123 Doc EDUCATION

CORE SURGERY JOURNAL

Volume 1, Issue 1

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Contents

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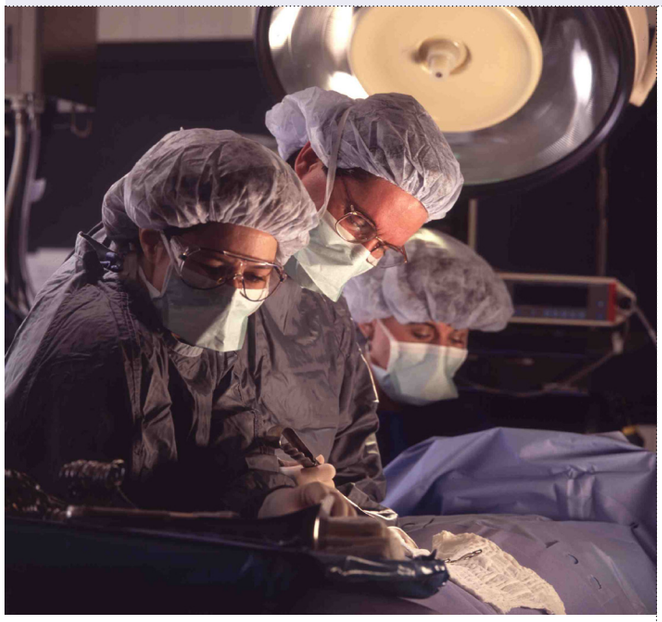
CORE SURGERY JOURNAL

Volume 1, Issue 1



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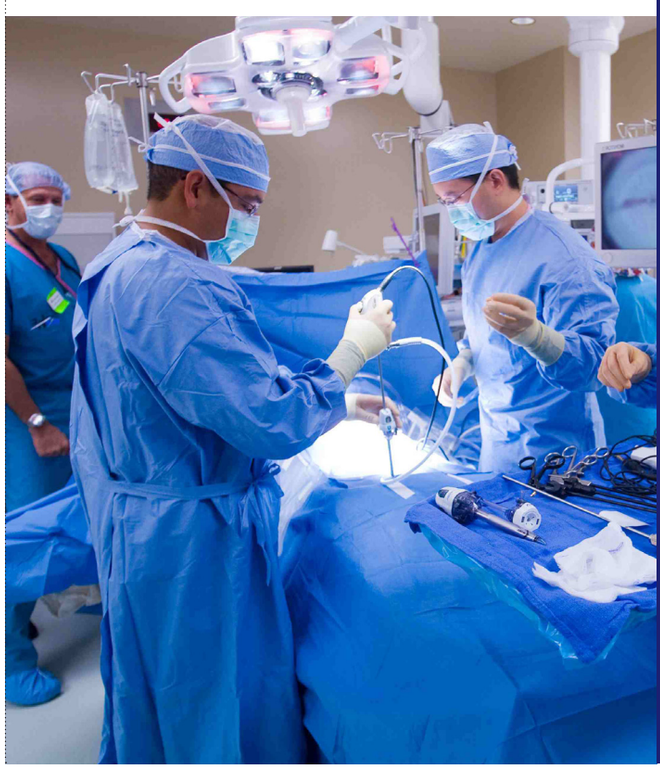

CORE SURGERY JOURNAL

Volume 1, Issue 1

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 each of eight selected subspecialty fields; General Surgery, Orthopaedics and Trauma, Plastic Surgery, Ear Nose and Throat Surgery, Neurosurgery, Urology and Paediatric Surgery. 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. Articles will be commissioned from this list and authors are advised to agree a topic 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 **coresurgicaltrainee@googlemail.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. References should be cited using superscript numerals 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.**

Copyright

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Format of Articles

Guidelines for the format of respective article types are as follows:

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:

- 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. Completed audit cycles are strongly preferred as are audits which have led to guideline development.

CORE SURGERY JOURNAL

Volume 1, Issue 1

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.

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)

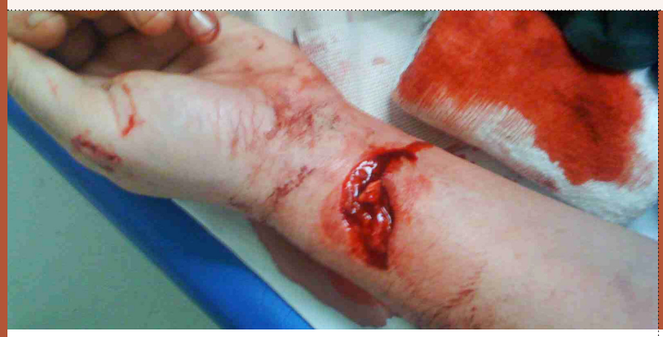
Please note that all articles should be submitted with five multiple choice questions (MCQs) or extended matching questions (EMQs) attached, in the style of the Member of the Royal College of Surgeons (MRCS) 'Part A' examination. These questions should have answers and brief teaching notes/discussion 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.

A STITCH IN TIME

Alana Mitchell



There is a wide choice of wound closure options from which to select. The surgical trainee must assess wound factors, such as healing rate of the tissue being sutured and requirements for wound support, as well as patient factors such as obesity and infection. Back to Basics.

A Stitch in Time...

There is a wide choice of wound closure options from which to select. The surgical trainee must assess wound factors, such as healing rate of the tissue being sutured and requirements for wound support, as well as patient factors such as obesity and infection.

The requirement for wound support varies depending on the tissue being sutured, from two to three days for muscle, to permanent support for vascular grafts. Ultimately, the surgeon must ensure the suture they choose will maintain support until the tissue is strong enough to manage the load applied to it.

This article aims to review the different types of suture material available and the needles onto which materials are swathed, as well as reviewing the other wound closure options that surgical trainees should know about.

Types of Suture

Sutures can be classified in different ways. Commonly they are divided into:

- Absorbable
- Non-absorbable

Absorption can be achieved by enzymatic means for biological sutures, e.g. catgut, which is not used in the UK, or by hydrolysis in the body tissue fluids, for most sutures currently used in the UK (1).

Non-absorbable sutures do not break down, and are removed after a period of time when used in skin closure.

Alternatively, sutures may be broadly classified as:

- Multifilament
- Monofilament

Suture Characteristics

There are several factors to consider when comparing sutures. These can be summarised as:

Advantages Of Multifilament Sutures	Advantages Of Monofilament Sutures
Handle more easily	Reduced risk of infection
Better knot security	Tie smoothly

Memory

This is the tendency of a suture to return to its former shape (3). Monofilaments have more memory then multifilaments. Greater memory can make a suture less easy to handle, and can reduce the security of knots tied.

Capillarity

As multifilament materials have increased capillarity, the increased absorption of fluid may act as a tract for the introduction of pathogens. Monofilament suture is made of a single strand and are considered more resistant to harbouring microorganisms; there is some evidence that braided sