diagnosis. Naturally when the child is acutely unwell the priority of investigation changes.

- **Blood Tests:** In cases of bruising a full blood count and clotting screen should be performed to rule out coagulopathy.[16]

- **X-ray:** A skeletal survey is a standard series of X-rays to visualise the entire skeleton.[17] Careful consideration must be given as a full skeletal survey is a lot of radiation for a child. It is indicated in all children under the age of 2 where NAI is suspected and in any child age 2 to 5 with a suspicious fracture.[18] It should be performed within 24 hours of admission except where the child is medically unfit when it may be deferred for up to 72 hours. Delayed X-rays where initial radiological findings are equivocal can improve detection of bony lesions by up to a quarter.

- **Scintigraphy:** Where plain X-ray remains equivocal scintigraphy (bone scan) is the supplementary imaging modality of choice.[19] A bone scan becomes positive 7 hours post fracture. Bone scans are sensitive but not specific for fractures so where a suspicious lesion is seen follow up x-rays should be taken.

- **CT:** CT is used in all children where head trauma or significant thoracic or abdominal injury is suspected.[20] It may also be used to assess complex injury to the axial skeleton or joints. CT neuro-imaging should also be performed in any child under one who has confirmed NAI due to the difficulty in assessing these patients for neurological injury. MRI may also be used to augment imaging in the same way.[21]

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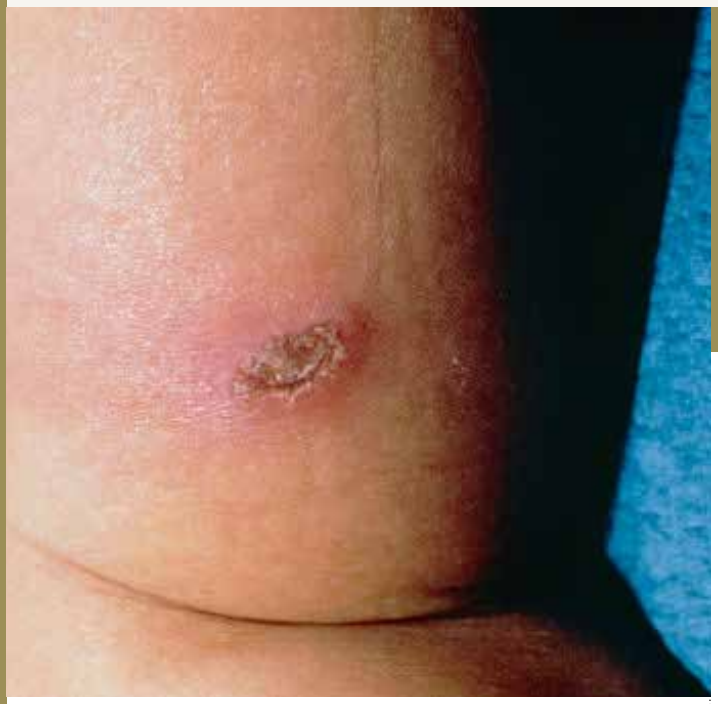

Summary

Non-Accidental injury forms part of the spectrum of child abuse, usually caused by the parent or caregiver. It is recognised that NAI is under-reported and under-diagnosed but figures approximate that 7% of children have been affected by physical abuse.[2] The long-term physical and psychological effects can persist into adulthood in the form of depression, anxiety, substance abuse and behavioral difficulties. They may also manifest as abuse of the patient's own children creating a vicious cycle.

As a clinician, one must maintain a high index of suspicion and manage any injured child in a safe environment. Good communication with the child and parents or carers is vital in establishing a professional relationship. Detailed and accurate record-keeping is essential. Once suspicion has been raised by any healthcare worker, concerns must be escalated to the appropriate clinical lead for safeguarding children or a consultant paediatrician. The responsibility does not stop at referral. The clinician who initially raises concerns regarding possible NAI is responsible for following up the case and ensuring that all appropriate steps are taken to investigate and prevent further injury

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Questions

1. Which of the following parts of the history should not necessarily raise suspicion of NAI?

a) Incomplete or inconsistent history
b) prompt presentation to seek medical attention
c) severity of injury out of proportion of mechanism
d) injury inconsistent with developmental stage
e) refusal of the parent to allow questioning of the child when alone

2. Which of the following factors when examining a bruise should not necessarily raise suspicion of NAI?

a) bruising on a non-bony prominence
b) multiple bruises of potentially differing ages
c) clusters of bruises
d) bruising on the anterior shin in an ambulatory child
e) bruises which take the shape of a belt-buckle

3. Which of the following factors when examining a burn should not necessarily raise suspicion of NAI?

a) burns in a glove and stocking distribution in a non-ambulatory child
b) a burn that takes the shape of a cigarette
c) burns to the palms in an ambulatory child
d) burns to the perineum
e) symmetrical burns to the limbs

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4. Which of the following fractures should not necessarily raise suspicion of NAI?

a) fracture of the femur
b) rib fractures
c) fracture of the parietal skull in an ambulatory child
d) fracture of the digits in a non-ambulatory child
e) fracture of the scapula in an ambulatory child

5. In which of the following circumstances should one consider a skeletal survey for further investigation of possible NAI?

a) 7 year old boy with a mid-shaft humeral fracture
b) 18 month old girl who has cluster of bruises around her back
c) 4 year old boy who has a metacarpal fracture
d) 3 year old ambulatory boy with a mid-shaft radius and ulna fracture
e) 5 year old boy with a tarsal fracture

Answers

1. b) 2. d) 3. c) 4. c) 5. b)

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A STEP-BY-STEP GUIDE TO FULL THICKNESS SKIN GRAFTING

R Jordan, E Dickenson, E Ginn & J Hardwicke

A Step-by-step guide to full thickness skin grafting
Plastic & Reconstructive Surgery

Abstract

Full thickness skin grafting offers the surgeon one option when faced with a skin defect. The technique involves excision of a graft including both the epidermis and dermis requiring primary closure of the donor site. The recipient site must be optimised to improve the chance of graft take which comprises of three stages; plasma imbibition, inosculation and revascularisation. This article explains the procedure in a step by step fashion including the indications and complications.

Keywords: Full thickness skin graft, wound, reconstruction.

Clinical vignette

A 70 year old gentleman is seen in the plastics outpatient with a biopsy proven nasal basal cell carcinoma. The surgeon discusses the need for surgical treatment which involves excision of both the lesion and normal skin to leave 'clear margins.' The surgery will leave the patient with an elliptical skin defect that will not be able to be closed directly with sutures and recommends the patient has a full thickness skin graft. The surgeon discusses the procedure at length including; the technique, possible donor sites, advantages and disadvantages, possible complications and post-operative management. This article aims to cover these points of discussion whilst taking the reader through each step of the technique.

History and pathology

The skin is made up of epidermis and dermis. It functions as a protective barrier, a sensory organ and for thermoregulation. A wound is defined as any damage leading to a break in its continuity which may affect these functions. The reconstructive ladder (see Table 1) is used as a guide to the principles of wound management and skin grafts offer one option for wound coverage.

Level	Reconstructive procedure
1	Healing by secondary intention
2	Primary closure
3	Delayed primary closure
4	Skin graft
5	Local flap
6	Distant flap
7	Free flap

Table 1: The reconstructive ladder.

The key distinction between skin grafts and flaps is that grafts lack their own blood supply and are reliant on revascularisation to survive. In contrast soft tissue flaps are either transferred with blood supply intact or have their blood supply reconnected during the initial surgery. Skin grafting can be either full thickness (FTSG) containing the epidermis and dermis in its entirety, or split thickness (STSG) containing the epidermis and a variable proportion of dermis.

The technique used affects donor site healing with the layer of dermis left in STSG containing pilosebaceous follicles and sebaceous glands that allow direct resurfacing. In FTSG only the subcutaneous layer remains and this lacks the capacity to reepithelialise and the donor site requires primary wound closure. The need for primary closure restricts the size of FTSG that can be taken from any one site. The donor site is chosen to best reflect the excised skin in terms of colour and texture whilst minimising the cosmetic effect to the donor site scar. For example pre and post auricular skin grafts are commonly used for grafting facial skin defects.

For a graft to successfully 'take' it must progress through a series of stages; plasmatic imbibition, inosculation and revascularisation. To give the graft the best chance of success the recipient wound bed must be non-infected, haemostatic and have a good underlying blood supply. Plasmatic imbibition occurs over the first 48 hours and sees the graft adhere to the wound bed through the deposition of fibrin and receive nutrition by diffusion. From days 2 to 7 capillary buds grow out from the wound bed into this fibrin layer and form a fine vascular network, a process known as 'inosculation.' The final stage is 'revascularisation' and sees these newly formed vessels restore the blood supply to the graft. The pathophysiology of revascularisation is debated with hypotheses including the connection of these new vessels to existent graft vessels or the continued growth of these new vessels into the graft.

After placement skin grafts are susceptible to both primary and secondary contraction. Primary contraction occurs immediately and is a result of elastin fibres within the graft. For this reason FTSG, which contain more dermis and elastin, undergo more primary contraction than STSG. Secondary contraction originates from the recipient bed itself and as FTSG contain more dermis they are prone to less secondary contraction than STSG making them more suitable for use on hands and around the eyes. A wide variety of donor sites are available for FTSG and those commonly used are highlighted in Table 2.

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Donor site
Pre or post auricular
Upper eyelid
Neck
Supraclavicular
Forearm
Groin
Antecubital fossa

Table 2: Examples of commonly used donor sites for full thickness skin grafts.

Indications and contraindications

FTSG are indicated for skin defects where direct closure is not possible and the area is cosmetically sensitive (e.g. the face) or where contractures may cause a functional problem (e.g. hand). All grafts need a well vascularised recipient bed to improve chances of take and suitable wound beds include muscle, fascia, peritenon and periosteum. FTSG are more at risk of graft failure as they have a reduced number of blood vessels on their surface and due to their thickness have an increased metabolic demand. The presence of either infection or tissue that lacks a suitable blood supply (e.g. bare tendons, cartilage or bone) is a contraindication to skin grafting. If a large defect is present then the inability to close the donor site primarily may restrict the use of FTSG, although it is possible to use multiple donor sites and close these individually. The advantages and disadvantages of these techniques are given in Table 3.

FTSG	STSG
Smaller grafts	Larger grafts possible
Better characteristics: colour and texture	Poorer cosmesis
Poorer survival	Better survival
Less secondary contraction	More secondary contraction
Requires primary closure at donor site	Heal by reepithelialisation
Tolerate trauma better	Tolerate trauma poorly

Table 3: Comparing the advantages of full and split thickness skin grafts.

Gaining informed consent/explaining procedure to patient

When counselling patients a realistic discussion regarding the likely cosmetic outcome is required. The outcome is unlikely to match their pre-morbid appearance and they should understand that it may take from 3 to 6 months before the final appearance can be properly judged. The patient should be counselled about bleeding, infection and haematomas with the increased chance of complete or partial graft failure if these occur. The risks of possible donor site infection or dehiscence, anaesthetic and need for further surgery should also be included in this discussion.

Equipment required

A basic plastics set is required, the graft is first marked using a pen and then local anaesthetic is infiltrated into both donor and recipient site using different needles to reduce the spread of infection. The graft is harvested using a scalpel and any remaining adipose tissue is trimmed using curved scissors. Bipolar diathermy is used for haemostasis at both donor and recipient sites, before the donor site is closed primarily with a suture of choice. The graft is sutured in place and secured with a bolster dressing (typically made up of cotton wool soaked in flamazine). Crepe bandage, a plaster of paris or various splints maybe required to immobilise the recipient site.

Draping/sterile field preparation

Draping of the patient is dependent on the site of surgery, the drapes can be applied to expose both donor and recipient sites together if adjacent or via two separate sterile fields. Both areas should be prepped with chlorhexidine or iodine, although saline is suitable for preparing facial sites.

Patient positioning and relevant anaesthetic points

The positioning of the patient is again influenced heavily by the site of surgery with the patient usually supine for cases involving the face and an arm board used for hand cases. The use of local anaesthetic is permissible in a large proportion of cases and should be administered to both the donor and recipient site. Even if the patient is given a general anaesthetic they should also have local anaesthetic infiltrated to improve post operative analgesia.

Documentation of procedure

The procedure itself involves a number of stages that will each be discussed; preparation of donor site, preparation of recipient site, harvesting graft and insetting of the graft.

Preparation of donor site

1) The surgeon should examine and choose a donor site where skin colour and texture best match the recipient site and sufficient tissue is available.
2) A simple test as to how much skin can be donated is to pinch the skin at the predicted incision edges and see if the remaining skin apposes without excessive tension.
3) Local anaesthetic is usually infiltrated at this point, if the patient is under a general anaesthetic this can be delayed until the end of the procedure but should still be considered to aid analgesia.

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Preparation of recipient site (see Image 1)

1) Sharp dissection should be used to debride the recipient site leaving a clean and healthy wound bed.
2) Pressure, adrenaline soaked gauze or bipolar diathermy are used for haemostasis which reduces the change of subsequent haematoma formation.
3) The site is then protected with either a saline or adrenaline soaked gauze whilst the graft is prepared.

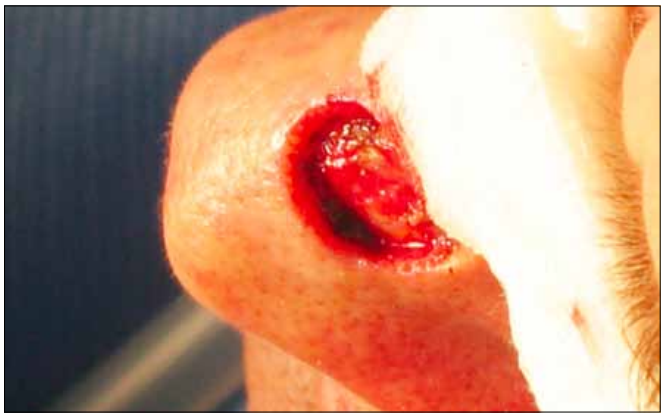

Image 1: The defect

Graft harvest

1) A pattern of the defect is taken using paper or foil and marked on the donor site. This pattern is then modified into an ellipse to allow for linear closure. (see Image 2)
2) An optional step is to infiltrate local anaesthetic (typically with adrenaline 1 in 200,000) which aids harvesting and reduces blood loss.
3) Harvesting of the graft is performed with a scalpel and the epidermis and dermis are carefully excised leaving subcutaneous tissue at the wound bed. This is best done by lifting one edge of the graft and keeping the tissue under tension during excision. (see Image 3)
4) Any remaining adipose tissue is then removed from the graft using curved scissors. (see Image 4)
5) Haemostasis is then ensured at the donor site using bipolar diathermy.
6) The wound is closed directly and a simple non-adherent and absorbent dressing is applied (e.g. opsite).
7) The donor site wound should be inspected at 5-7 days.

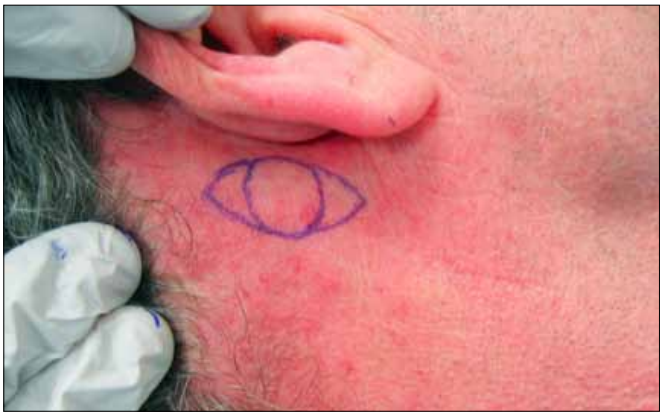

Image 2: The planned FTSG

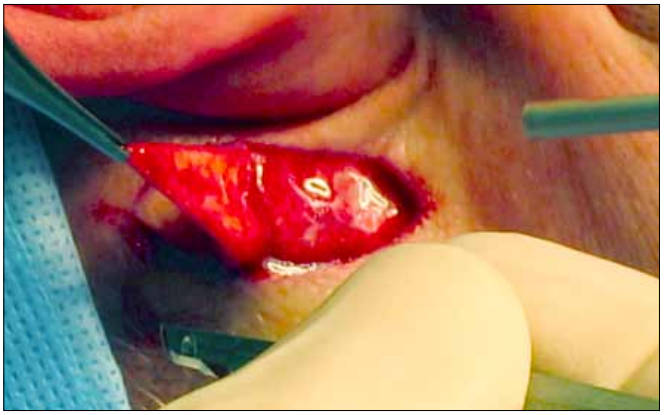

Image 3: FTSG being harvested

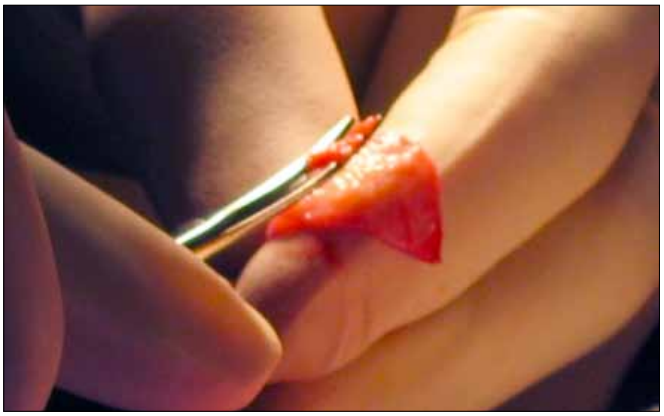

Image 4: FTSG being de-fatted

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Application of graft to recipient site

1) The harvested graft is cut to the pre-marked pattern.

2) The surgeon may choose to insert small cuts in the graft using a scalpel to allow wound exudates to escape.

3) The graft is placed dermal side down into the recipient site.

4) The graft is fixed in position using sutures with care to avoid inversion of the graft edges. This is typically performed with interrupted sutures to the four graft corners followed by a running stitch around the perimeter. (see Images 5 and 6)

5) A technique of 'quilting' the graft is occasionally employed in large wounds or those with a concave recipient surface to aid adherence. In this technique interrupted sutures are applied throughout the graft to the wound bed, before the recipient site is dressed as below.

6) The recipient site is then dressed, a non adherent dressing is an optional first layer that aids removal at graft check. A dressing bolster, typically composed of an absorbent cotton wool layer soaked in proflavine (a bacteriostatic agent), is carefully placed over the graft. This is usually secured with a suture tie over using a new stitch or sutures purposively cut long at the earlier stage, although an alternative method is to use skin staples. (see Image 7) Securing the dressing effectively prevents shearing forces and aids adherence of the graft to the wound bed.

7) Additional techniques to reduce shearing forces at the graft-wound interface are the use of a splints and keeping the patient initially on bed rest. Typical materials used for splinting are heavy bandaging, plaster of paris and thermoplastic splints. If the graft is placed on a joint or the patient is judged to be unreliable then these measures are more likely to be employed.

8) The dressings are usually left undisturbed for around 5 days, at which point the graft is assessed to see whether it has been successful. If the graft has taken and become adherent to the wound bed then the splinting can be discontinued at his point.

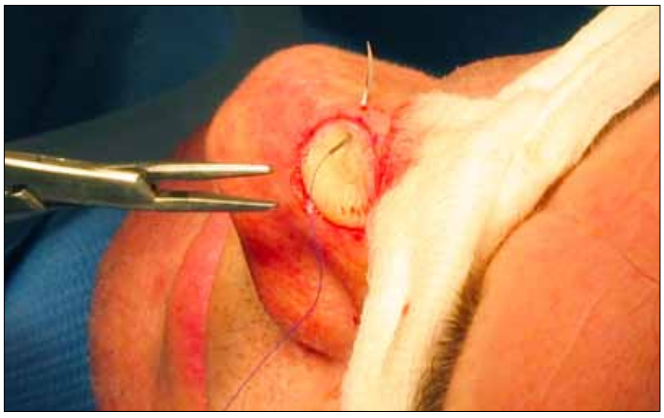


Image 5: Graft being sutured in place

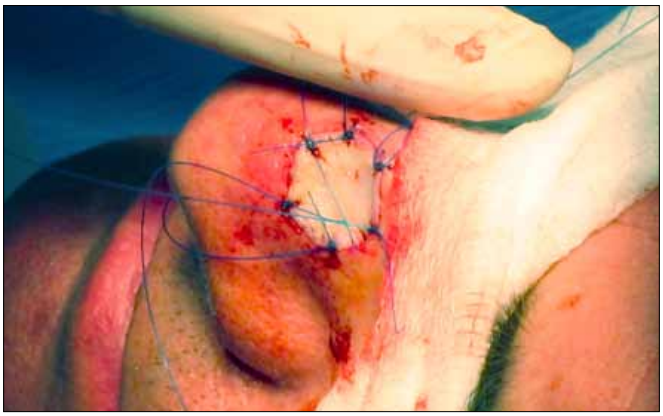


Image 6: Graft sutured in place.
Note sutures left long for tie-over of bolus dressing

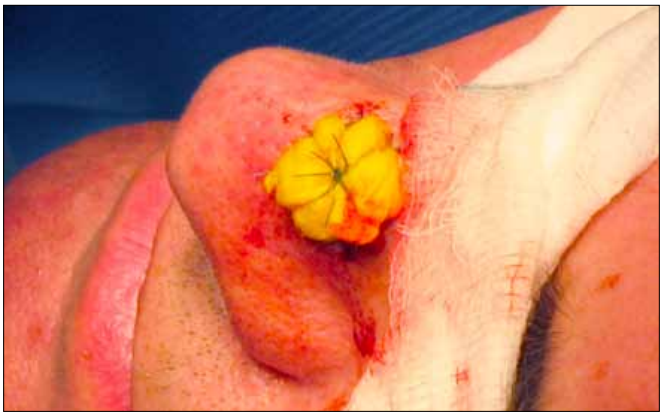


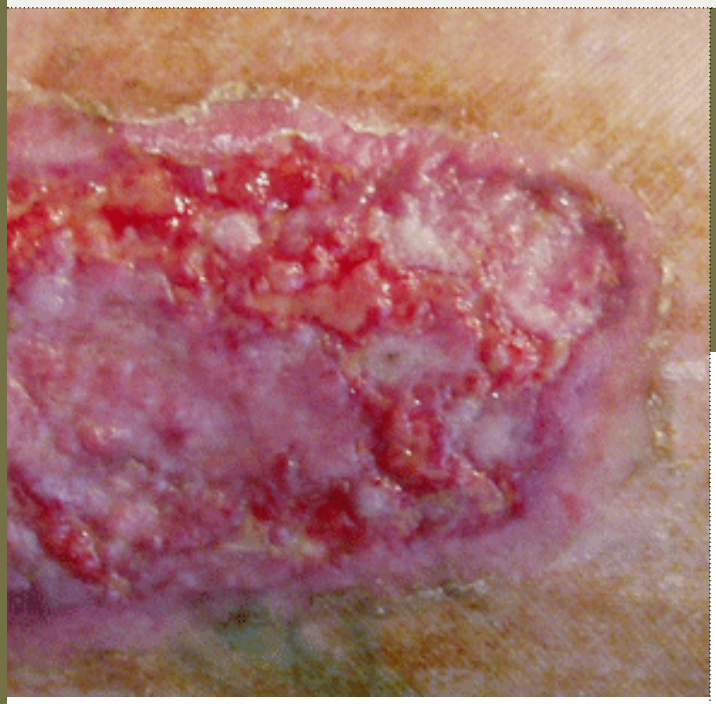
Image 7: Tie-over dressing applied

Recording of complications and management of such

The most common reason for graft failure is the development of a haematoma and strict haemostasis must be achieved before the graft is positioned to reduce this risk. If discovered post-operatively then a small incision should be made over the area and evacuation of the clot through this incision should be attempted. This method prevents disruption of any adhered graft surrounding the haematoma that would be lost if the clot were evacuated through the wound edges. Infection of the recipient site can result in graft failure and this risk can be reduced by meticulous surgical technique, preparation of the recipient site and effective dressings. Excessive shearing forces may prevent adherence of graft and result in failure. This scenario can be avoid by using suitable dressings and splinting the limb if appropriate.

A STEP-BY-STEP GUIDE TO FULL THICKNESS SKIN GRAFTING

R Jordan, E Dickenson, E Ginn & J Hardwicke



Bleeding from the recipient site may result in patients representing to the emergency department and puts the 'take' of the graft at risk. Successful treatment includes achieving haemostasis whilst attempting to salvage the graft. If active bleeding persists then under local anaesthesia, ideally in theatre, the graft should be carefully removed, haemostasis ensured and the graft should be reapplied according to the instructions already given. However if at assessment the bleeding has ceased then a decision must be made as to whether the graft remains adherent to the wound base, if this is not the case then the graft should be managed as a haematoma.

Graft failure can either be partial when a proportion of graft has taken or total when the entire graft has failed. Total failure requires the recipient site to be debrided either surgically or using dressings and the aetiology of failure identified before deciding on whether this factor can be corrected before further grafting is considered. Partial failure requires debridement of those unsuccessful areas whilst maintaining an optimum environment for those parts of the graft that have taken. Managed correctly then these failed areas will heal by secondary intention, whether this will produce an acceptable result is dependent on the proportion of graft taken, the recipient site and patient expectations. This process can be aided by the use of negative pressure therapy that removes wound exudates and bacterial load whilst promoting wound contraction and angiogenesis.

A Step-by-step guide to full thickness skin grafting
Plastic & Reconstructive Surgery

Questions True or false?

1) During the process of graft 'take':

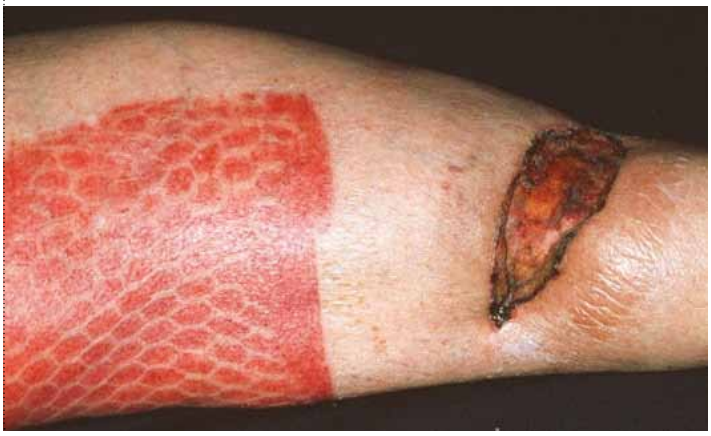
a) The graft is fully dependent on the in growth of new vessels for nutrition.
b) Fibrin is responsible for adherence of the graft during the first stage.
c) The graft passes through four stages; plasmatic imbibition, contraction, inosculation and revascularisation.
d) The process of revasularisation typically occurs after day 7.
e) FTSGs are less at risk of graft failure than STSGs.

2) The surgeon should remember that full thickness skin grafts:

a) Are only indicated for the reconstruction of cosmetically sensitive areas.
b) Leave varying degrees of dermis in-situ depending on the area of the body from which they are recruited.
c) Undergo a higher degree of primary than secondary contraction.
d) Can only be performed under a general anaesthetic.
e) Are matched according to the colour and texture of the donor site.

3) Regarding the donor site:

a) Selection is solely dependent on where sufficient tissue is available.
b) Inconspicuous areas are preferred.
c) Healing occurs via secondary intention.
d) Should routinely have dressings left in place for two weeks without inspection
e) Complications include infection and poor cosmesis.



A STEP-BY-STEP GUIDE TO FULL THICKNESS SKIN GRAFTING

R Jordan, E Dickenson, E Ginn & J Hardwicke

4) When the graft is applied:

a) A well vascularised wound base improves chances of survival.
b) The quilting technique is used to help keep dressings in place.
c) Trimming of adipose tissue improves chances of the graft taking.
d) The graft can be fenestrated to aid adherence.
e) The graft must always be checked at 48 hours.

5) When considering complications:

a) Infection is the commonest cause of graft failure.
b) The risk of a haematoma can be avoided by meticulous haemostasis.
c) The development of a haematoma seals the fate of the graft which must then be removed to allow healing.
d) Splints can be employed to help adherence of the graft and prevent shearing forces.
e) Any graft failure is an absolute contra-indication to further attempts.

Answers

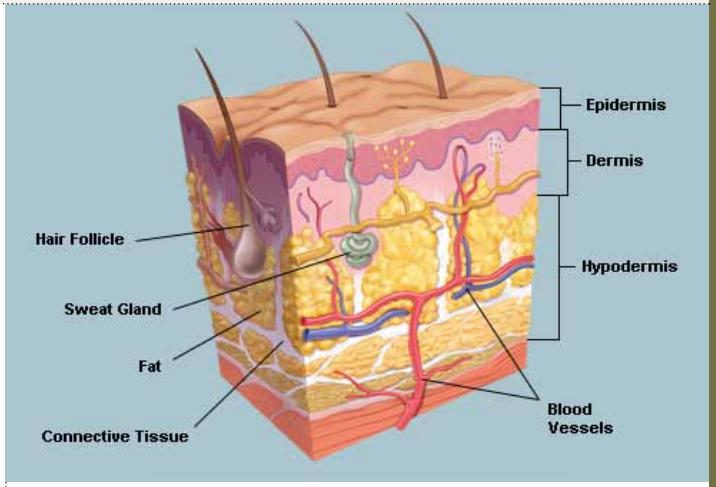
1) False, True, False, False, False

2) False, False, True, False, True

3) False, True, False, False, True

4) True, False, True, True, False

5) False, True, False, True, False



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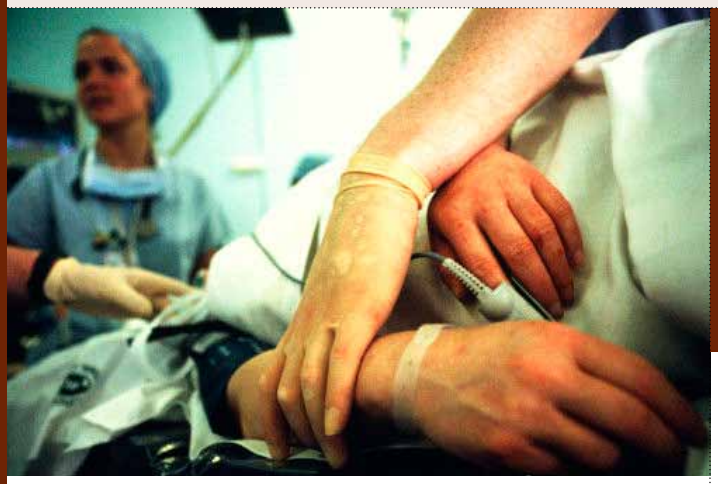
NON-INVASIVE AND INVASIVE MONITORING DURING ANAESTHESIA

A Radford & LP Gan



Abstract

There is substantial evidence that monitoring patients during an anaesthetic reduces the risk of critical incidents. In the UK, the Association of Anaesthetists of Great Britain and Ireland have set the standards which must be used whenever a patient is anaesthetised. This article aims to provide an overview of the different types of monitoring used in this minimum standard, as well as additional more invasive monitoring commonly used during anaesthesia. The indications for the use of different monitors of patient physiology are discussed, along with the basic principles of how they work and common disadvantages and pitfalls associated with their use. An understanding of why different monitoring is used is vital to anyone working in the operating theatre and knowledge of how this equipment works and its limitations can avoid potentially serious errors.

Keywords: Non-invasive, invasive, monitoring, Pulse oximeter, Blood pressure, Electrocardiogram, oxygen analysis, carbon dioxide, cardiac output.

Monitoring of physiological variables is routine for all patients undergoing an anaesthetic. There is substantial evidence that monitoring patients during an anaesthetic reduces the risk of critical incidents.[1-3] This article aims to provide an overview of the different methods used to monitor patients during anaesthesia, the basic principles of how this is achieved, indications for their use and the common pitfalls when using different equipment.

Clinical Vignette

A fit and well 45 year-old man presented to A&E with abdominal pain two days post investigative colonoscopy. A CT scan showed bowel perforation and he was booked onto the CEPOD list for a laparotomy. On examination he was alert and orientated, peripherally well perfused and his observations were normal except for a sinus tachycardia of 110 bpm. The anaesthetic team therefore decided that two wide bore cannulae and an oesophageal Doppler would be sufficient to manage him intra-operatively. Routine monitoring included an automatic non-invasive blood pressure cuff, a pulse oximeter, an electrocardiogram (ECG) and connection to an anaesthetic machine which provided monitoring of airway pressures and volumes, carbon dioxide, oxygen and anaesthetic vapours.

An oesophageal temperature probe was also inserted. On surgical exploration, however, there was a significant perforation of the transverse colon with faecal soiling of the peritoneum. As a result an arterial line and central line were inserted intra-operatively to provide closer monitoring of the patient's blood pressure and allow regular arterial blood gas sampling and central venous access. The patient was then transferred to ITU post operatively.

In the UK, the Association of Anaesthetists of Great Britain and Ireland (AAGBI) has set the standards of monitoring which must be used whenever a patient is anaesthetised. These minimum standards are uniform irrespective of duration, location or mode of anaesthesia.[4]

The presence of an appropriately trained and experienced anaesthetist is the main determinant of patient safety during anaesthesia.[2,5] The patient's physiological state and depth of anaesthesia needs continual assessment. Monitoring devices will supplement the clinical assessment of the anaesthetist. This clinical assessment may include: mucosal colour, pupil size, response to surgical stimulus and movements of the chest wall and/or reservoir bag, assessment of the patient's pulse, auscultation of breath sounds and measurement of urine output and blood loss.[4]

The minimal monitoring equipment required in a spontaneously breathing anaesthetised patient includes:

- **Pulse oximeter**
- **ECG**
- **Non-invasive blood pressure**
- **Airway gases: oxygen, carbon dioxide and vapours**

In addition if the patient is artificially ventilated they require monitoring of:

- **Airway pressure**
- **Ventilation volume**
- **Ventilation disconnection warning**

Also available should be a means of temperature measurement and (whenever muscle relaxant is used) a nerve stimulator.[4]

NON-INVASIVE AND INVASIVE MONITORING DURING ANAESTHESIA

A Radford & LP Gan

Non-invasive monitoring

Pulse oximeter

The pulse oximeter is a convenient, automatically calibrated, non-invasive monitor of oxygen saturation and arterial pulsation. It is widely used throughout hospitals in the UK but perhaps its greatest value is the ability to provide an early warning of hypoxaemia.[3]

The pulse oximeter consists of a probe and a processing/display unit. The probe contains two LEDs which illuminate the tissue with monochromatic light of red (660nm) and infrared (940nm) wavelengths. The light is then detected by a photodiode, converting it to an electrical signal. The different wavelength light is absorbed by deoxyhaemoglobin and oxyhaemoglobin to different degrees. The difference in absorption of these two wavelengths allows calculation of the concentration of oxyhaemoglobin compared with deoxyhaemoglobin (% saturation)[6, 7]

The two LEDs are cycled, including an 'off' period when both LEDs are off to correct for ambient light. This cycle is then repeated at high frequency, several hundred times a second. As a result the oximeter detects the change in absorption due to arterial pulsations. A microprocessor is programmed to ignore the non-pulsatile component of the signal which allows for the monitoring of the arterial oxygen saturation. The computer then uses internal algorithms to calculate the percentage saturation based on the absorption/transmission of the two light wavelengths.

The calculations used by the pulse oximeter are calibrated based on data from human volunteer studies using arterial blood gas samples while subjects breathed various oxygen concentrations. On ethical grounds studies were limited to minimum oxygen saturations of 80%. As a result, saturation values below this have been obtained by extrapolation and are subject to increasing error.[6]

Other sources of error include states of poor tissue perfusion where the reduced perfusion decreases signal amplitude. This means that the oximeter compensates by amplifying the total signal which worsens the signal to noise ratio. When noise becomes the predominant signal the reading will display a false saturation of 85%. Modern machines do however have signal to noise ratio limits above which the display is interrupted which can provide a useful warning of poor peripheral tissue perfusion.

Carboxyhaemoglobin (from carbon monoxide poisoning) has a similar absorbance of red light at 660nm to oxygenated haemoglobin which results in overestimation of the oxygen saturation. Conversely, methoxyhaemoglobin (caused by either congenital enzyme deficiency or certain oxidising drugs such as dapsone, local anaesthetic agents, aniline dyes and nitrates) leads to underestimation of the true oxygen saturation level.[8] Anaemia can also lead to underestimation of oxygen saturation levels.[6]

Non-invasive blood pressure

Manual occlusive cuff

Historically the earliest method of blood pressure measurement, the manual occlusive cuff is a simple method which does not require a power source or sophisticated equipment. In 1905, Korotkov first described the measurement of blood pressure using an occlusive cuff and auscultation over the occluded artery. Termed Korotkov Sounds, as the extent of arterial occlusion from the cuff decreases, five phases of sound as a result of turbulent blood flow and vibration of the arterial wall can be heard. The appearance of sound representing systolic pressure, and its disappearance representing diastolic pressure.[9] The main disadvantages of this technique are that it is dependent on the operator's technique and it requires manual intervention.

Automated occlusive cuff

The automatic occlusive cuff allows the operator's hands to be freed and measurements can be obtained conveniently when access to the patient is difficult. It allows calculation of the mean arterial pressure and has the capacity for alarms and data transfer.

Oscillometry is the most common method of automatic blood pressure measurement and is the main method used during anaesthesia.[10] A single cuff is used and a pressure transducer measures both the pressure and the oscillations in pressure caused by the arterial pulse. As the cuff is deflated, these oscillations start when the systolic pressure is reached, reach a maximum at the mean arterial pressure and decrease at a less precise point around the diastolic pressure. The cuff is inflated automatically to a suitably high initial pressure, for example 160mmHg or in repeated readings, 25mmHg above the previous systolic measurement. The pressure is then slowly released at 2-3mmHg per second.

Although correlating well with invasive blood pressure measurements, the automatic cuff is less accurate at extremes of blood pressure and over-reads at low pressures while under-reading at high pressures. The automatic cuff assumes a regular cardiac cycle and is therefore less accurate in dysrhythmias such as AF. The measurement is also dependent on appropriate cuff size and tends to over-read if too small a cuff is used and under-read if the cuff is too large for the patient's arm. There is also an increased risk of tissue damage from the cuff, especially when the frequency of measurements is high and used for prolonged periods.[10]

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Electrocardiogram (ECG)

An ECG is used during anaesthesia to monitor the rate and regularity of the heartbeat and is useful for the detection of cardiac ischaemia and dysrhytmias. The ECG monitors the electrical activity of the heart with electrical potentials of 0.5-2mV at the surface of the skin. The bipolar leads (I, II, III, aVR, aVL and aVF) measure the voltage difference between two electrodes. The unipolar leads (V1-V6) measure the voltage at different electrodes relative to a zero point.

Skin electrodes detect the electrical activity of the heart. This signal is boosted using an amplifier. It also filters out electrical frequencies considered to be noise. This signal is recorded onto standard graph paper or displayed on a monitor.

The ECG can have two modes;

1. Monitoring mode

Filters are used to limit the electrical frequency response to 0.5-40Hz, reducing environmental artefact. This helps to reduce distortions from muscle movement, mains current and interference from other equipment while providing a stable baseline by reducing respiratory and body movement artefact.

There are many different configurations for placement of the skin electrodes, however usually during anaesthesia, 3 electrodes are used (right arm, left arm and indifferent lead). With the ECG set to lead II, this provides the best detection of the P wave and improves detection of dysrhythmias.

2. Diagnostic mode

A wider electrical frequency response of 0.05-100Hz allows assessment of the P wave, QRS and T wave morphology, ST segment analysis and assessment of tachyarrhythmias.

A display speed of 25mm/s and sensitivity of 1mV/cm is standard.

Electrical interference (especially as a result of diathermy or mains frequency supply and muscle movement) can still be a problem. Differential amplifiers attempt to minimise this using a process called common mode rejection whereby, if the interference is common to two electrodes, it is not amplified. Interference from muscle movement can be minimised by placement of electrodes over bony prominences.[10]

Airway gas analysis

A number of different methods are used to analyse the different gases and vapours during general anaesthesia.

End-tidal carbon dioxide analysis (capnograph)

Carbon dioxide monitoring during anaesthesia is useful for a number of reasons:

1. It is used as an indicator of the level of ventilation (to detect hyper or hypoventilation).

2. To diagnose oesophageal intubation (very little carbon dioxide detected).

3. As a disconnection alarm if there is a sudden absence of end-tidal CO_2.

4. To diagnose lung embolism if there is a sudden drop of end-tidal CO_2.

5. To diagnose malignant hyperpyrexia due to a continually rising end-tidal CO_2.

Gases with molecules which contain two or more dissimilar atoms absorb infrared radiation at various different wavelengths. This property can be used to provide a continuous measure of inspired and expired carbon dioxide concentration. The amount of infrared radiation absorbed is proportional to the amount of CO_2 in the analysis chamber.[10]

Inhalational agent analysis

In order to estimate the amount of anaesthetic vapour in a patient's blood stream and hence the depth of anaesthesia, as well as the amount being delivered to the patient, it is important to monitor the inspired and end tidal concentrations of anaesthetic agents. This is done using infrared absorption in the same way that carbon dioxide is measured, applying the different absorption spectra of the different anaesthetic vapours.

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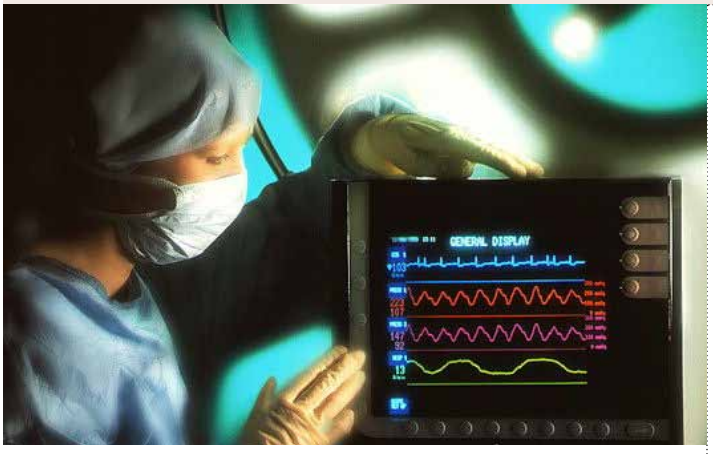

Oxygen analysis

Oxygenation is impaired in almost all patients during anaesthesia.[11] Supplemental oxygen is provided and this must be monitored to ensure the correct concentration is delivered to the patient. The most commonly used method of oxygen analysis is the paramagnetic analyser. Unlike other gases used in anaesthesia, oxygen is attracted towards magnetic fields. The paramagnetic analyser uses this property to cause differences in pressure which can be detected by a pressure sensor.[10] Other methods of oxygen analysis include the galvanic oxygen analyser (fuel cell) and polarographic oxygen analyser (Clark electrode) both of which rely on the diffusion of oxygen across a membrane and hence have a slow response time. They generate a current which is proportional to the partial pressure of oxygen.

Airway pressure and ventilation volume

Measurement of airway pressures and volumes is vitally important during anaesthesia in order to ensure the patient is receiving an adequate tidal volume and, especially during mechanical ventilation, to avoid trauma caused by overly high pressure or volume. Alarms also provide early warning of inadequate ventilation or risk of lung trauma.

Anaesthetic machines commonly use a device called a pneumotachograph to measure pressure and flow of gas to and from a patient. The basic principle involves passing the gas through a fixed resistance tube and using pressure sensors to detect the pressure change across it.

Nerve stimulators

When muscle relaxants are used during general anaesthesia, nerve stimulators can be used to assess the depth and type of neuromuscular blockade and whether the relaxant has been adequately reversed. It involves the delivery of an electrical stimulus to a peripheral nerve via skin electrodes and assessment of the level of muscle contraction as a result.

Temperature

The importance of monitoring a patient's temperature under general or regional anaesthesia must not be underestimated as inadvertent hypothermia can lead to delayed wound healing, increase in wound infections, higher blood loss and even cardiac arrest.[12] Current NICE guidelines state that temperature should be measured before induction of anaesthesia and that anaesthesia must not begin until a patient's temperature is above 36°C. Temperature should be measured at least every 30 minutes during anaesthesia, and in recovery, patients should not be discharged to the ward until their temperature is above 36°C.[13]

Modern non-invasive methods of temperature measurement such as the tympanic thermometer measure the infrared frequency emitted by the patient's tympanic membrane which then correlates with the patient's core temperature. Another method is the Tempadot, which consists of a plastic strip impregnated with heat-sensitive chemical dots. Usually attached to the patient's forehead, the chemicals melt, change colour and indicate the patient's temperature on a scale on the strip.[14]

Temperature can also be measured invasively using an oesophageal or bladder temperature probe. These devices usually incorporate a thermocouple; consisting two dissimilar metals which produce an electrical potential that changes with temperature.[15]

Invasive monitoring

Arterial line

An arterial line allows beat-by-beat monitoring of systolic, diastolic and mean arterial blood pressure, as well as other useful parameters such as stroke volume and cardiac output by using waveform analysis. Indications for invasive blood pressure monitoring include surgical factors such as; surgery where blood loss is expected to be high, high risk procedures, major/prolonged operations and where blood pressure must be tightly monitored (e.g. neurosurgery). Patient factors include critically ill patients, patients with fixed cardiac output states (e.g. severe aortic stenosis), obese patients where non-invasive blood pressure readings are inaccurate due to incorrect cuff size and when frequent arterial blood gas sampling is needed, for example patients with exacerbations of COPD.

The arterial cannula is attached to a pressure transducer via a column of heparinised saline that is pressurised to 300mmHg. The pressure transducer contains a wire that, when stretched, increases the wire's resistance. A beat-by-beat change in resistance changes the amount of current that passes through the wire, and this signal is processed and amplified to give a reading.

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Figure 1 shows the arterial trace produced by this signal. This trace should be displayed in order to detect damping which can be caused by obstruction or kinking of the system or by air bubbles or clots within the arterial line. Damping lowers the systolic and elevates the diastolic pressure reading with a loss of detail of the waveform. The transducer must be placed at the level of the patient's right atrium (mid-axillary line) in order for the reading to be accurate.[16]

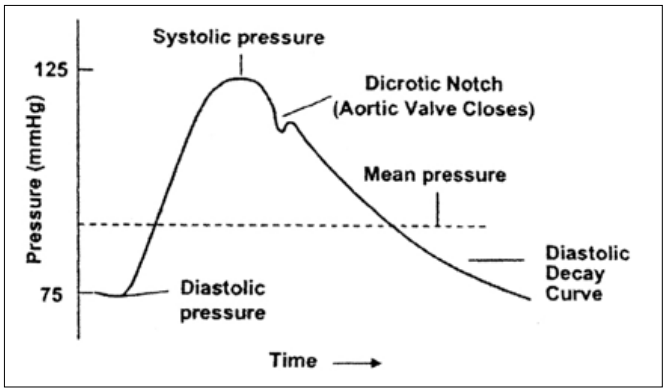

Figure 1. Taken from Syeda et al. (2003) (17).

Arterial thrombosis occurs in up to 35% of arterial lines.[18] This rarely leads to ischaemia or necrosis but should be monitored for. There is a risk of bleeding due to disconnection. Inadvertent drug injection through the arterial cannula can cause distal vascular occlusion and necrosis.

Central line

Central venous pressure (CVP) refers to the pressure within the right atrium and great veins of the thorax. It is measured via a central line in the internal jugular or subclavian vein, and produces readings using a transducer and amplifier similar to that described for arterial blood pressure measurements. CVP is affected by the level of the transducer and the position of the patient, and thus is usually measured with the patient lying flat with the transducer at the level of the right atrium, giving a normal CVP of 0-8cmH_2O. The CVP can be used as an indicator of fluid status and to help guide fluid therapy of critically ill patients or during major surgery.

However the CVP is affected by many factors and a high CVP does not necessarily mean that the patient is adequately fluid resuscitated, it can be increased in cardiac failure, vasoconstriction, tamponade, coughing or during intermittent positive pressure ventilation.[16] A more useful indicator of a patient's fluid status is the CVP trend with fluid challenges. For example, a fluid challenge of 3ml/kg (i.e. 210ml in a 70kg man) which causes a CVP rise of 2-5cmH_2O which is not sustained for more than 10-15 seconds suggests hypovolaemia. If however there is a continuous rise in CVP of 2-5cmH_2O post fluid challenges, it suggests fluid overload or heart failure.[19]

Cardiac output monitoring

The aim of cardiac output monitoring is to allow goal-directed fluid resuscitation in order to prevent inadequate tissue perfusion and tissue hypoxia, while avoiding fluid overload and pulmonary oedema.[20] Indications for use include a need to monitor left ventricular function and cardiac performance, evaluate the cardiac response to treatment and to detect cardiogenic or hypovolaemic shock and low cardiac output states. Again this mainly applies to critically ill patients, those with pre-operative poor cardiac function and those undergoing high risk/major surgeries.

The stroke volume is the volume of blood ejected from the left ventricle during one cardiac cycle, and cardiac output is the amount of blood ejected in one minute. See the formula below.

Cardiac output (CO) = Stroke Volume (SV) x Heart Rate (HR)

In most of the commonly used devices, a 10% increase in the stroke volume after a fluid challenge indicates that the patient is fluid responsive and therefore should receive further fluid challenges until the 10% increase is no longer reproducible. The patient is then said to be fluid optimised. Other parameters can also be used to guide fluid management such as flow time corrected (FTC) and stroke volume variation (SVV).[19]

There are a number of methods by which cardiac output can be monitored. Including the oesophageal doppler, pulse contour analysis, thermodilution, lithium dilution, PiCCO and LiDCO.

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The oesophageal doppler measures the velocity of blood flow through the descending aorta via a probe inserted into the distal third of the oesophagus. The cross-sectional area of the aorta is estimated (often using the patient's height, weight, age and sex). These values are used to calculate the patient's cardiac output. The main advantage of this technique is that it is minimally invasive and does not require vascular access. The oesophageal Doppler has several limitations: it can only be used in anaesthetised patients, the probe can be subject to rotation which makes the readings inaccurate, and the use of diathermy causes significant interference. It can cause trauma to the oesophagus and is relatively contraindicated in patients with varices.[21]

Pulse contour analysis is commonly used in the intensive care setting and measures the stroke volume using the arterial pulse pressure waveform[22] its use is extrapolated from patient demographic data and is dependent on the quality of the arterial line trace.[21]

The thermodilution technique involves a rapid bolus of cold saline into the central venous system which is then detected by a sensor in the arterial system. The speed at which the cold saline is detected in the arterial system is used to calculate the cardiac output. When measured using a pulmonary artery catheter this is the clinical 'gold standard' for cardiac output monitoring. However, it is a significantly invasive procedure and, due to its associated complications its use is controversial.[23]

Lithium dilution is a very similar method to thermodilution, whereby a small amount of lithium is injected into the central venous circulation and is detected in the arterial system.

Newer techniques include the PiCCO method which uses a combination of thermodilution and pulse contour analysis. The advantage is that this technique is less invasive, using central and arterial lines. However it may be less accurate due to heat dissipation of the cold saline bolus. The LiDCO technique is similar and uses a combination of lithium dilution and pulse contour analysis. It requires regular calibration and cannot be used in patients on lithium.[21]

Conclusion

Routine monitoring during anaesthesia is essential in maintaining patient safety. Further invasive monitoring may be indicated depending on the type of surgery and health of the patient. An understanding of how monitoring works is vitally important, especially as it provides us with knowledge about the limitations of the equipment and how to avoid potentially significant errors.

Questions

1. Measurement of oxygen in a mixture of gases can be achieved by which one of the following:

a. Severinghaus electrode

b. Clark electrode

c. Pneumotachograph

d. Fuel cell

e. Infrared analyser

2. Which of the following statements is true with regards to the pulse oximeter:

a. It under-reads in carbon monoxide poisoning.

b. It is unreliable in jaundiced patients.

c. It measures carboxyhaemoglobin.

d. It under-reads in the presence of methylene blue.

e. It regularly needs re-calibrating.

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3. Regarding automated blood pressure cuffs, which of the following is true?

a. Calculates the mean arterial pressure from the measured systolic and diastolic pressures.

b. Oscillations of the cuff are maximum at the systolic pressure.

c. Reliable at low blood pressures

d. A cuff which is too small will under-read the pressure

e. Requires a cuff that covers 2/3 of the upper arm

4. Which of the following monitors is not a minimum standard requirement for monitoring a patient while ventilated under a general anaesthetic?

a. Temperature

b. Pulse oximetry

c. End tidal carbon dioxide

d. ECG

e. Ventilation volume

5. Concerning end tidal carbon dioxide analysis in a mechanically ventilated patient which of the following is least true?

a. The absence of a carbon dioxide trace is an indication of oesophageal intubation.

b. The absence of a carbon dioxide trace is an indication of cardiac arrest.

c. The absence of a carbon dioxide trace is an indication of ventilator disconnection

d. A sudden drop in the end tidal carbon dioxide is an indication of pulmonary embolism

e. A continual rise in the end tidal carbon dioxide is an indication of malignant hyperpyrexia

Answers

1. d)
The Severinghaus electrode and Clark electrodes are used in arterial blood gas analysers to respectively measure carbon dioxide and oxygen partial pressure in solution. The pneumotachograph measures gas flow. The infrared analyser can only be used to measure gases with molecules that contain two dissimilar atoms as these will absorb infrared radiation (oxygen does not).

2. d)
The pulse oximeter over-reads in carbon monoxide poisoning. Jaundice does not affect the accuracy of the pulse oximeter. Carboxyhaemoglobin (from carbon monoxide poisoning) affects the accuracy of the oximeter but it is not itself measured. Methylene blue is an aniline dye and can induce methaemoglobinaemia and as such cause the oximeter to under-read

3. e)
The automatic cuff directly measures the mean arterial pressure. Oscillations begin at systole as the cuff is deflated and reach maximum at the mean arterial pressure. It is inaccurate at low blood pressures. A cuff which is too small will over-read the pressure. The cuff should cover at least 2/3 of the upper arm and the width of the bladder should be 40% of the mid-circumference of the limb.

4. a)
Temperature monitoring is only necessary if the duration of surgery is longer than 30 minutes. The rest are all minimum requirements.

5. b)
In the event of a cardiac arrest, ventilated patients will still demonstrate a small end-tidal carbon dioxide trace. The rest are all true.

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MANAGEMENT OF FOURNIER'S GANGRENE

J Smith

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Urology

Abstract

We present a clinical vignette of Fournier's gangrene in a 55 year old male. Initially, he presented with increasing scrotal pain and swelling. There was no history of trauma or associated lower urinary tract symptoms. Clinically, he was diagnosed with Fournier's gangrene of the skin of the scrotum and perineum. Following resuscitation, he underwent emergency surgical debridement and subsequently, elective reconstruction. The management of Fournier's gangrene is discussed.

Keywords: Fournier's, gangrene, orchidectomy.

Clinical Vignette

A 55 year old man presented to accident and emergency with a 3 day history of increasing scrotal pain and swelling. He complained of feeling unwell and fever. He had a past medical history of type 2 diabetes mellitus and peripheral vascular disease.

On examination, he was pyrexial at 38.1°C, tachycardic, tachypnoeic and a BP of 90/60mmHg. Scrotal exam revealed erythematous swellings extending onto the perineum. There was a small black area discharging pus. On palpation, crepitus was present. DRE was normal.

Laboratory tests were as follows; white cell count 18 x 109/L; Sodium 134 mmol/L; potassium 3.5 mmol/L; creatinine 170 µmol/L; venous bicarbonate 115 mg/dL; HbA1c 8.1%. Urgent ultrasonography revealed scrotal wall thickening and subcutaneous pockets of gas.

A clinical diagnosis of Fournier's gangrene was made. He was resuscitated with IV fluids and broad spectrum antibiotics. Subsequently, he underwent extensive surgical debridement of the necrotic tissue. The resection was to healthy, bleeding skin. Post-operatively, he was managed on ITU and remained stable. A further inspection of the area at 24 hours revealed no further necrotic tissue so the wound was redressed. Microscopy, culture & sensitivity yielded multiple gram positive and negative bacteria, including Eschericia coli. He remained on broad spectrum antibiotics for 14 days.

Following the appearance of healthy granulation tissue at the base of the wound he underwent a pudendal fasciocutaneous flap which achieved good coverage of the defect. He was later discharged and followed up by the plastic and urological surgeons.

Discussion

Fournier's gangrene is necrotizing fasciitis of the scrotal skin. Although relatively uncommon it is a urological emergency which requires prompt diagnosis and management. Despite aggressive management mortality rates are between 7.5% and 10%.[1,2] It is more common in men than women and has a prevalence of around 1.6 cases per 100,000 males per year, rising to 3.3 cases per100,000 males per year over the age of 50.1 Risk factors include diabetes mellitus, alcoholism, scrotal surgery, ano-rectal tumours and peri-anal infections. Diabetes Mellitus and chronic alcoholism are present in approximately 40% and 25-50% of cases, respectively.[3,4] Many different organisms have been implicated in the development of Fourniers gangrene of which the majority are perineal or genital commensals, such as Eschericia coli, Enterococcus, Klebsiella spp., Pseudomonas, Streptococcus, Staphylococcus and Clostridium.[2] The majority of cases are polymicrobial and demonstrate a mixture of aerobic and anaerobic bacteria. It is the combination of proteins and enzymes (e.g. heparinises, coagulase) released by this microbial mixture which stimulates platelet aggregation and complement fixation.5 These changes lead to the development of micro-circulatory thrombosis with subsequent ischaemia and necrosis. Many of the organisms also produce endotoxins resulting in severe endotoxosis, sepsis, organ dysfunction and death.[4]

Clinical Presentation

Fourniers gangrene can present in a variety of different ways and therefore, a high index of suspicion is required when attempting to diagnose this, potentially, fatal condition. Although it commonly follows a rapidly progressive pattern of onset, the time to presentation can vary between 1 to 10 days following onset of symptoms.[6] The most common symptoms are pain and swelling over the genital region with associated fever. Erythema and purulent discharge may also be observed in the overlying skin, although the pain is commonly out of proportion to the cutaneous signs.[4,6] Crepitus in the underlying tissue is reflective of the ongoing gangrenous process and attributable to presence of gas forming micro-organisms.

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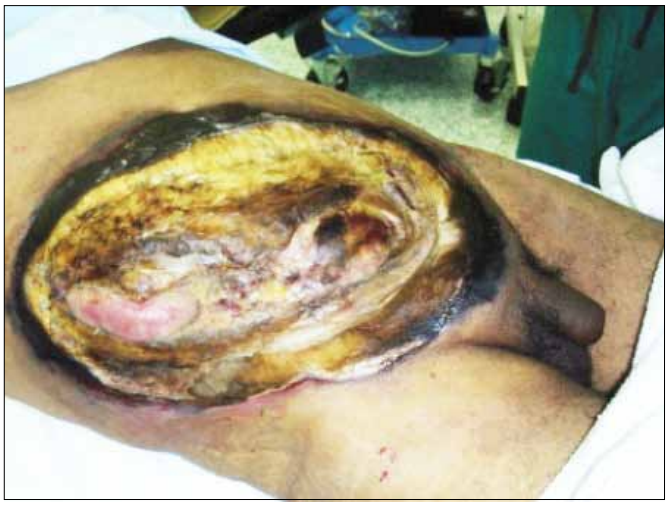


Figure 1: Fournier's gangrene following Renal transplant (Transplanted kidney can be seen in right flank).[4]

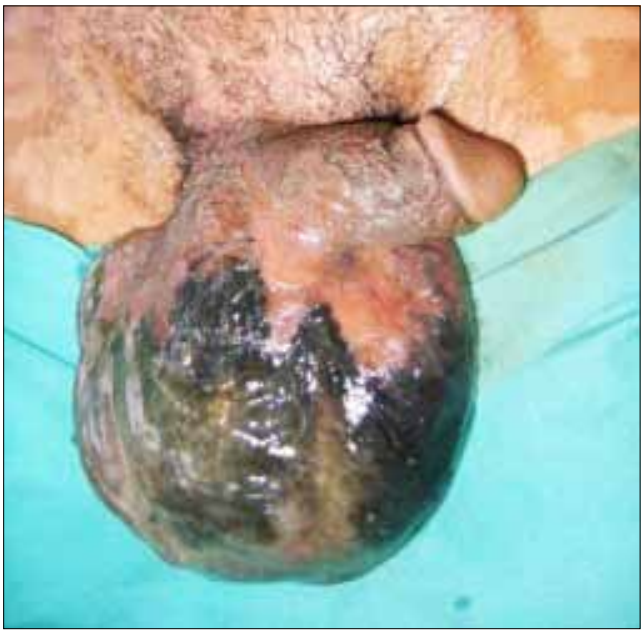
Spread

When considering the spread of Fournier's gangrene it is important to understand the anatomy of the superficial and deep fascial layers of the penis, scrotum, perineum and abdomen. The superficial fascia of the abdomen and urogenital triangle can be divided into membranous and fatty layers. The membranous layer of the superficial fascia in the urogenital triangle is called Colle's fascia. It is attached posteriorly to the perineal body and superficial transverse perineal muscle and is attached laterally to the pubic arch. Supero-anteriorly it is continuous with the membranous layer of the abdominal wall, Scarpa's fascia. The postero-lateral attachments of Colle's fascia limit extension of urogenital sources of infection and, instead, the gangrenous process ascends supero-anteriorly up the abdominal wall. Anorectal disease, however, involves the perianal region before tracking between the external rectal sphincters to involve the urogenital triangle. This difference in presentation may allow for early recognition of the source of infection.[7]

Colle's fascia is continuous with dartos fascia of the scrotum and the superficial fascia of the penis. Infection can track along these fascial planes resulting in penile and scrotal involvement. The erectile tissues of the penis and the testicles, however, are usually spared. The presence of Buck's fascia, a deep layer of fascia continuous with the tunica albuginea of the cavernosa, provides a barrier to infection with the external and internal spermatic fascia performing the same role for the testicles.[4,5,7] The testicles also take their blood supply from the abdominal aorta via the gonadal arteries and therefore haematogenous spread of infection does not occur.

Investigation

Any patient presenting with suspected Fournier's gangrene requires prompt investigation and management. Although initial investigations should aim to assess severity and achieve definitive diagnosis they should not delay the early administration of fluids, broad spectrum antibiotics and surgical debridement. Initial investigations should focus on assessing the patient's haemodynamic status and biochemical parameters, both of which have prognostic importance. The Fournier Gangrene Severity Index (FGSI) score may be useful in predicting outcome and is more accurate that any individual parameter alone. The FGSI was first developed in 1995 and is a modification of the APACHE II scoring system.[8] The APACHE II classification is a well validated tool for scoring the severity of several septic processes and assesses 12 routine physiologic measurements.[9] FGSI, however, measures 9 physiologic parameters; Temperature, heart rate, respiratory rate, serum sodium, serum potassium, serum creatinine, haematocrit, white cell count and serum venous bicarbonate.[8] In the initial study by Laor E, et al8 a score of 9 was used as a cut-off. They found that patients with a score >9 had a 75% probability of death and those with a score <9 had a probability of survival of 78%. Although many studies have looked into the use of the FGSI there has yet to be a consensus on its usefulness in clinical practice at predicting outcome. Results do show, however, that increasing FGSI scores do correlate with a poorer outcome and it is therefore a simple, objective way of quantifying risk in patients with Fourniers gangrene.[10,11] Interestingly, surface area of the skin involved in the necrotic process does not seem to correlate with prognosis, although some controversy remains.



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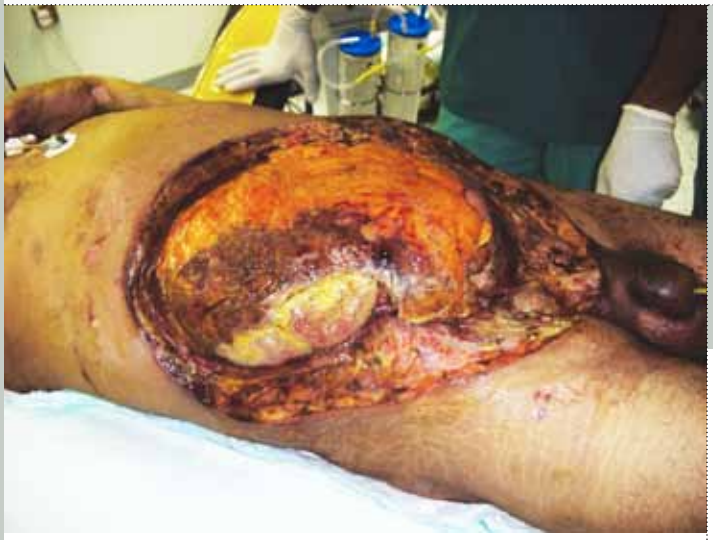

Although, the diagnosis of Fournier's gangrene is based upon clinical suspicion some cases in which diagnostic confusion exists or the disease extent is uncertain warrant further investigation. Plain radiography, ultrasonography and computed tomography can all help confirm the diagnosis. Radiography may demonstrate scrotal soft tissue swelling and subcutaneous gas, even before clinically detectable crepitus. Ultrasonography is a quick investigation which can be useful in differentiating between Fournier's gangrene and incarcerated inguinal hernia by demonstrating scrotal wall thickening and subcutaneous air pockets as opposed to intra-luminal bowel gas, seen in the latter condition. Computed tomography has the highest specificity out of the three imaging modalities for diagnosis and defining disease extent. It also allows visualisation of any retro-peritoneal extent and possible identification of the disease focus.[7] Magnetic resonance imaging can also be used as an alternative to computed tomography.

Treatment

The effective treatment of Fournier's gangrene relies on early recognition, fluid resuscitation, broad spectrum antibiotic therapy and extensive surgical debridement. Although it is important to stabilise the patient and administer antibiotics, delaying surgical debridement has a negative effect on prognosis.[6,11] Initial management, as with any acutely unwell patient, begins with a thorough assessment of the patient's haemodynamic status and subsequent stabilisation. Aggressive fluid administration, with either colloid or crystalloid, is essential and in patients with septic shock inotropic support may also be required. Appropriate intavenous broad spectrum antibiotics with polymicrobial cover for aerobic and anaerobic organisms should be administered as early as possible. Although local policy should be followed a common approach is a triple therapy regimen including a third generation cephalosporin, an aminoglycoside and metronidazole.[12]

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Following stabilisation of the patient rapid debridement is the key to reducing morbidity and mortality. Debridement should take place in theatre with the patient in the lithotomy position.[5] Debridement should be aggressive but not excessive and should include all overtly necrotic tissue whilst maintaining the continuity of the deep fascia, as this is rarely involved. Visual appearance is a poor method for estimating disease extent and many authors recommend extending the resection margins to 5-10mm beyond where the subcutaneous tissue is no longer abnormally easy to dissect from the deep fascia.[13]

During debridement cultures should be obtained from the tissue and frozen-sections may be required to confirm diagnosis. The use of diversion colostomy should be reserved for disease involving the anal sphincter, anorectal region, colonic and rectal perforation or in patients at high risk of faecal contamination. Cystostomy for urinary diversion is a controversial topic and should be reserved for cases in which there is massive urinary extravasation or peri-urethral inflammation where a urethral catheter will not suffice.[5,14] Orchidectomy is not routinely required as the testicles have an independent and abundant blood supply from the gonadal vessels. If, however, the testicles are involved in the disease process from either a scrotal abscess, epididymo-orchitis or retro-peritoneal spread their removal is warranted.[3,5] If the testes have been exposed during the initial debridement they may be placed in a medial thigh pouch or lower abdominal pouch until further procedures or healing takes place.[5]

Post-operatively careful wound management is required. Initially wet dressings should be applied containing either saline, anti-septics (e.g. Dakins solution) or enzymatic agents with two to four dressing changes daily.[15] The wound should be carefully monitored for further signs of necrosis which, if present, should prompt further surgical debridement.[6] Some studies have shown that patients require on average 3 debridements before all necrotic tissue has been removed.[14] The use of vacuum-assisted closure (VAC) therapy is pioneered in some units as an alternative to conventional dressings. Although it can achieve cleaner wounds with less exudate it has failed to demonstrate superiority in retrospective trials.[15] Another adjunct to aid wound healing and microbial clearance is the use of hyperbaric oxygen therapy.

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In Fournier's gangrene the ongoing necrotic process combined with reduced blood flow, secondary to systemic hypotension, leads to localised ischaemia impairing leucocyte function. Hyperbaric oxygen therapy increases the partial pressure of oxygen in the blood, independent of haemoglobin concentration, and therefore improves leucocyte function and wound healing. Some studies have shown a significant survival advantage in patients receiving two treatments a day for one week.[16]

Many patients are left with large perineal and scrotal defects following radical debridement and therefore require reconstruction procedures to correct them. Any reconstructive procedure should not take place until healthy granulation tissue is observed at the base of the wound and only after consultation with a plastic surgeon.[17] There are many different reconstructive procedures and the technique employed depends upon the defect location, size and depth. Commonly employed procedures include split-thickness skin graft, full-thickness skin graft, local advancement flap, fasciocutaneous flap, muscle flap and myocutaneous flap whilst small defects may heal with primary or secondary closure.[6,17]

Split thickness skin grafts (STSG) involve auto-grafting skin onto the defect site. Their use has been described for scrotal and large abdominal defect repair. The benefits of using STSG for scrotal repair include better cosmetic result, short hospital stay, minimal morbidity and a simpler operative technique.[18] If the tunica vaginalis is exposed, however, the STSG may not take satisfactorily and the resulting scar is often unsightly due to contracture of the graft.[6] It is therefore recommended that STSG is reserved for large defects of the lower abdominal wall with little dead space beneath the graft.[17]

Scrotal advancement flaps can be utilised if the extent of the defect involves less than 50% of the scrotal surface area and there is no extension onto the perineum or abdomen. Because the skin is easily stretchable closure is simple resulting in good cosmetic results providing the flap is not under too much tension.[6,17]

If the patient has a scrotal defect over 50% of the scrotal surface area with extension onto the perineum then a pudendal thigh fasciocutaneous flap or a pedicle anterolateral thigh flap can be used.[17] Pudendal thigh flaps involve mobilising a fasciocutaneous flap supplied by the superficial perineal artery and nerve and tunnelling it medially through the subcutaneous layer to repair the defect.[6] The anterolateral thigh flap is similar in principal to the pudendal thigh flap except that the pedicle is based around the musculocutaneous and septocutaneous perforators of the descending branch of the lateral circumflex femoral artery and associated venae comitantes.[19]

Using pedicled fasciocutaneous flaps has a number of advantages over STSG and myocutaneous flaps. It can not only cover larger and deeper defects than a STSG it provides a good cosmetic outcome with minimal donor site morbidity and may also be sensate. It is also less bulky than a myocutaneous flap with no sacrifice of functional muscle.[6,17]

Gracilis myocutaneous flaps are useful for repairing deep soft tissue defects in the perineum. They tend to be well vascularised and therefore resistant to bacteria and wound contamination.[6] The gracilis muscle is easily elevated from the donor site which itself is easily repaired by primary closure.[17] They may also be combined with STSG if further skin coverage is required. An algorithm has been developed for the reconstructive management of defects.

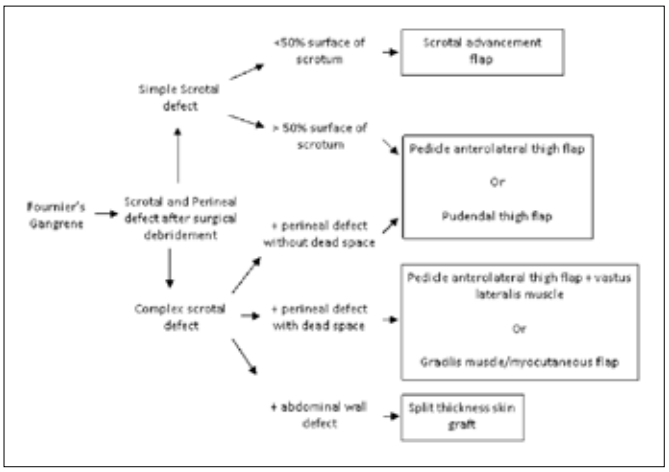
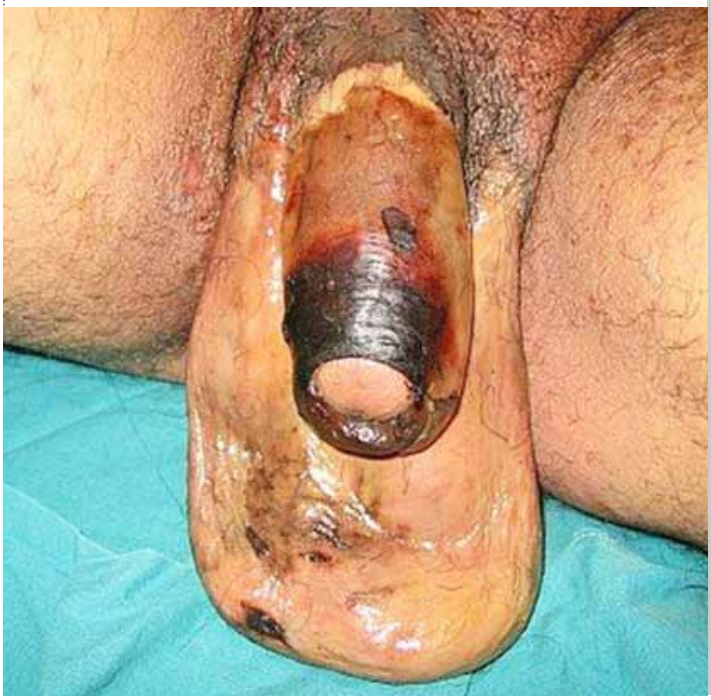


Figure 2: Algorithm of surgical management of defects left following debridement of Fournier's gangrene.[17]



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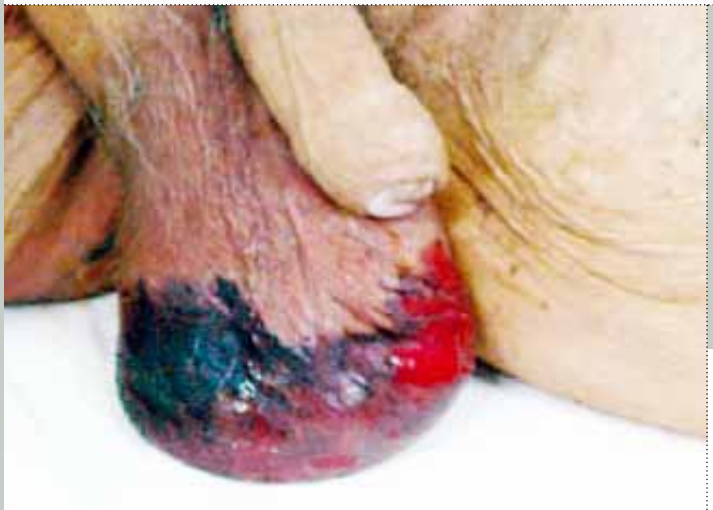

Conclusion

Fournier's gangrene is a rare, rapidly progressive necrotising fasciitis which carries high rates of mortality and morbidity. Risk factors include Diabetes Mellitus, alcoholism and immunosuppression. Infection commonly originates from perianal or urogenital infections or following local trauma, either accidental or iatrogenic. Diagnosis requires a high level of clinical suspicion as the cutaneous signs may be minimal. Signs of sepsis or localised pain in excess of the cutaneous signs should prompt further investigation or treatment. Although the diagnosis is usually clinical, X-ray, ultrasonography, computed tomography and MRI may all help identify Fournier's gangrene. The mainstay of treatment is rapid and aggressive debridement of affected area plus fluid resuscitation and broad spectrum antibiotics. Debridement may leave large defects requiring reconstructive procedures.

Questions

1. Which of the following is not an organism commonly implicated in Fournier's gangrene?

a) Eschericia coli
b) Klebsiella spp.
c) Streptococci
d) Helicobacter pylori
e) Bacteroides spp.

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2. Which of the following is not a common symptom of Fournier's gangrene?

a) Swelling
b) Pain
c) Malaise
d) Fever
e) Priapism

3. Which of the following structures does not limit the spread of Fournier's gangrene?

a) Pubic arch
b) Urogenital diaphragm
c) External rectal sphincters
d) Bucks fascia
e) Superficial Transverse perineal muscle

4. When performing radical debridement on necrotic tissue where should your margins of debridement be?

a) 5-10mm beyond the border of visually apparent necrosis
b) 5-10mm beyond the point where the subcutaneous and deep fascia do not separate abnormally easily
c) 15-20mm beyond the border of visually apparent necrosis
d) 20mm beyond the point where the subcutaneous and deep fascia do not separate abnormally easily
e) Debridement should always include the whole of the scrotal and perineal skin and subcutaneous fascia

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5. For each of the following defects select the most appropriate reconstruction procedure from the following list:

A. Split thickness skin graft **B. Full thickness skin graft**

C. Gracilismyocutaneous flap **D. Pudendalfasciocutaneous flap**

E. Scrotal advancement flap **F. Anterolateral thigh flap + vastuslateralis muscle**

a) <50% scrotum involved without deep defect
b) Deep defect involving >50% of the scrotum and extending onto the perineum in a patient with peripheral vascular disease and a persistently contaminated wound
c) Large defect extending involving the lower abdomen
d) Deep defect involving >50% of the scrotum

Answers

1. d)

2. e)

3. c)

4. b)

5. a) E **b) C** **c) A** **d) D**

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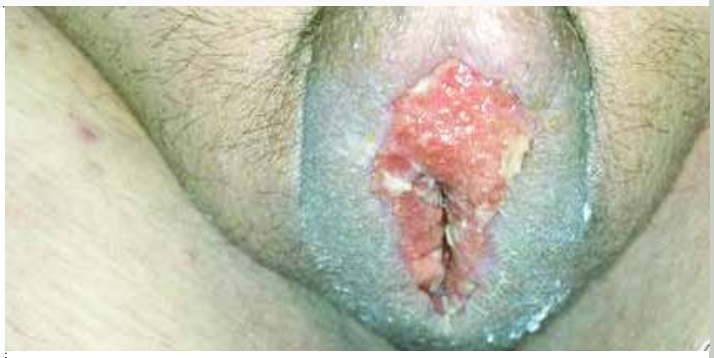

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OTITIS MEDIA WITH EFFUSION

M Rollin

Abstract

Otitis media with effusion (OME, "glue ear") is chronic inflammation of the middle-ear mucosa with associated accumulation of uninfected mucus and/or serous fluid in the middle-ear space. Aetiology is debated, but is probably secondary to viral infection, with Eustachian tube dysfunction the major mediating factor. Classical presentation involves childhood hearing loss, but symptoms can also involve disruptiveness, clumsiness or poor balance. Many cases resolve spontaneously; persistent cases require myringotomy, grommet placement, and possibly adenoidectomy. Treated prognosis is excellent; untreated OME can blunt language skills, potentially leading to educational delay and poor subsequent attainment, as well as increased risk of otopathology such as attic retraction.

Keywords: Otitis media, glue ear, Eustachian tube.

Case Study

By the age of five, Steven already had a bad reputation among his teachers. Frequently disruptive in classes, he ignored his teacher and was often found messing around rather than working. His parents described him as energetic and "quite a handful". His teacher called him a naughty disruptive child. His language development and educational milestones were found to be behind his peer-group.

Anatomy and Pathology

The middle-ear cleft is an air-filled space bounded laterally by the tympanic membrane and medially by the hard bone of the otic capsule, surrounding the inner-ear structures. Posteriorly the aditus ad antrum is the opening into the mastoid air-cell system, and the Eustachian tube orifice lies anteriorly. The ossicles, associated muscles and chorda tympani cross the space; the facial nerve runs in its own thin-walled bony canal along the posterior wall.

The Eustachian tube is lined by the same respiratory-type ciliated mucus-producing epithelium as the nasal cavity. This gives way within the middle-ear space to the flat cuboidal epithelium lining the mastoid system.

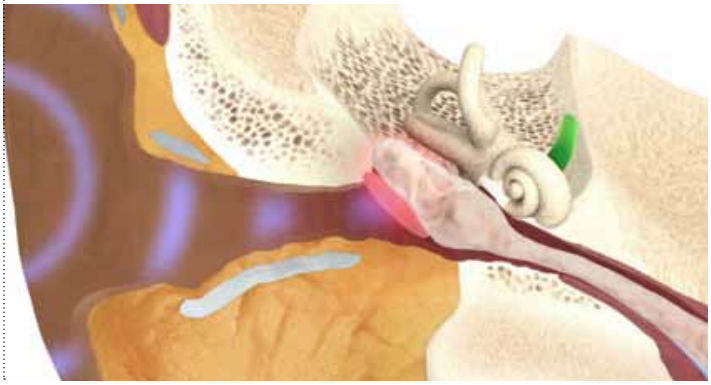
In the standard theory, initial mucosal damage is viral. Swollen Eustachian-tube mucosa closes the tube, leading to poor aeration of middle-ear mucosa and perpetuating mucosal injury. Chronic mucosal inflammation leads to disorganised de-differentiation into thick pseudostratified epithelium, and a combination of inefficient mucosa and submucosal inflammation leads to retention of secretions in the middle-ear space.[1,2,3,4] Bacteria may secondarily-infect these stagnant secretions, leading to acute suppurative otitis media, but most often the secretions are found to be sterile.[5]

Pressure in the middle-ear space naturally falls over time as oxygen is absorbed by the mucosa. The Eustachian tube normally re-supplies air to the middle-ear space, such that the pressure is equal on both sides of the tympanic membrane. In OME, low pressure and the presence of fluid in the middle-ear space result in highly inefficient tympanic membrane vibration, causing conductive hearing loss.

Other theories of pathogenesis, including allergy and irritation from cigarette smoke, have not been shown convincingly to be important. Acid reflux has been demonstrated in children, and pepsin has been recovered from middle-ear effusions,6 but this is currently debated.

Epidemiology

Age-distribution is bimodal, with peak prevalence of 20% at two years of age, and a secondary peak of 16% at approximately five years of age.[7] It may be worth noting that the first of these approximates to the age when most children first attend nursery, and the second approximates to the age when most children start primary school. In temperate climes, there are roughly twice as many diagnoses in winter as in summer.[8,9,10,11] Independent risk factors include history of acute suppurative otitis media, family history of OME, number of older siblings and nursery or day-care attendance.[12,13,14]



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Clinical Features

There is no standardised diagnostic test for OME, however diagnosis in secondary-care typically rests on the combination of otoscopy, pneumatic otoscopy, tympanometry and pure-tone audiometry. Not all these techniques are available in primary-care, therefore diagnosis rests more on history and otoscopy. (Figures 1 and 2).

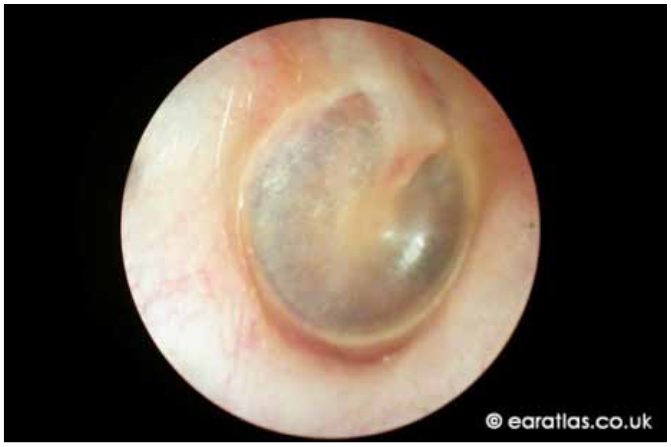


Figure 1. A normal tympanic membrane (right ear). Copyright earatlas.co.uk, used with permission.

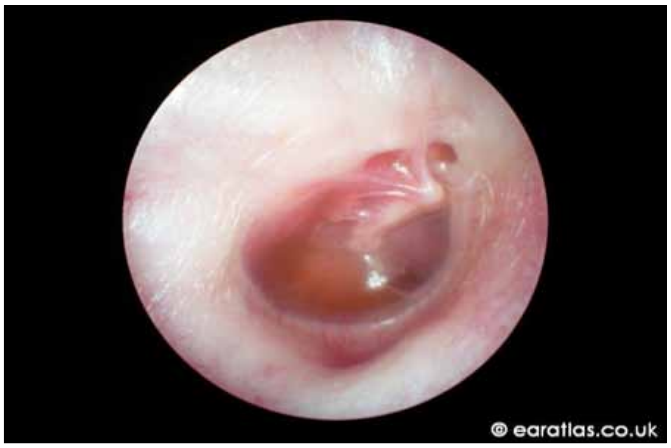


Figure 2. A tympanic membrane showing features of OME (right ear). Copyright earatlas.co.uk, used with permission.

Parental report of a child's hearing ability has been shown to be a poor discriminator,[15,16] however parental report of poor hearing in association with snoring, mouth-breathing and frequent URTI does show a positive association with OME.

Levels of hearing loss vary between children and episodes, however best current evidence shows mean thresholds of 21dB (standard deviation 10) in the better-hearing ear, and 31dB (sd 13) in the poorer-hearing ear.[17] These figures come from pure-tone audiometry, which by definition is performed in ideal conditions, not real-world situations – and therefore there may be some merit in using questionnaires such as the MRC's Reported Hearing Disability questionnaire, completed by adults in close contact with a child.[18]

Natural History

The majority of episodes of uncomplicated OME resolve spontaneously. There is a highly-skewed distribution of duration, with the median at three months, but the 95th centile at twelve months. Large studies have shown that, while half the affected ears resolve after three months, half of these will suffer a further episode.[19,20] This pattern of resolution is broadly consistent across the childhood age-range, although episodes of OME diagnosed in autumn and winter tend to last longer than those diagnosed in spring and summer. Other risk factors for persistence of episodes include presence of concurrent URTI, significant hearing loss and maternal smoking.[21,22,23]

Evidence exists that there are long-term sequelae from persistent or recurrent OME: in one cohort followed-up until eighteen years of age, sufferers had a mean air-conduction deficit of 4dB, and a mean bone-conduction deficit of 2dB, compared to unaffected peers.[24]

It is important to appreciate that the majority of episodes of OME will resolve spontaneously – and that the key challenge is to identify and intervene on those which do not resolve and are causing harm to a child's language and intellectual development.

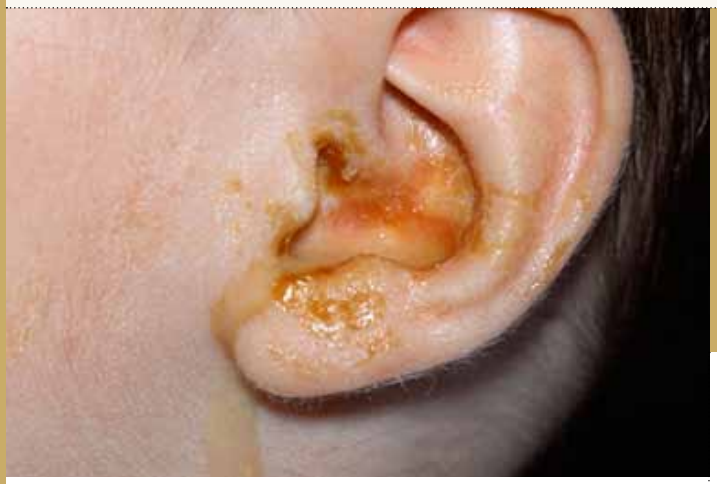
Language-development is a complex and incompletely-understood phenomenon, and assessments generally use progress against standardised milestones. Prospective studies and meta-analyses have suggested that there is a deleterious effect from OME on language development, that there is a correlation between overall number of childhood days with OME and severity of effect, and that children affected in early childhood have largely caught up with their peers by the age of eight.[25,26,27] Studies of intellectual development in early childhood have reached broadly similar conclusions.[28,29,30]

However, there may be subtler effects which are more persistent. Evidence from a long-term cohort study suggests that the OME-related deficit in IQ testing scores remains significant at thirteen years of age, and that the OME-related deficit in reading ability remains significant at eighteen years of age, with cases of persistent bilateral OME in childhood showing a two-year delay in mean reading scores at that age compared to unaffected peers.[31,32] More concerning still, a further study on this cohort suggests that diagnosis and treatment at five years of age comes too late to prevent these effects.[33]

Questionnaire-based studies have shown significant effects of persistent OME on childhood behaviour,[18,34] balance and clumsiness.[35,36]

OTITIS MEDIA WITH EFFUSION

M Rollin



Otitis media with effusion
Otorhinolaryngology & Neck Surgery

Management

Current national guidelines recommend a three-month watchful wait after formal diagnosis, to encourage spontaneous resolution. GPs who perform this wait themselves before referring are outside guidelines, as all children must have the possibility of sensorineural hearing loss excluded as early as possible. During the waiting period, or if the hearing loss is not that severe, hearing tactics (such as reducing background noise, facing the child and getting their attention before talking) can be useful.

No medical therapy has been adequately shown to be useful – including local steroid, systemic steroid, nasal mucolytics, antibiotics and decongestants. Autoinflation of the middle-ear can be beneficial in older children, but relies heavily on compliance and is not really practical in younger children.

Surgery typically involves myringotomy and placement of grommets (ventilation tubes, VTs, Artificial Eustachian Tubes), with concurrent adenoidectomy where indicated. Grommets designed for long-term placement are associated with higher complication rates, so short-term designs are recommended for use in children.[37] There are many studies examining the natural history of grommets, and typical findings at six-month follow-up are that around 55% are functioning, and 30% to 55% have extruded,[37,38,39]

Recent Literature

The key study to know is the MRC-funded TARGET study (Trial of Alternative Regimens in Glue Ear Treatment). This UK-based multi-centre trial examined 3,831 children between three and seven years of age, setting a diagnostic standard of 20dB conductive hearing loss bilaterally, in the presence of bilateral effusions. 34% of GP referrals met these criteria and were admitted to the study, and all were given an initial twelve-week watchful wait. Symptoms persisted in 51% of children, and these were then randomised to various treatment pathways.

Of those assigned to further waiting ("non-specific medical management"), half had effusions which persisted beyond another twelve weeks. This equates to 25% of those diagnosed with OME (8.5% of all GP referrals).[21,22,23]

Grommets (VTs) conferred a mean hearing threshold advantage of 12 dB over non-surgically-managed cases. Thresholds in the non-surgical group improved over time, but approximately half of the parents in this group opted for surgical management after randomisation. Differences between the two groups were negligible after twelve months' follow-up.[40]

TARGET also compared VTs against VTs plus adjuvant adenoidectomy. The additional procedure conferred no hearing benefit after three or six months of follow-up. However after both twelve and 24 months, when the benefit of VTs alone had diminished compared to non-surgical management, adjuvant adenoidectomy provided an additional 4.2dB of hearing benefit, and significantly reduced the requirement for revision surgery.[40,41]

This large study also included the MRC's Reported Hearing Disability questionnaire. On this measure, the benefit of VTs over non-surgical management was "large, even when adjusted for the expectation-effect of surgery". Over two years of follow-up, the improvement in hearing disability was "modest" (an expected finding, as the benefit of VT placement is naturally skewed towards the early post-operative period), however parental report of children's hearing ability showed continued benefit of VTs well into the second year of observation, even when there was no further difference as measured by pure-tone thresholds.[40]

Core Trainee's How-to Guide

Consultation

History: ask about hearing loss, mishearing words, whether the child comes when called, whether they are turning up the TV volume, how their language-development is going, whether there are any behavioural issues currently, and whether the teacher has commented on the child's hearing.

Other conditions: ask about snoring and OSA, URTIs, mouth-breathing at rest, asthma, eczema and any balance problems or clumsiness.

Examination: perform otoscopy and anterior rhinoscopy. An older child may allow flexible endoscopy of the post-nasal space, but in a younger child the misting test will suffice.

Investigations: pure-tone audiogram will show bilateral conductive hearing loss. Tympanometry will show flat traces (type B) or possibly a peak below –200daPa (type C2).

OTITIS MEDIA WITH EFFUSION

M Rollin

Managment

All children should undergo an initial three-month wait. This is more likely to succeed in spring/summer than in autumn/winter. Persistent symptoms at three-month review should provoke listing for grommet placement. Concurrent adenoidectomy is currently debated. Concurrent tonsillectomy can be offered if there is a history of OSA or recurrent tonsillitis – both subject to individual department or consultant practice.

Pre-op Assessment

Explore history of bleeding disorders, sickle-cell, recent URTI, asthma and allergies. Ensure audiogram/tympanometry available in notes.

Theatre

Place the child supine, with the head laterally flexed away from you and turned 45 degrees away. Spend as much time as necessary to obtain a clear view with the microscope. Clear wax from the canal with a Jobson-Horne or Formby probe, unless confident in the use of microsuction. Adjust the microscope to obtain a clear view of the tympanic membrane (TM). Position the myringotome so that the tip is clearly visible, and aim to make a radial myringotomy in the anterior-inferior quadrant of the TM (Figure 3). If access to this quadrant is difficult owing to a prominent TMJ bulging into the canal, make the incision directly inferior to the umbo. Ask for the fine tip on the microsuction and aspirate glue through the incision. The anterior-inferior quadrant is recommended as there are no important middle-ear structures in the area to damage; nevertheless try not to push the sucker through the membrane too far, as bleeding may result, leading to clot and scar tissue formation.

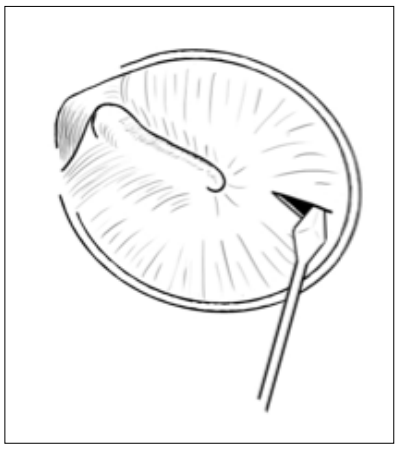


Figure 3. Technique of myringotomy creation (right ear).

Carefully mount the grommet on crocodile forceps and slowly insert through the incision under direct vision (Figure 4). If it is a Shah grommet, insert the tab first (bending the tab down slightly may help), remove the forceps and manipulate the grommet with a needle, which will allow better vision. Use ear-drops to dilute any blood (or it will block the grommet lumen) and place a cotton-wool ball in the canal.

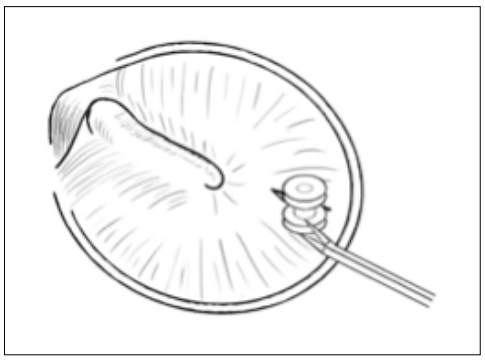


Figure 4. Technique of grommet placement through a myringotomy.

Talking to the Boss

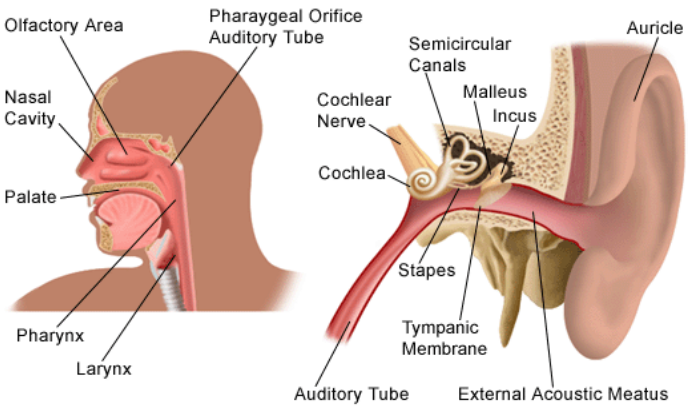
Mention the TARGET trial, and ask about their personal and departmental policy regarding adjuvant adenoidectomy with grommet placement. Be aware that there are increasingly strict thresholds for these procedures, and that extra paperwork may be required when listing. If in doubt, ask for a copy of the local PCT guidelines.

Case Study Conclusion

The family GP was alerted when Steven failed school hearing screening. In the local ENT department, he was found to have a 30-40dB conductive hearing loss bilaterally, and features of OME on examination. Symptoms were not improved after 3 months, although his teacher did take a more sympathetic view during this time. He underwent myringotomy and grommet placement plus adenoidectomy, and on recovery from anaesthesia complained that everyone was shouting. At six-week follow-up his audiogram was normal, his parents described him as "a completely different child", and reported that his teacher had stopped them at the school gate to tell of the transformation in his behaviour and school-work. They were offered six-monthly follow-up.

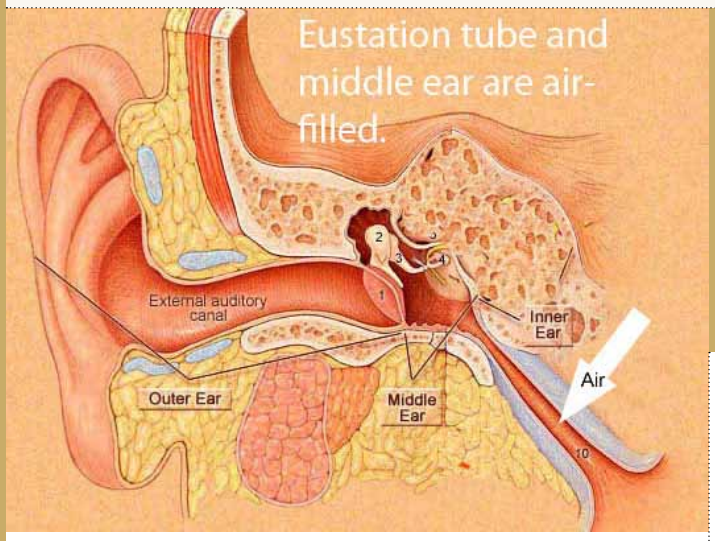
Acknowledgements

Thanks to David Pothier for the use of otoendoscopic photographs from earatlas.co.uk.



OTITIS MEDIA WITH EFFUSION

M Rollin



Questions

True or false?

1. The middle-ear cavity is lined with pseudostratified columnar ciliated epithelium.
2. OME is likely to occur in children with cleft palate.
3. Allergy is an independent risk factor for development of OME.
4. Parental report of a child's hearing ability correlates well with audiometry results.
5. The number of days spent with OME in childhood correlates to severity of effect on language-development.

Select the single best answer for the following questions.

1. A child is suspected of suffering from OME. Which of the following findings DOES NOT support that diagnosis?

a) type C1 tympanogram
b) retracted tympanic membrane on otoscopy
c) 25dB conductive hearing loss bilaterally
d) immobile tympanic membrane on pneumatic otoscopy
e) no discernible nasal airflow on misting test

2. In theatre, a surgical core trainee is unable to visualise the whole tympanic membrane owing to residual wax, and places a grommet in the visible segment. On recovery, the child complains of loud noises and a strange taste. Which structure may have been damaged?

a) facial nerve
b) stapedius tendon
c) chorda tympani
d) tensor tympani
e) nerve to stapedius

3. You see a child in a busy clinic, newly-referred with suspected OME, and send them for audiometry. The audiogram is returned showing raised air-conduction thresholds, and has a note attached explaining that the audiologist is too busy to obtain bone-conduction thresholds. What is the appropriate management?

a) three-month watchful wait
b) list for grommet placement and adenoidectomy
c) refer for otoacoustic emissions
d) insist that the audiologist obtains bone-conduction thresholds
e) bring the child and parents back to clinic next week

4. Which of the following is NOT a risk factor for development of OME?

a) maternal smoking
b) history of recurrent acute otitis media
c) family history
d) day-care attendance
e) number of siblings

5. Which of the following structures DOES NOT cross the middle-ear cavity?

a) Tensor tympani
b) descending portion of the facial nerve
c) Incus
d) Chorda tympani
e) Stapedius tendon

Answers to true/false

1. False: only the most anterior part of cavity contains this epithelium. The majority is lined by flat cuboidal type.
2. True: such children have congenitally-poor Eustachian tube function.
3. False: studies have not shown this to be the case.
4. False: parental report is a poor predictor of true hearing thresholds.
5. True: such effects are also detectable years later.

OTITIS MEDIA WITH EFFUSION

M Rollin

Single best answers

1. a) Type C1 tympanogram implies mild Eustachian tube dysfunction, not OME.

2. c) The chorda tympani may be damaged by a posteriorly-placed grommet. Complaint of loud noise is almost universal after this procedure.

3. d) Bone-conduction thresholds must be obtained at the earliest opportunity, to identify the child with sensorineural hearing loss, who requires a hearing aid.

4. a) Maternal smoking is a risk factor only for persistence of existing OME.

5. b) The descending portion of VII lies entirely within the mastoid.

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NECROTISING ENTEROCOLITIS

N J Hall

Abstract

NEC is a condition characterised by intestinal necrosis which affects predominantly preterm, low birth weight infants. The diagnosis is based on specific clinical and radiographic features. The mainstay of treatment is supportive intensive medical therapy with between 30% and 50% requiring surgical intervention. Outcomes are related to the extent of intestinal disease, the severity of the systemic inflammatory response and co-existing morbidities. Despite several decades of advances in medical management and surgical techniques and active research programs in several units worldwide, the morbidity and mortality associated with NEC remains high.

Keywords: enterocolitis, necrotising, paediatric laparotomy.

Illustrative Case

An 11 day old infant on the neonatal intensive care unit (NICU) developed abdominal distension and bilious vomiting. She had been born at 27 weeks gestation and birthweight 760 grams by normal delivery following spontaneous onset of maternal labour. Apgar scores were recorded as 6 at 1 minute and 8 at 5 minutes. She was intubated at 6 minutes of age and transferred ventilated to the NICU. She initially received a 3 day course of intravenous antibiotics but these were stopped as her inflammatory markers were normal and there were no sepsis risk factors. She required a small dose of dopamine for the first day of life to maintain her blood pressure within normal limits. Trophic feeds of maternal breast milk were started on day 5 and increased to full feeds by day 10 of life. She was extubated on day 4 of life onto CPAP on which she remained.

She was assessed by the neonatal registrar who identified abdominal distension and confirmed the presence of bile in nasogastric aspirates. The abdomen, although distended, was soft and not tender. He requested that feeds be stopped, ordered some blood tests and an abdominal X-ray. Blood results were normal with the exception of a raised CRP of 15 (last measured 4 days previously when it was 2). The X-ray (Figure 1) demonstrated pneumatosis intestinalis and a presumed diagnosis of Necrotising Enterocolitis (NEC) was made.

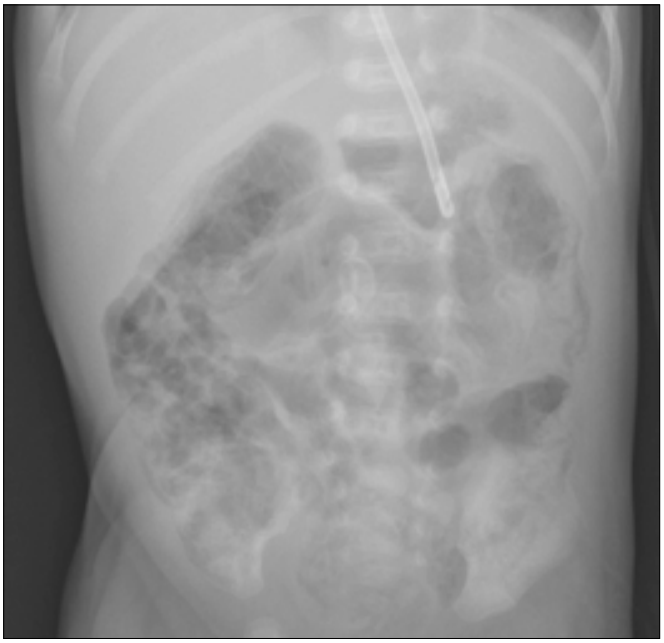

Figure 1 – Plain AXR demonstrating pneumatosis intestinalis in an infant with suspected NEC.

Over the next 12 hours she developed increasing bradycardias and apnoeas requiring intubation and ventilation and required[2] fluid boluses and dopamine (10mcg/kg/min) to maintain her mean blood pressure at 30mmHg. The following day her abdomen was more distended and the X-ray was repeated. The pneumatosis intestinalis persisted with a similar gas pattern in the loops of bowel and some intestinal dilatation. Repeat blood tests showed the following abnormalities: Hb 9.4, WCC 17.2, Platelets 100, CRP 41. Because of the deterioration in her clinical condition she was transferred to the regional Neonatal Surgery Unit for assessment.

By the time she arrived at the tertiary unit she had deteriorated further and now required High Frequency Oscillatory Ventilation (HFOV) to maintain adequate gas exchange. She received a blood transfusion, a further fluid bolus and an adrenaline infusion (0.1mcg/kg/min). She was assessed by the Paediatric Surgical team who felt that her abdomen was distended and tense. A repeat X-ray was taken (Figure 2) which revealed a pneumoperitoneum. She was taken to theatre for laparotomy for presumed perforated NEC.

NECROTISING ENTEROCOLITIS

N J Hall

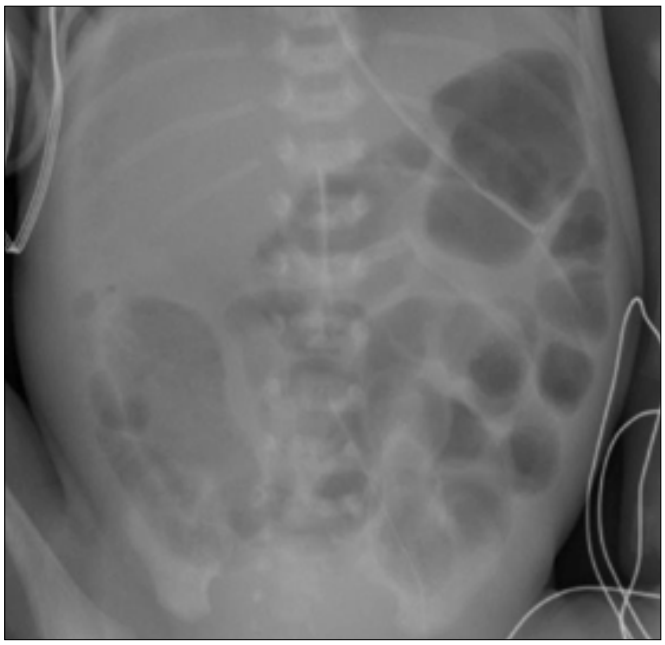

Figure 2 – Plain AXR demonstrating pneumoperitoneum in an infant with NEC.

At laparotomy the distal 25cm of ileum, the caecum and 8cm of ascending colon were dusky and ischaemic with bubbles of gas within the wall of the bowel in places. Between 5 and 10cm proximal to the ileocaecal valve the bowel was white and necrotic with an obvious perforation that had caused only minimal peritoneal contamination. The surgeon elected to resect all of the diseased intestine and planned to perform a primary anastomosis. During mobilisation of the ascending colon, the anaesthetist commented that the infant was requiring significant volumes of intravenous fluid and was becoming unstable. The surgeon removed the diseased intestine and formed a terminal ileostomy in the right iliac fossa. He brought out a mucus fistula in the right lateral end of the transverse laparotomy wound.

Post-operatively the infant remained ventilated on HFOV and on inotropes for 3 days. She became significantly oedematous. She was treated with 7 days of intravenous antibiotics and nil by mouth. Nutrition was maintained with total parenteral nutrition. She gradually improved and was extubated 5 days after her laparotomy. Feeds were re-introduced and tolerated. Fourteen days after her laparotomy she returned to her referring unit for ongoing care.

Three weeks later the paediatric surgeon received a phone call stating that despite adequate calories and electrolyte intake the child was not growing well. She was re-admitted to the tertiary centre where a distal contrast study was performed. This did not show any evidence of a colonic stricture and her stoma was closed the following day. Following this her weight gain improved and she returned once more to her local neonatal unit. She was discharged home 3 weeks later.

Introduction

The case above illustrates many of the salient features of the presentation and management of necrotising enterocolitis (NEC) a devastating disease of infants and the commonest gastrointestinal emergency in the newborn period. NEC is a condition characterised by intestinal necrosis affecting the ileum and/or colon. There is a wide spectrum of clinical manifestations. In the least severe cases there may be mild inflammation of the intestinal wall in a baby with mild abdominal distension and minimal systemic upset. The most severely affected cases, however, may show evidence of full thickness intestinal necrosis with perforation, respiratory and cardiovascular collapse, multi-system organ failure and in some cases death.

The reported incidence of NEC varies from 0.5 to 5 per 1000 live births(1) but NEC is predominantly a disease of preterm infants and those of low birth weight. The incidence is as high as 12% in infants less than 750g(2) and more than 90% of affected infants are born prematurely. Despite over 20 years of active research in the field of NEC, the mortality rate remains unchanged.(3) New treatments are desperately needed to improve outcome from this devastating condition.

Pathogenesis and Risk Factors

The predominant aetiological factors predisposing to NEC are prematurity and low birth weight. It is thought that intestinal immaturity in preterm infants is central to the pathogenesis if this condition. In addition, an array of risk factors have been proposed, none of which explain fully the pathogenesis of NEC in isolation. Moreover it is believed that a number of risk factors interact to cause NEC. Proposed risk factors are shown in Table 1.

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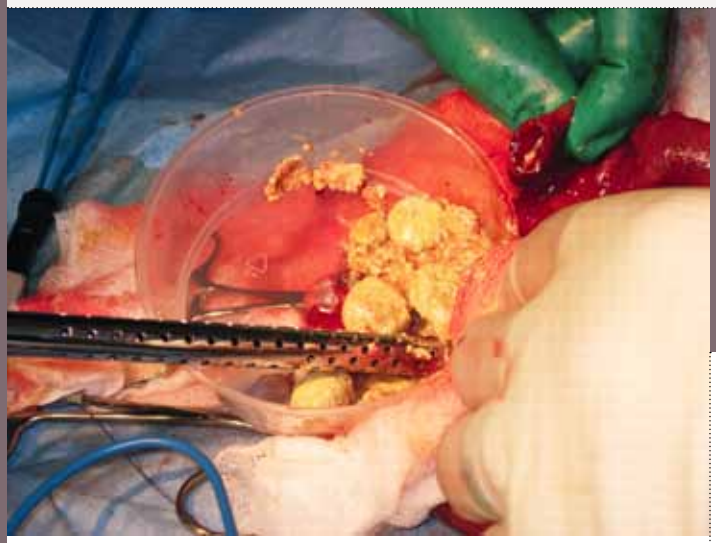

Peripartum events
absent or reversed end diastolic umbilical artery blood flow
maternal eclampsia
fetal distress
premature rupture of membranes
delivery by Caesarean section
perinatal asphyxia
perinatal hypothermia

Neonatal period
respiratory distress syndrome
apnoeic episodes
congenital heart disease
persistent fetal circulation,
persistent ductus arteriosus (PDA)
sepsis
umbilical catheterisation
exchange transfusion
NSAID treatment of PDA

Feeding regimen
formula feed (as opposed to breast milk)
high density milk formulae
early enteral feeding
rapid advancement of enteral feeding

Bacterial involvement
Precise role unclear but intraluminal bacteria
almost certainly essential for the development of NEC

Table 1 – Proposed risk factors for NEC.

Clinical Features And Diagnosis

Infants with NEC usually display specific gastrointestinal signs. In the early stages of the disease abdominal distension with or without tenderness, feeding intolerance with increased gastric residuals, vomiting and occult blood in the stools may all be present. These findings may become more severe as the disease progresses to include abdominal wall oedema, erythema and ascites. A small proportion of infants with NEC present with a palpable abdominal mass (usually due to matted loops of bowel around an area of gangrene or perforation) and/or persistent intestinal obstruction.
In addition to these gastrointestinal signs, generalized non-specific signs indicative of systemic deterioration or sepsis are often present. In their mildest form these include temperature instability, hypovolaemia, tachycardia, and mild respiratory distress. In more advanced disease clinical features of a systemic inflammatory response frequently develop including hypotension requiring inotropes, respiratory failure requiring ventilatory support, coagulopathy and renal failure.

Whilst there are no defining laboratory parameters of use in the diagnosis of NEC, a number of haematological and biochemical abnormalities may be observed including raised or depressed white cell count, thrombocytopenia, metabolic acidosis, glucose instability and elevated C-reactive protein levels. None of these are universally present in all cases.

Radiographic imaging is essential in the diagnosis of NEC. The pathognomonic radiological finding is that of pneumatosis intestinalis (Figure 1) representing gas within the wall of the bowel believed to originate from pathogenic bacteria. If this gas becomes absorbed into the mesenteric circulation it may result in the presence of portal venous gas seen as a narrow, linear air-dense area in the hepatic region on X-ray. The most significant radiological finding is that of pneumoperitoneum resulting from intestinal perforation as this is a clear indication that surgery is required. Free gas may be seen in a number of ways including:

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- **The football sign (free gas outlining the falciform ligament and umbilical arteries)**

- **As a triangular gas shadow clearly not within the intestinal lumen bordered by the subhepatic space and hepatorenal fossa**

- **As Rigler's sign, in which there is clear visualization of the outer as well as the inner wall of a loop of bowel**

In many cases the identification of perforation is challenging and a lateral decubitus radiograph may be useful (Figure 3). There are cases in which intestinal perforation may be represented by a completely gasless abdomen and it is not unusual to find a sealed perforation at laparotomy in the absence of free air on the abdominal radiograph. An abdominal ultrasound scan may be helpful for diagnosis in these cases.

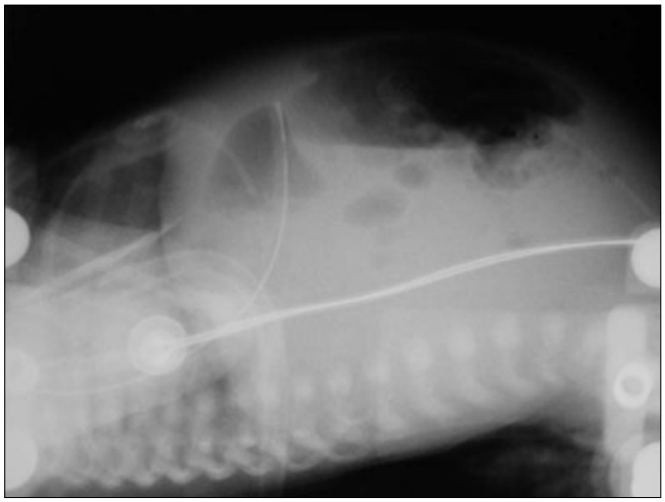

Figure 3 – Lateral decubitus AXR in an infant with NEC demonstrating pneumoperitoneum.

Staging

This combination of clinical features, laboratory indices and radiological findings have been grouped together to form a staging system for NEC known as Bell's staging. This is shown in Figure 4. The use of such a staging system has been used by some surgeons to select the most appropriate treatment but its value is probably greatest in defining severity of disease in determining the effectiveness of therapy on survival and outcome.

Stage	I	IIA	IIB	IIIA	IIIB
Description	Suspected NEC	Mild NEC	Moderate NEC	Severe NEC	Severe NEC
Systemic signs	Temperature instability, apnoea, bradycardia	Similar to stage I	Mild acidosis, thrombocytopenia	Respiratory and metabolic acidosis, mechanical ventilation, hypotension, oliguria, DIC	Further deterioration and shock
Intestinal signs	Increased gastric residuals, mild abdominal distension, occult blood in the stool	Marked abdominal distension ± tenderness, absent bowel sounds, grossly bloody stools	Abdominal wall oedema and tenderness ± palpable mass	Worsening wall oedema with erythema and induration	Evidence of perforation
Radiographic signs	Normal or mild ileus	Ileus, dilated bowel loops, focal pneumatosis	Extensive pneumatosis, early ascites ± PVG	Prominent ascites, fixed bowel loop, no free air	Pneumoperitoneum

Figure 4 – Modified Bell staging criteria for NEC(9).

Clinical Management

Medical management

Most infants with NEC are treated non-surgically although they may require intensive medical management. This may be described as predominantly supportive as there are no specific treatments for NEC. Treatment includes appropriate ventilatory support, adequate fluid resuscitation, inotropic support as required and correction of acid–base imbalance, coagulopathy, and thrombocytopenia. The intestine is rested and decompressed and the infant given broad spectrum antibiotics which can be modified appropriately in light of culture results.

Serial clinical and radiological examination is of extreme importance to monitor progression of disease and detect any evidence of intestinal perforation or other indication for surgical intervention. In the absence of such indication, medical management should continue for 7-10 days depending on severity of illness. Following this feeds may be slowly reintroduced paying particular attention to feed intolerance suggestive of a repeat episode of NEC or intestinal stricture. From the time of diagnosis to re-establishment of full enteral feeds it is essential to maintain nutritional input adequate for tissue healing and repair, and organic growth with parenteral nutrition (PN).

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Surgical management

Despite aggressive medical treatment, a proportion of infants with NEC require acute surgical intervention. Surgeons differ over indications for surgery since there is the potential to cause serious harm by operating on a fragile, critically unwell preterm infant. Indications for surgery are listed in Table 2.

Absolute indications
Pneumoperitoneum
Clinical deterioration despite maximal medical treatment
Abdominal mass with persistent intestinal obstruction
Relative indications
Increased abdominal tenderness, distension and/or discolouration
Fixed intestinal loop on X-ray
Portal vein gas
Thrombocytopenia

Table 2 – Indications for surgery in acute NEC.

The principles of surgical treatment for acute NEC are to remove necrotic intestine and control intra-abdominal sepsis whilst preserving as much intestinal length as possible. Within these principles a number of surgical options exist and the procedure of choice is somewhat contentious. The traditional surgical approach to NEC has been to perform a laparotomy, resect all areas of necrotic intestine and exteriorize the bowel to allow adequate time for healing and growth before restoring intestinal continuity at a later stage.

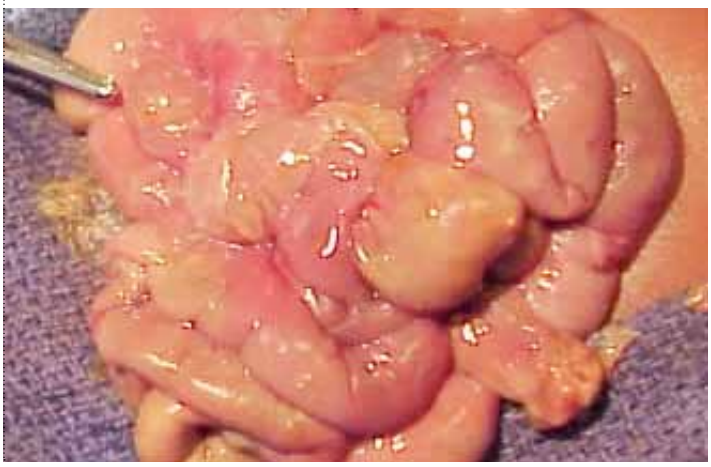
However, stomas, and in particular ileostomies are poorly tolerated by preterm infants as they may predispose to nutritional and metabolic disturbances and poor growth as a consequence of fluid and electrolyte depletion. Some surgeons therefore advocate primary anastomosis following intestinal resection for NEC wherever possible and this is feasible even in small, critically unwell infants.(4) However there is no good evidence to support one approach over the other. In children who are unstable during surgery or have intra-operative complications such as haemorrhage the quickest approach is usually preferable; this is usually to fashion a stoma.

Some children have more than one section of bowel affected by NEC, so-called multifocal disease. For this a number of operations have been proposed including multiple resections and multiple primary anastomoses. A 'clip and drop' approach may also be useful in multifocal disease followed 24-48hrs later by a 'second-look' laparotomy.

Unfortunately a number of infants present at laparotomy with extensive or panintestinal NEC. Surgical options in this scenario are limited and many surgeons would consider withdrawing care faced with an infant with panintestinal gangrene.

A final surgical manoeuvre used occasionally in infants with perforated NEC is placement of a peritoneal drain. Primary peritoneal drainage (PPD) was initially proposed as a method of stabilizing infants with intestinal perforation prior to definitive surgical treatment.(5) Subsequently it was reported as definitive treatment for intestinal perforation as some infants required no further surgical treatment. There have been two recent prospective randomised controlled trials investigating the use of PPD in infants with perforated NEC compared to laparotomy.(6,7)

Whilst neither definitely demonstrated an advantage of either PPD or laparotomy over the other, one study concluded that PPD was not an effective definitive procedure for perforated NEC and that its use should always be followed by a laparotomy.(6) Whether there remains a role for PPD in the stabilisation of a critically unwell chid with perforated NEC and respiratory compromise prior to laparotomy, as originally described, remains unclear.(8). The author's proposed surgical management of NEC is illustrated in Figure 5.



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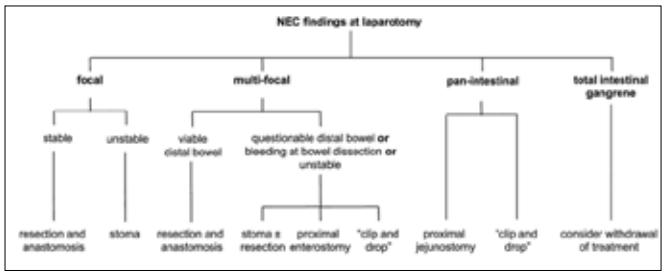


DIC = disseminated intravascular coagulopathy; PVG = portal venous gas.
Figure 5 – Author's surgical strategy for NEC (reproduced from Hall & Pierro (10).

Outcome

Despite intensive medical and surgical treatment a number of infants do not survive the acute episode of NEC. These fall broadly into two groups: those who have panintestinal disease whose intestine cannot be salvaged and those who have surgically and medically and treatable disease but who develop a significant inflammatory response syndrome resulting in multi-organ dysfunction syndrome. Whilst overall mortality from NEC is approximately may be as high as 35%, birth weight is a significant determinant predictor of mortality such that the mortality from NEC is as high as 42% in infants born <750g.(2) With increasing birth weight, mortality from NEC decreases (Figure 6).

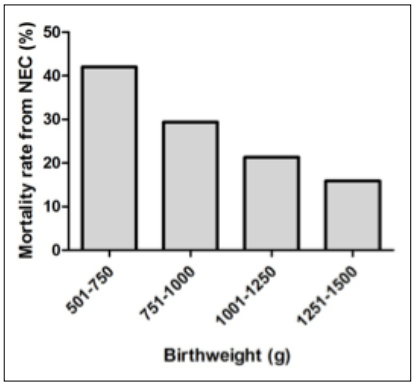
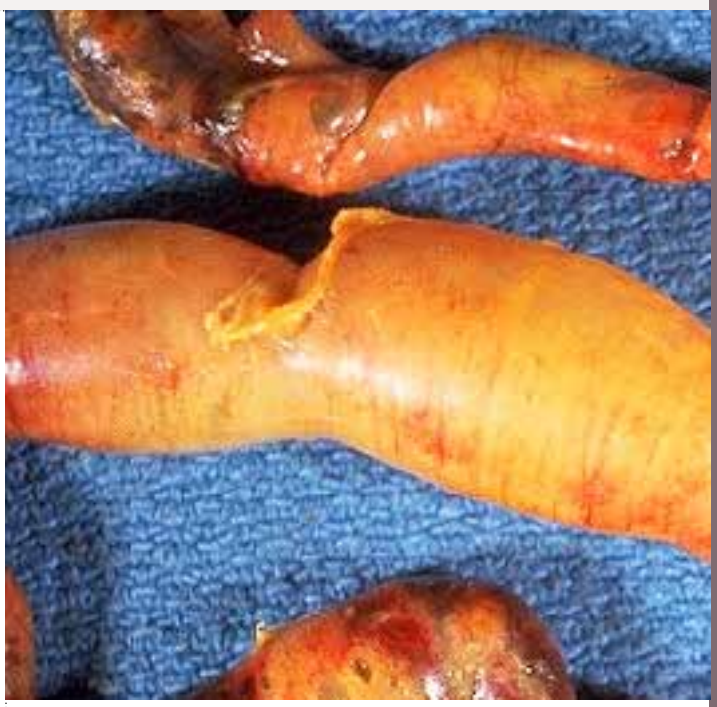


Figure 6 – Mortality of NEC by birthweight category (data from Fitzgibbons et al. (2).

In those who survive the acute episode of NEC, a proportion will develop an intestinal stricture either related to medically treated NEC or at the site of a previous anastomosis. Surgical resection of such strictures is usually necessary. Longer term outcome is related to remaining intestinal length and its capacity for adequate nutrient absorption.



Malabsorption may result from a variety of factors including gut dysmotility, enzyme deficiency, abnormal intestinal mucosa, bacterial overgrowth, decreased bowel length and vitamin B12 deficiency as a result of ileal resection. Short bowel syndrome is the most serious gastrointestinal complication associated with NEC and great efforts are taken to avoid resection of more bowel length than is absolutely necessary. Supporters of resection and primary anastomosis cite this as one of the advantages over stoma formation.

PN-related complications are commonly encountered in infants with NEC and include sepsis, suppression of the immune response and impairment of liver function. Standard strategies to minimise the risk of these complications are used aggressively as some of these infants may have a long term PN dependency.

In addition to the intestinal sequelae of NEC, it is being increasingly recognised that NEC has a deleterious neurodevelopmental effect, the mechanisms of which are not understood. Whilst it is recognised that many preterm infants suffer from neurodevelopmental impairment, neurodevelopmental outcome appears worse in infants who have had NEC. In addition to the intestinal function following NEC it is essential that this important outcome measure is monitored as we strive towards novel therapeutic strategies that are desperately needed.

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Questions

1. The pathognomonic radiological feature of NEC is:

a) Dilated intestinal loops

b) Pneumatosis intestinalis

c) Lack of gas in the pelvis

d) A large gastric gas shadow

2. Which of the following is not a risk factor for NEC?

a) Intra-uterine growth restriction

b) Preterm delivery

c) Breast milk feeds

d) Perinatal hypoxia

3. Regarding treatment of NEC, which of the following is true?

a) All infants with NEC require surgery.

b) All infants with NEC require ventilatory support.

c) A 48 hour course of antibiotics is adequate for advanced disease.

d) The intestine is rested and nutrition provided parenterally.

4. Answer each of the following statements as true or false:

a) Outcome from NEC has improved steadily over the past 10 years

b) About one third of infants who develop NEC require surgery

c) Mortality from NEC does not depend on gestational age

d) Neurological outcome in infants is universally good

5. Regarding surgery for NEC which of the following is true?

a) Only babies with a perforation need an operation

b) The stomach is the part of the bowel most commonly affected

c) Stoma formation is the only surgical option after intestinal resection

d) The entire small bowel may be dead in some cases

Answers

1. b)
2. c)
3. d)
4. a) - False, b) - True, c) - False, d)- False
5. d)

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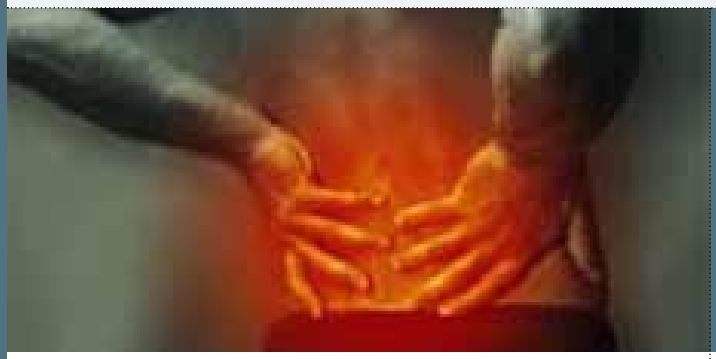
LUMBAR BACK PAIN: IMPORTANT PRESENTATIONS & AETIOLOGIES IN SECONDARY CARE

F Shivji & M Petrie



Lumbar Back Pain: Important Presentations & Aetiologies in Secondary Care
Neurosurgery

Abstract

Lumbar back pain is a common presentation during acute surgical takes. It has a vast number of aetiologies, of which serious pathologies must be excluded. Four important causes of lumbar back pain are disc prolapse, cauda equina, spinal stenosis, and spinal metastases. These conditions can present in a similar fashion to a surgical trainee, hence this article allows surgical trainees to extrapolate the important features of each. Four clinical vignettes are used to describe the salient aspects of the above conditions, including history and examination findings. The pathophysiology and possible management techniques are explained. Surgical treatments are described to allow a surgical trainee to become aware of such procedures.

Keywords: lumbar, cauda equina, spinal stenosis.

Introduction

Lumbar back pain is a common complaint worldwide, across all ages of the population. The majority of patients presenting with lumbar back pain may be managed conservatively. However, there are serious pathologies which must be excluded.

The purpose of this article is to focus on four common presentations of back pain during an acute orthopaedic surgical take and explain the investigation and management of such patients. Brief explanations of spinal surgery techniques are given to allow surgical trainees to become aware of such treatment options.

Musculoskeletal	Paraspinal muscle spasm
Trauma	Vertebral fractures eg. Wedge, Chance
Degenerative	Disc Prolapse (Herniation of nucleus pulposus) Cauda Equina Spinal Stenosis Spondolysis Spondylolisthesis
Infective	Discitis
Neoplastic	Primary bone tumours eg. Giant cell tumour, osteosarcoma Metastases eg. Breast, Lung, Prostate, Renal
Non Spinal Causes	Abdominal Aortic Aneurysm Pyelonephritis

Table 1: Common Causes of Lumbar Back Pain.

Case 1

A 45 year old construction worker, Mr S, presented to the emergency department with a 2 day history of lower back pain radiating down the back of his left leg to his foot. He did not complain of any bowel or bladder symptoms.

On examination, Mr S did not have any palpable spinal tenderness. He had normal sensation throughout his lower limbs but some weakness on dorsiflexion of his left great toe. A straight leg raise of his left leg reproduced the pain at 50 degrees. This was worsened by dorsiflexion of the foot (Lasègue's sign), and relieved by knee flexion.

Disc Prolapse

This is a common presentation of sciatica, caused by a disc prolapse, also known as a herniation of nucleus pulposus. Herniation of nuclear material beyond the intervertebral space results from disc degeneration and dehydration. It has been previously thought that the symptoms of sciatica are solely produced by this mechanical compression of nerve roots. However, recent research has indicated a role for chemical factors causing symptoms.[2] It is speculated that the nucleus pulposus secretes proinflammatory substances such as TNF alpha, which results in nerve root pain. Most herniations are posterolateral, where the posterior longitudinal ligament is weakest, resulting in compression of nerve roots. Central prolapses can also occur which can cause cauda equina syndrome or cord compression, depending on the level of prolapsed.

The highest prevalence of disc prolapse is among people aged 30-50 years, with a male to female ratio of 2:13. Most commonly this involves the L4/5 and L5/S1 discs.[5] Risk factors for disc herniation include occupations that involve heavy lifting, weight bearing sports, and smoking.[5]

Patients complain of a unilateral sharp, shooting pain from buttocks to feet. They may also have weakness of muscles related to the nerve root affected. Important features to exclude from the history are those of cauda equina, which are described below.

On examination, the patient may have no abnormal findings apart from pain during a straight leg raise. This is exacerbated on foot dorsiflexion as the nerve is stretched further.

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Plain X-rays of the lumbar spine have little role in diagnosis. MRI scans (see Figure 1) may provide a definite diagnosis and also may define suitability for invasive treatment, but are usually only obtainable in the acute setting if cauda equina is suspected. It is important to note that many asymptomatic patients have an incidental disc prolapse on MRI.

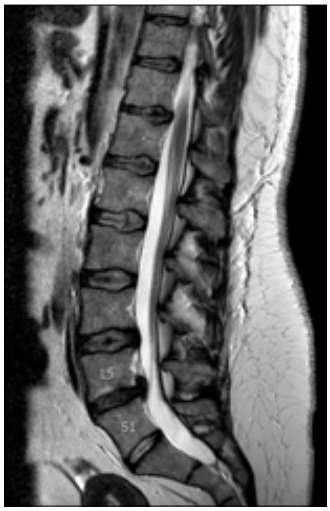


Figure 1: Disc protrusions at L5/S1 and, to a lesser extent L4/5. Lower most rudimentary disc is counted as S1/2.

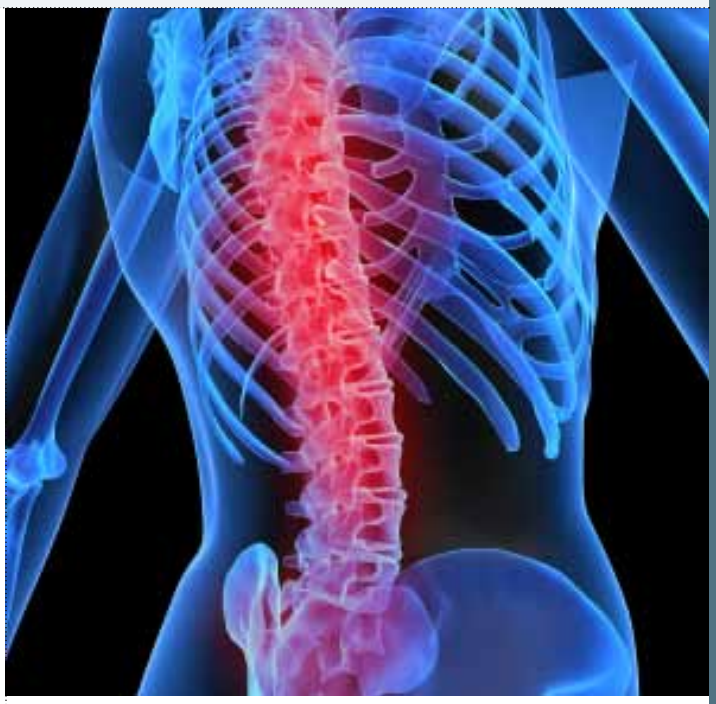
Conservative Treatment

This is usually the treatment of choice for newly presenting sciatica. Bed rest for 2 days is recommended. This can be combined with positional changes such as lying on one side with hips and knees flexed, which has been shown to reduce intradiscal pressure. Analgesia, such as non-steroidal anti-inflammatories, should be used and normal activity started as soon as pain allows.

Interventional Treatment

Epidural injections containing a combination of local anaesthetic and steroid can reduce symptoms in patients. However, this does only lead to a finite period of pain relief.

Operative treatment should be undertaken once conservative management has failed, unless the severity of compression or motor/neurological symptoms is high. It should be noted that operative treatment does not cure the pathology, but can relieve symptoms of leg pain. Discectomy is the operation of choice for disc prolapse (Figure 2). However, although this surgery has been shown to provide faster pain relief than conservative treatment, there is little evidence to show long term effectiveness.[5]



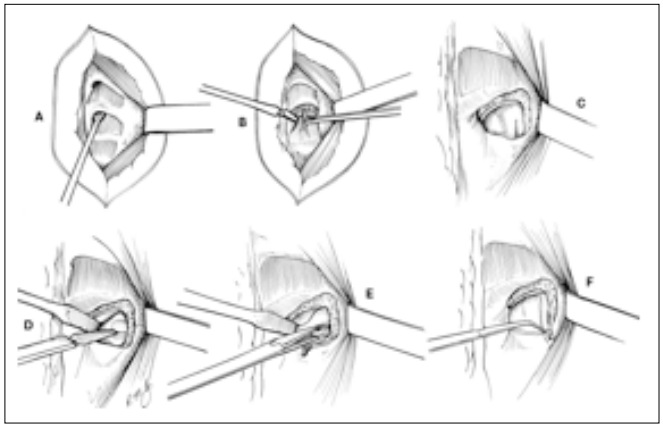


Figure 2: Lumbar Disc Excision Technique (11)

a) Vertical incision over affected lumbar level, expose ligamentum flavum

b) Use curette to remove the ligamentum flavum from inferior surface of lamina

c) Expose dura and root

d) Expose the disc by retracting the nerve root and dural sac

e) Visualise the capsule and remove any extruded disc material

f) Explore the foramen, subligamentous region, beneath the dura and the disc space for additional fragments of disc

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Case 2

Mrs C is a 35 year old female with a history of one episode of sciatica in the past. She presented with a sudden onset of lower back pain radiating down both legs. She did not report any weakness in her limbs. She did not complain of having difficulty passing urine but also admitted that she did not feel the need to pass urine anyway.

On examination, Mrs C did not have any palpable spinal tenderness. Sensation in the lower limbs was normal. There was generalised weakness in her lower limbs due to pain. The house officer performed a digital rectal examination which showed poor anal tone and saddle anaesthesia. A post void bladder scan was performed which revealed 800ml of urine in the bladder.

Cauda Equina Syndrome

Mrs C has Cauda Equina syndrome. The cauda equina is a continuation of nerve roots distal to the conus medullaris, where the spinal cord terminates. The termination of the spinal cord varies from vertebral level T12 to L2, and so it is worth noting that compression in the upper lumbar levels can potentially cause cord compression or cauda equina. The cauda equina can be compressed by disc herniation, spinal stenosis, haematomas and tumours. Cauda equina occurs in 2% of lumbar disc herniations.[3]

Clinical features of cauda equina are bladder, bowel, or sexual dysfunction which may be associated with back pain, sciatica, or neurological changes in the lower limbs.

Potential other causes of cauda equina should be excluded during history taking. For example, anticoagulation, previous spinal surgery, and systemic symptoms should be excluded. It is also important to elicit the timing of onset of symptoms, as this will determine the how urgently the imaging must be performed. If suspecting cauda equina syndrome, a digital rectal examination is essential to aid diagnosis. A catheter should be inserted if the patient is in urinary retention.

Treatment

First line management should be adequate analgesia whilst relevant imaging is awaited. Imaging of choice is an MRI which should be performed urgently so that spinal decompression can be expedited. The timing from onset of symptoms to surgery should be less than 48 hours. The timing of surgery is a controversial issue with regards to recovery of bladder function if incontinence is present. Patients with bladder incontinence at time of surgery have a poorer prognosis.[13] However, if urinary retention is present, earlier surgery has a better outcome. The surgery of choice is lumbar decompression and discectomy (wide laminectomy and extensive decompression with foraminotomies if needed for stenosis).[3]

Case 3

A 65 year old lady, Mrs N, was referred by her GP to the spinal outpatient clinic. She complained of pain and parasthesia in her right leg when she walked. This was relieved if she sat down and rested, but continued on further mobilisation. She had a history of ischaemic heart disease (IHD) and was a smoker. On examination, Mrs N had normal power and sensation in her legs, and normal pulses.

This case poses a problem for surgeons. Mrs N's symptoms appear to be claudication. However, a detailed history is vital to differentiate Neurogenic from Arterial claudication (see Table 2). In this case, arterial disease should be excluded. A simple test that can be carried out in clinic is an Ankle Brachial Pressure Index (ABPI).

Activity	Arterial Claudication	Neurogenic Claudication
Walking	Fixed claudication distance	Variable distance
Uphill walking	Symptoms develop earlier	Symptoms develop later or not at all
Bicycling	Claudication still arises	No claudication
Relief	By standing	Pain can continue whilst standing

Table 2: The Differences between Arterial & Neurogenic Claudication.

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On further questioning, Mrs N explained her symptoms were much worse when walking downhill, and simply standing and resting did not help. The ABPI in her right leg was 0.9.

Spinal Stenosis

These symptoms of neurogenic claudication in this age group are commonly caused by spinal stenosis. Spinal stenosis can affect any part of the spine, with the lumbar spine being afflicted the most.[3] It consists of a narrowing of the central or lateral canals due to degenerative changes in the facet joints, vertebral discs, or hypertrophy of the ligamentum flavum.[3] Causes of spinal stenosis include Osteoarthritis, Inflammatory Arthritides, and Diffuse Idiopathic Skeletal Hyperostosis. Spondylolisthesis can also cause spinal stenosis. Degenerative Spondylolithesis tends to cause central canal stenosis whereas the isthmic (lytic) type tends to cause foraminal stenosis.

Central spinal stenosis can cause neurogenic claudication, contrasting with lateral canal compression of the nerve roots which can result in radiculopathy.[3] Spinal stenosis due to degenerative changes in the spine most commonly affects L4/5.[3] Symptoms are variable, with complaints of back, buttock, and thigh pain precipitated by walking and extension of the spine. 33% of patients can experience muscle weakness and 70% can experience altered sensation, but not necessarily in an exact myotomal or dermatomal distribution.[3]

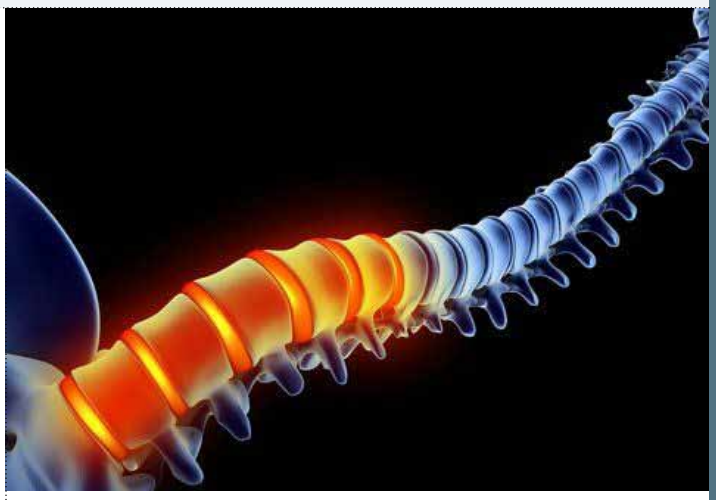
Examination of the spine is usually normal, and therefore a hip examination should be performed as well as a vascular examination, to exclude other pathologies.[3] Imaging is needed to confirm a diagnosis of Spinal Stenosis. A CT scan will identify degenerative changes affecting bony structures, and an MRI is useful to exclude other causes of pain such as disc prolapse. Myelography may also be useful in patients who have had inconclusive scans.[3]

Conservative Management

An initial trial of non operative management is recommended in Spinal Stenosis,[15] much the same as for disc prolapse. This may consist of a mixture of analgesia and physiotherapy. Epidural injections can also be trialed. However, in a recent systematic review,[15] the evidence for the effectiveness this management was found to be lacking, as only small trials have been conducted.

Operative Management

Spinal decompression by laminectomy is the treatment of choice for Spinal Stenosis, once conservative management has failed.[3] This may be combined with fusion if stability is compromised by large bone loss as a result of the laminectomy, or having spondylolisthesis. A brief description is described in



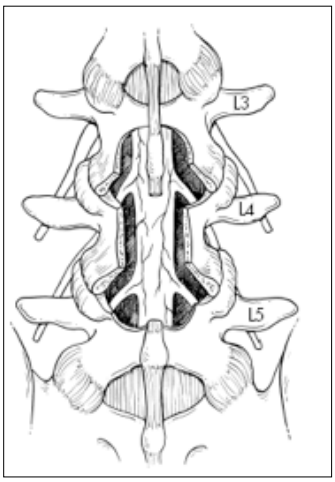


Figure 3: Laminotomy Technique:

1. Midline incision

2. Strip the fascia and muscle to expose the facet joints and pars interarticularis.

3. Remove the spinous processes at the relevant level

4. Dissect the ligamentum flavum and remove the lamina

5. Identify the nerve roots compressed and remove the medial portion of the superior facet.

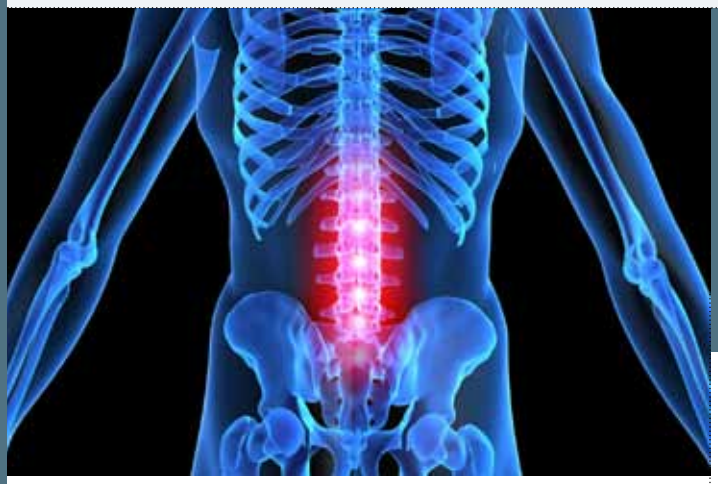
6. If the foramen is narrowed, part of this may also need to be removed

7. Pedicle screws are commonly used if one has to fuse the spine

Typical midline decompression for spinal stenosis. Note medial facetectomy and foraminotomy with preservation of the pars. Decompression is from inferior border of L3 pedicle to superior border of L5 pedicle, exposing both lateral borders of dura in lateral recess.[16]

LUMBAR BACK PAIN: IMPORTANT PRESENTATIONS & AETIOLOGIES IN SECONDARY CARE

F Shivji & M Petrie



Lumbar Back Pain: Important Presentations & Aetiologies in Secondary Care
Neurosurgery

Case 4

A 70 year old lady, Mrs M, presented to the Emergency Department complaining of severe lower back pain. The pain was constant and was not relieved by rest. It regularly woke her from sleep. She had noticed some weight loss but had put this down to a poor appetite. Mrs M had previously been treated and 'cured' after being diagnosed with breast cancer 20 years ago. On examination, Mrs M was locally tender over her lumbar spine. Neurological examination was normal.

Spinal Metastases

This is a relatively classical presentation of spinal metastases in a patient with a history of cancer. Metastatic tumours are the most common types of tumours found in bone,[3] with the lumbar area the second most frequent part of the spine affected (the thoracic region being the most common). Patients usually present with unremitting pain (over 65% of patients[3]) just as Mrs M described. Neurological symptoms can occur but are less common, varying from 5% to 20% of patients.[3] Advanced spinal metastases can present as the acute emergency of spinal cord compression.

Common tumours that metastasise to the spine are breast, prostate, renal, and lung. The reason for this lies in the anatomy of the valveless Batson's venous plexus which allows free drainage between the thorax and pelvis to the spine. In addition, pelvic and mammary veins connect to epidural veins, which may explain how breast and prostate cancer are the leading causes of spinal metastases.[3]

During history taking, it is important to ask about specific 'red flag' symptoms such as night pain and weight loss. Suspicion should always be increased in patients with any history of cancer, even if it was 'cured' years previously. Examination findings may range from completely normal to paralysis.

Imaging depends on the clinical presentation. For example, Mrs M was showing no signs of cord compression and so plain X-rays would be a good choice of initial imaging. A CT of the spine would then be useful to confirm the lesion. Once metastatic disease is the predominant diagnosis, bone scanning would be useful to see the extent of spread. Clearly, at the same time as this is occurring, the primary should be sought.

If the patient had presented with an acute onset of paraparesis with no history of trauma, spinal cord compression would be the main concern. In this situation, an urgent MRI would be imaging of choice so as to identify soft tissues.

Treatment

Treatments for spinal metastases depend on the presentation and life expectancy.[2] For acute spinal cord compression, dexamethasone should be started immediately and liaison with Oncologists and Spinal surgeons should be conducted. Irradiation is an effective choice if symptomatic relief is required. It can remove the severe pain patients' endure and does not subject them to surgery with prolonged recovery time.

Operative management has many indications[17] but it is especially considered when a tissue diagnosis is needed or spinal instability or cord compression results from a lesion.[17] Malignant tumours usually affect the vertebral body, hence anterior decompression is needed. However, there is a vast array of potential surgery which can be carried out to manage spinal tumours.[17]

Summary

Lumbar back pain has numerous aetiologies and will no doubt present to every doctor, no matter what specialty. This article has concentrated on common pathologies which are relevant to the surgical trainee. Important symptoms and characteristics of diseases have been explained, and the management of these has been discussed. It is hoped this will benefit surgical trainees during the acute take and also outpatient clinics.

Questions

1. Regarding Neurogenic Claudication, patients commonly experience which feature?

a) Pain which is worse walking uphill
b) Pain which is worse walking downhill
c) Patients have a fixed claudication
d) Patients have an ABPI <0.8
e) Patients have skin changes in the lower limb

LUMBAR BACK PAIN: IMPORTANT PRESENTATIONS & AETIOLOGIES IN SECONDARY CARE

F Shivji & M Petrie

2. Regarding Spinal Metastases, which venous plexus is implicated in the spread of metastases to the spine?
a) Batson's Plexus
b) Pampiniform Plexus
c) Coeliac Plexus
d) Pterygoid Plexus
e) Intercostal Plexus

3. Regarding cauda equina, saddle anaesthesia refers to sensory disruption of which dermatomes?
a) L1-L3
b) S3-S5
c) L3-S1
d) L4-S2
e) S1-S3

4. A patient known to have a disc prolapse at L4/5, may have what symptoms?
a) Weakness of dorsiflexion of the foot
b) Weakness of knee flexion
c) Weakness of dorsiflexion of the great toe
d) Weakness of plantarflexion of the foot
e) Weakness of knee extension

5. Regarding cauda equina, which feature is associated with a poor functional outcome?
a) Bilateral leg weakness
b) Faecal incontinence
c) Urinary incontinence
d) Urinary retention
e) Altered sensation

Answers

1. b
2. a
3. b
4. c
5. c

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A CAREER IN ORAL AND MAXILLOFACIAL SURGERY

A Tahim, A M C Goodson, K F B Payne & A Chadha

Abstract

Oral and Maxillofacial Surgery involves the diagnosis and management of diseases affecting the face, mouth, jaw and neck. It's demanding, unconventional yet unique training pathway requires trainees to complete both dental and medical undergraduate degrees. In the following article the authors aim to highlight different aspects of the specialty, giving a realistic insight into the training pathway including the second undergraduate degree.

Keywords: oral and maxillofacial surgery; trainee; career; training pathway.

What is Oral and Maxillofacial Surgery?

Oral and Maxillofacial Surgery (OMFS) involves the diagnosis and management of diseases affecting the face, mouth, jaw and neck. Far from the common misconception amongst some doctors that the speciality deals primarily with teeth, the remit of an Oral and Maxillofacial surgeon covers a wide array of diagnoses and procedures – ranging from dental extractions to the management of congenital and acquired facial deformity through to the reconstruction of the face after the excision of a major cancer. Indeed, OMFS is one of the few specialties to resect and reconstruct within its anatomical territory and to deal with both hard and soft tissue pathologies with equal attention.

In this article, we aim to provide the historical context for the development of OMFS and to highlight the different aspects of the specialty. Furthermore, we hope to give a realistic insight into the training pathway and to discuss the practical considerations of a return to undergraduate dental education.

The historical background to OMFS

It is well known that facial surgery has been performed for thousands of years.[1] Relatively speaking OMFS is a young speciality. It has a colourful past with its historical roots in dentistry - developed from the requirement to treat complex jaw injuries sustained by soldiers during the Second World War at which time its practitioners were dentally qualified. However it soon progressed into a hospital based specialty managing civilian traumatic injuries along with other pathological conditions of the face, jaws and teeth when it became logical that both dental and medical qualifications were required. The specialty continued to evolve and in 1994 was formally established as one of the nine surgical specialities and defined under medical directives.[2]

Traditionally trainees complete their dental qualifications before undertaking medical undergraduate qualifications, followed by specialist training. Undergraduate medical curricula provided limited insight into the speciality and therefore the majority of trainees were recruited from dentistry, where, by the nature of the course, exposure to the speciality was far greater. There is now an increasing undergraduate exposure, typically in the form of special study modules, and a rising number of specialist trainees who have studied medicine as their primary degree.

OMFS as a specialty

As defined by the British Association of Oral & Maxillofacial Surgeons, and as documented on the Intercollegiate Surgical Curriculum Programme website,[3] the scope of OMFS is extensive, broadly including, although not limited to:

- **cranio-maxillo-facial trauma**
- **cancer of the head and neck region including appropriate reconstruction**
- **salivary gland disease**
- **surgical treatment of congenital and acquired facial disproportion**
- **cleft lip and palate surgery**
- **aesthetic facial surgery**
- **facial pain**
- **disorders of the temporo-mandibular joint**
- **jaw surgery including surgical removal of impacted and buried teeth, cysts and benign tumours**
- **pre-prosthetic surgery including the placement of osseointegrated implants,**
- **management of infections of the head and neck including life-threatening fascial space infection**

A CAREER IN ORAL AND MAXILLOFACIAL SURGERY

A Tahim, A M C Goodson, K F B Payne & A Chadha

In most OMFS units in the UK, the majority of junior trainees are dentally qualified with an increasing number being drawn from medically-qualified doctors who are undergoing foundation or core surgical training. OMFS junior trainees who are singly qualified in medicine or dentistry face the challenge of dealing with aspects of the specialty pertaining to the qualification they do not yet possess, which presents a steep learning curve.

Medically qualified trainees will necessarily rely on the expertise of their dental colleagues concerning acute dental presentations, while offering their services in the management of the medically compromised patient that they have become accustomed to dealing with during their prior training. Such a heterogenous work force requires excellent team-work and communication – typical hallmarks of the specialty.

Aside from ward duties, the surgical scope of OMFS is broad. OMFS is uniquely placed to satisfy those who enjoy all aspects of the face, both anatomical and pathological. Procedures are diverse, being intra- or extra-oral, elective or emergency, long or short, simple or extremely complex. All, however, are unified by the especial consideration of aesthetics as well as functional rehabilitation of the face leading to the development of a unique management algorithm and surgical skill set.

Towards the end of the five year specialist training pathway there is ample opportunity to apply for one of several Interface subspecialty training schemes from which places are competitively allocated to trainees in OMFS, ENT, plastics and paediatric surgery. In recent years, several OMFS trainees have successfully completed fellowships in Aesthetics, Head and Neck Cancer and Cleft Surgery.

The OMFS Training Pathway

The requirement for dual qualification in medicine and dentistry lengthens the OMFS training pathway somewhat. There is little doubt that the total length of the pathway is one of the longest in the UK health system with recent estimates of training lasting up to 17 years after commencing a primary degree. However time spent in specialist training has been shown to be similar to other specialties.[4]

All trainees are required to complete Foundation and Core training competencies, including passing the MRCS examination. Provided this is achieved by the end of CT1, and the candidate has already completed dental school, they may apply for specialist training at ST3 level at this stage, without completing CT2. The pathway is described diagrammatically on the British Association of Oral and Maxillofacial Surgeons website.[5] This pathway is straightforward for those who emerge from medical school having undertaken dentistry first. But for those who emerge from medical school, interested in an OMFS career, it becomes slightly more complicated. This is largely due to the decision about when to undertake the undergraduate dental degree.

The majority of medically qualified applicants to dental school have sensibly completed Foundation training, although beyond this the range of experience varies. Some applicants apply directly after Foundation training, others after CT1, CT2, CT3 or time spent in other research or trust grade posts. In addition there is no requirement to pass the MRCS examination prior to attending dental school. Once a candidate is dually qualified, they must re-enter the training pathway at the appropriate level to ensure all their surgical competencies are achieved.

ST applications are now centralised by the Severn Deanery, with two annual recruitment rounds. Compared to its competitive surgical counterparts, the application to offer ratio in OMFS is currently favourable although candidates are impressively qualified. Importantly, the current regulations for the ST3 application process requires a candidate to possess their BDS qualification at the time of application, which may delay their application to ST3 immediately after completing their dental studies by one application round. At present it is desirable, but not essential, to have gained 12 months prior OMFS experience before applying for specialist training.

A CAREER IN ORAL AND MAXILLOFACIAL SURGERY

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A Career in Oral and Maxillofacial Surgery
Career Focus

The return to undergraduate education – what is it like at dental school?

The decision to return to undergraduate study is not to be taken lightly, and no doubt stops a great many potential OMFS trainees from considering OMFS as a career. However, for the enthusiastic trainee the benefits outweigh the disadvantages. The knowledge and appreciation of the facial development and the oral cavity, its physiology and pathology, are absolutely integral to the understanding of OMFS and thus of key importance in becoming a successful oral and maxillofacial surgeon. With the limited emphasis placed on this area in both the medical undergraduate curriculum6 and the postgraduate core surgical curriculum, the only avenue to understand this lynch-pin of OMFS resides in formal undergraduate dental education.

The return to university is different second time around for several reasons. Aspiring trainees are often seconded to their local OMFS units and actively enrolled in the on-call rota. Such clinical roles not only provide invaluable experience but a source of income. Furthermore the relationship that is built within these departments allows for participation in audit projects, research and teaching, furnishing opportunities to present at conferences and build and maintain a profile within the specialty.

However, there are important financial implications to consider particularly in the current economic climate.

The lack of a regular income can often be noticeable especially at a time in life when trainees may be considering paying mortgages or starting a family. Many students supplement their work with locum jobs overnight or at weekends, whilst attending classes at university during the day, which can be very tiring. At present, students completing their second degree are eligible for an NHS bursary, although it is likely this may change in the near future and it is not yet clear how this may or may not cover the newly increased tuition fees in addition to the proportion of fees that may be required up front.

Several universities offer four year postgraduate entry dental degrees, and for the past five years King's College London has offered a three year Dental programme for Medical Graduates (DPMG). In light of recent developments arising from EU legislation that question the validity of such shortened courses and their recognition by formal regulatory bodies, all shortened courses are currently undergoing a period of re-evaluation.

This has caused some temporary disruption to the OMFS training pathway at second-degree level although this is fully expected to resolve. In the meantime, any prospective candidate applying to dental school is strongly advised to keep abreast of current developments within this field and liaise fully with the appropriate admissions offices to ensure they are fully appraised of the current situation.

Further information about the specialty

Despite these difficulties that face trainees during their second degree, it is important to remember the transient nature of this part of training. Rather than a sacrifice, to a dedicated trainee who is interested and enthusiastic about this specialty as a career, the return to university will be both clinically beneficial and interesting – a necessary step on the ladder, as any period of formal study such as a Masters or PhD would be.

It is important to gain as much insight into OMFS as possible to make an informed career choice. Unfortunately there are limited opportunities at present for postgraduate trainees at Foundation and Core Surgical training level. Currently the Trent, Northern and Oxford Deaneries all offer Foundation jobs in OMFS. The London and Northern Deaneries offer Core Training posts in the specialty. There are however opportunities to gain further experience in clinical fellow or trust grade posts. Also OMFS exposure is often gained from plastic surgery or ENT rotations, where trainees often cross-cover OMFS overnight.

A CAREER IN ORAL AND MAXILLOFACIAL SURGERY

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There are numerous other resources available to those interested in finding out more about the specialty. These include the British Association of Oral and Maxillofacial Surgeons website, along with its Junior Trainees Group. Other information is outlined in a recent article by Tahim and Awal,[7] describing the various avenues of learning more about OMFS as a career. The online OMFS trainee journal Face Mouth & Jaw Surgery8 is an excellent source of information. The authors who have worked across the country have always found their local OMFS units to be extremely beneficial for further advice and guidance.

Conclusion

Oral and Maxillofacial Surgery is an exciting, expanding and engaging specialty with a demanding, unconventional yet unique training pathway. As such a career in OMFS is attractive to many ambitious junior doctors with an interest in surgery. An increasing awareness of OMFS as a potential career for medical graduates coupled with recent changes to bring training in line with other surgical curricula has made it more accessible and popular as a career for those who have studied medicine as a primary degree. The decision to undertake the second degree cannot be taken lightly but once completed it does provide the passport to undertake some of the most complex and exciting surgical procedures on a part of the body which many lay people regard as requiring the skill of the most able surgeon.

This article has outlined what the specialty consists of, explored its background and discussed the training pathway hopefully providing a valuable insight into the merits of OMFS as a training pathway and career.

The authors all completed medical degrees prior to attending dental school. Arpan Tahim completed Core Surgery and applied to dental school after a Post Core Fellowship in OMFS. Karl Payne entered dental school after completing Foundation Training and Alex Goodson completed Core Surgical Training prior to attending dental school. All three are currently studying for a second degree in dentistry and work part time as Clinical Fellows in Oral & Maxillofacial Surgery. Ambika Chadha completed Basic Surgical Training prior to attending dental school and currently is an ST4 in Oral and Maxillofacial Surgery.

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HOW TO GET THE MOST OUT OF ISCP

JS Nichols, NA Ferran, DJ Bryson & RU Ashford

Abstract

Like it or not, the Intercollegiate Surgical Curriculum Project (ISCP) is here to stay, and it forms an essential part of surgical training in the UK. Used across the nine surgical specialties, trainees are required to complete more and more of their assessments online. Work Based Assessments (WBAs), evidence for the Annual Review of Competence Progression (ARCP), and increasingly, the ARCP itself, will be conducted online. As the website has evolved into its current version 9.3, the functions and capabilities have broadened and adapted to accommodate changes in the surgical curriculum. In Leicester, Orthopaedic trainees have been using ISCP since its inception and have played a role in guiding the integration of the Orthopaedic Competency Assessment Programme (OCAP) into ISCP, so we hold a unique position in helping the surgical trainee, to get to grips with ISCP.

Keywords: ISCP, OCAP, training

Background

Where did it come from? New working practices, influenced by restrictions imposed by the European Working Time Directive (EWTD) have significantly reduced the time that trainees can spend in both formal and informal learning environments. The introduction of shift working for both trainers and trainees has led to a mis-match in working patterns, so the time you can be in the same place as your trainer has been greatly reduced.

On this background, in 2002, the Chief Medical Officer commissioned the report "Unfinished business". The report found in particular that senior house officer training (SHO) training was poorly structured, inadequately supervised, had no definable endpoint and needed major reformation. As a result, it was proposed that a supervised, curriculum-based, time-capped SHO training programme be implemented. Following responses to Unfinished Business by the Surgical Colleges of Great Britain and Ireland, the government published Modernising Medical Careers (MMC) in 2003.

This provided the mandate for the surgical Royal Colleges, and specialty associations to institute major changes to the curriculum, including the development of ISCP.[1] Increasing public expectations for accountability and transparency necessitated a nationally standardised curriculum, with clearly defined expectations, and assessment of competence for each stage with plans for clear progression.

The Joint Committee on Surgical Training (JCST), previously known as the Joint Committee on Higher Surgical Training (JCHST) is an intercollegiate body, working on behalf of the four surgical colleges in the UK and Ireland as well as with the surgical specialty associations. The GMC is responsible for regulating postgraduate training. The GMC requires the Colleges – working through the JCST - to support surgical training. It is the parent body for the ISCP and is responsible for developing and maintaining standards across surgical training.

Why a web based system? ISCP was designed to be convenient, easily accessible site providing a 'one stop shop' for all the information trainees, trainers, managers, patients and the general public require on content, expected standards and assessment methods. It is supposed to be more efficient – a shared site for trainees, trainers and programme directors to work on objectives, progression and assessments which can be easily updated. And finally, it is designed to be secure – a safe location for trainees to record and deposit evidence of training and their experience in preparation for each placement and Annual Reviews.[2]

Why is there a charge for it?

The fee applies to all trainees entering core level, FTSTA or ST3 programmes in surgery, or those continuing these programmes from 2007, who will need to use the curricula and associated websites to complete their training. If your training is to count towards your CCT, the fee must be paid. In previous years, the JCST was funded by The Department of Health (DoH) and by the Joint Surgical Colleges. The costs include those associated with trainee registration/enrolment and recommendation for CCT; developing, maintaining, updating and delivering the curriculum and the online training management system; and supporting national quality assurance and local quality management processes.

HOW TO GET THE MOST OUT OF ISCP

JS Nichols, NA Ferran, DJ Bryson & RU Ashford

So why a charge when it was previously free? In recent years the funding from the DoH gradually diminished and has now ceased completely. A trainee fee for surgical training was subsequently introduced for UK trainees in 2008. The income generated (£590k in the financial year 2010-11) currently accounts for just under 50% of the cost of provision of services to trainees (£1,204k in the same period). Currently the Joint Surgical Colleges receive no funding for any of the trainee-related work that JCST carries out for the GMC. While a re-negotiation of this arrangement is currently in progress, at a meeting of the Joint Surgical Colleges on 17 April 2012, the colleges decided they could no longer meet these financial demands and as a consequence the decision was made that the trainee fee would rise to £150 pa.[3]

So what happens if the fee is not paid? For any given year that the fee is unpaid, you will be unable to access the interactive elements of the curriculum's web platform, and will not be able to use the ISCP system as your online training portfolio. As has been said, if the training is to be recognized for CCT, the fee has to be paid.

So what are the requirements to pass the ARCP?

All training rotations will be slightly different, as all training programme directors are different, but most rotations are trying to do away with all paper records and complete more and more ARCPs entirely online. As a guide, here are minimum requirements for the majority of you.[4]

- **Administrative**

The self-declaration forms should be completed at the beginning of each level in the training programme.

Educational Contract

Statements of Health and Probity

- **Learning Agreement for each placement**

Each stage of the Learning Agreement must be signed off. The Interim Review is not mandatory for 4-month placements but is strongly encouraged. It is essential for 6-month or 1 year placements. The Final Review must comprise an Assigned Educational Supervisor's summative report of overall performance (including evidence of reflective practice).

Objective Setting

Interim Review
Final Review

- **Record of educational achievement**

There should be up to date details of completed educational achievement and self-directed learning. Examples are listed below:

Audits/research/project
Examinations
Courses
Other educational activities

- **Record of Operative Experience**

Records of operative experience should be up to date.

Surgical logbook

- **Workplace-based assessments**

CBD
CEX
MSF
Surgical DOPS or PBA

- **QA Questionnaires**

Trainee evaluation (for each placement)

The ISCP website facilitates completing all these documents online in order to achieve successful outcome in your ARCP.

Essentials from the website

Whilst this paper is not meant to be an exhaustive manual outlining all the functions and capabilities of the ISCP website, we hope to provide a brief run through of the important and useful features of the website and aim to highlight some of the recent improvements.

Curriculum

The curriculum section outlines the way surgical training is delivered across the 9 specialties and core training. There is extensive information on the system concerning appraisal and feedback and an explanation of the development and use of WBA's. The syllabus section is useful as it allows the trainee to peruse the overview of the syllabus of the nine specialties, and core training, or to download the detailed versions in PDF format.

Dashboard

The dashboard portion of the website has many useful features. Among them, the dashboard notice page allows one to monitor their WBA summary and notifies the trainee if they are lagging behind in undertaking WBA's and learning agreements using a traffic light system. Through the dashboard one can receive ISCP messages, pay the JCST fee, and upload your current CV.

HOW TO GET THE MOST OUT OF ISCP

JS Nichols, NA Ferran, DJ Bryson & RU Ashford

How to get the most out of ISCP
Current Training Issues

Learning plans

The learning agreement is an integral part of each training placement and outlines the proposed manner that learning will take place throughout a placement. This section of the website allows the electronic learning agreements to be recorded. The trainee and their supervisors can electronically sign the plans, putting into place a structured agreement outlining objectives and goals for each surgical rotation. When the placement is complete, both the trainer and trainee can sign off the agreement remotely, removing the need for face-to-face meetings which can sometimes prove challenging when parties are in different locales. At a time when training opportunities are increasingly constrained, a planned and focused approach to training is required. Detailed learning agreements and learning plans promote structure and direction and provide measurable and assessable goals.

There have been a number of functional upgrades to the learning agreements as ISCP progressed through its version upgrades. The most recent upgrade saw the ability to record OCAP learning agreements for Orthopaedic trainees, and the introduction of an easy to use procedure profile. Orthopaedic trainees, ST3 or above, are required to set the learning agreement to OCAP at the beginning of each placement. Also contained in the learning plan section of the website is the option of developing a personal development plan and the development of a journal for reflective practice.

WBAs

The WBA section is probably the section most used by trainees. It is here that the ISCP comes into its' own. All WBAs and the Multi-source Feedback (MSF) tool can be found. These assessments are linked to specialty specific syllabi and curricula and are undertaken throughout training with specific numbers of assessments expected of each trainee. Assessments include Case Based Discussions (CBD), Mini Clinical Evaluation Examinations (CEX), Direct Observation of Procedural Skills (DOPS) and Procedure Based Assessments (PBA).

Recent WBA's added include the Observation of Teaching's (OOT) and Assessment of Audit. Procedure Based Assessments (PBAs) have evolved into the principal workplace-based assessment method for technical skills (5) and each surgical specialty has compiled a list of index procedures.

The PBA assessments encompass all aspects of the procedure including such elements as consent, patient positioning, communication with members of the theatre team, and post-op note documentation. In order to maximise their value, PBAs should be prospective. Criticism has previously been leveled at retrospective approach to completion of these assessments with trainees requesting that their supervisors complete the PBA only on those occasions when a procedure has gone well and the desired level has presumed to have been achieved. Trainers are actively dissuaded from completing WBAs retrospectively.

A very useful update is the addition of a 6th version of DOPS. The introduction of a generic DOPS form allows trainees of all specialties to record a learning experience when taking part in any surgical procedure. This allows for feedback and refection to occur for procedures other than those covered by PBAs.

Evidence

Surgical trainees are required to show evidence of continuing professional development and participation in audit and research. The Evidence section allows you to document and collate all extra-curricula activities and accomplishments, including attendance at courses and seminars, publications, presentations and posters, and examinations. This provides a global and holistic picture of you as a trainee and may be cited as evidence for a commitment to a specialty and person specifications for applications to higher specialist training.

The introduction of revalidation requires all doctors to provide evidence of patient feedback. The Evidence section facilitates the recording of the necessary ARCP evidence and has been expanded to include a patient feedback facility, which allows trainees to record episodes of written and verbal feedback. It gives you the opportunity to log journal entries on the website which may be personal and hidden from those who have access to a trainees' portfolio, or made visible to educational/clinical supervisors and training programme directors. All forms of evidence can be supplemented with uploaded files. Abstracts, publications, conference programmes, attendance certificates and written patient feedback can be scanned and uploaded. A maximum of 40 megabytes of data storage is allowed.

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Logbook

Since 2003 it has been compulsory for all specialist registrars in the UK and Ireland to submit data to the Trauma and Orthopaedic Logbook.[6] In 2011 the Pan-surgical e-Logbook was accepted as the universal logbook for all surgical subspecialties in the United Kingdom and Ireland. The e-Logbook has been linked to the ISCP website and a summary tool has been integrated to allow generation of logbook summaries by placement or for all of training.

Portfolio

The portfolio summarises your learning agreements and achievements for review at ARCP. Each part of the portfolio can be viewed by placement, training level, or all of training.

ARCP

The ARCP section contains a record of completed ARCP's. Each ARCP report can be reviewed in detail by the trainee.

Engaging a trainer in ISCP?

In order to get a trainer engaged and on the website, so you can get the most out of ISCP, they need to know how they can benefit.

Using ISCP as a trainer allows them to accurately monitor all the trainees they supervise. It lets them track the WBAs they have validated, and all the WBAs their trainee has completed. Validating the trainees' logbook seamlessly transfers the details into the trainers surgeon's portfolio, without having to manually enter all that data themself. It provides useful for evidence for job planning and supports the use of your SPA (Supporting Professional Activity) time.

As a trainee gets accustomed to preparing for the ARCP process, a consultant needs to get used to the revalidation process. They have to undertake a yearly apprasials and regular revalidation. The information currently recorded on ISCP is very similar to that which consultants need to present for revalidation. If trainees are adept at using ISCP, the changes that are faced as a consultant will not be as daunting. The Edinburgh College surgeon's portfolio is similar to ISCP, and can help collate the evidence needed to be produced at appraisals.

As well as a logbook and surgeon's portfolio, it can show evidence of continuing professional development by maintaining knowledge using CBDs and OOTs. It will show reflective practice in the 360 degree appraisal of trainers, but also shows evidence of audit of practice and maintaining standards.

So what next?

As we said initially, like it or not, ISCP is here to stay. It is an essential part of surgical training in the UK. Hopefully we've shown you how to get to grips with it, and some of the benefits of using the site. ISCP has become increasingly user-friendly and updates and improvements will continue throughout your surgical training. Who knows, maybe the ISCP will merge with the consultant's portfolio in the way it has with OCAP? So, getting the hang of ISCP now will allow you to progress smoothly throughout your surgical training.

Surgical training necessitates the acquisition of many new skills. Trainees will never be assessed on using ISCP, but this is one skill and one competency that cannot be overlooked.

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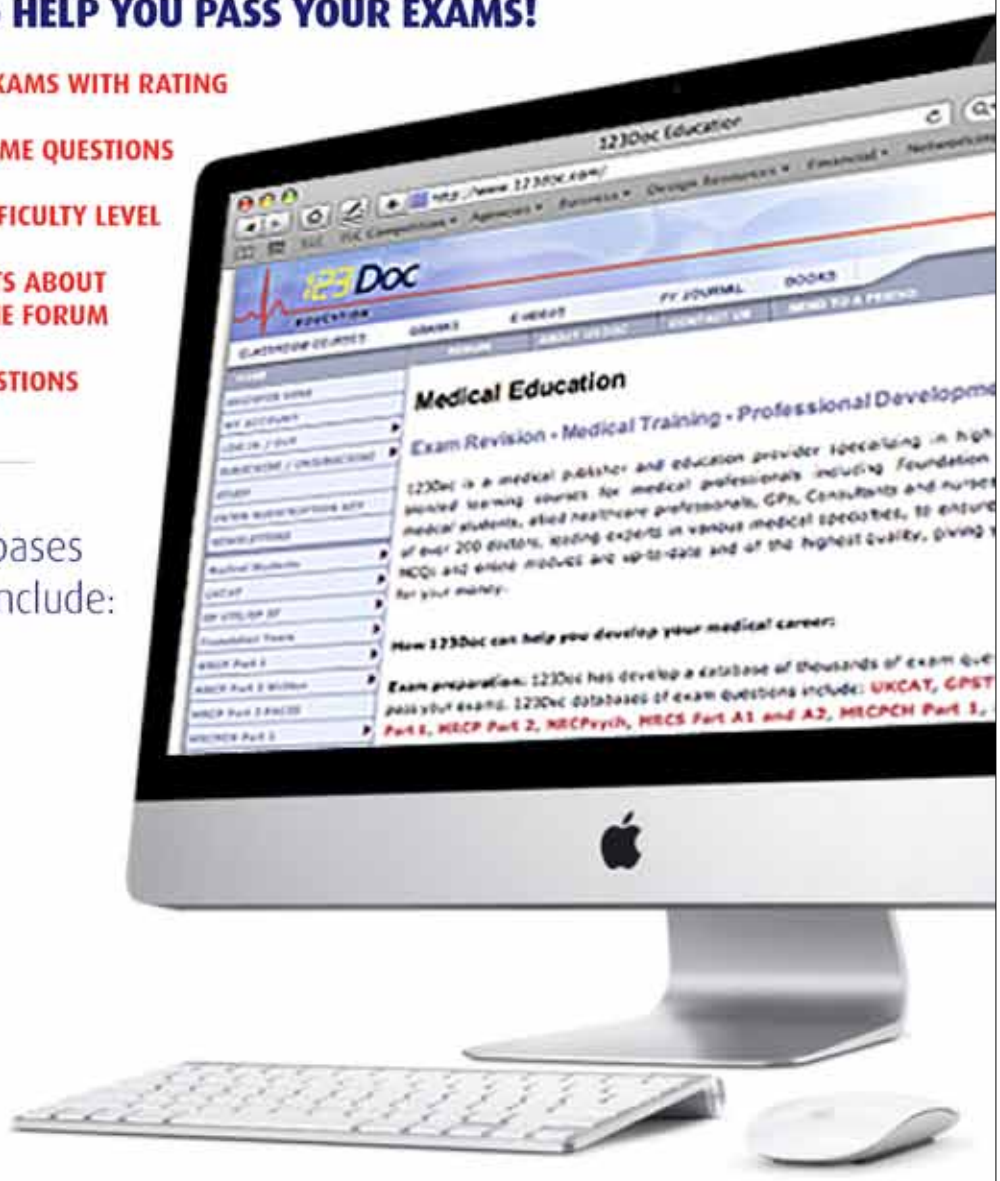

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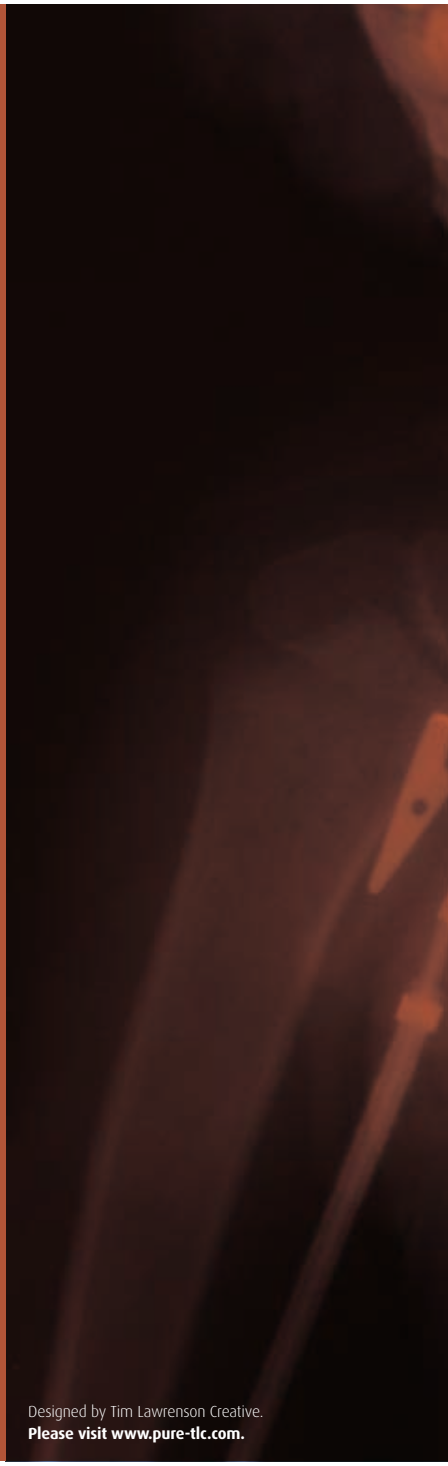

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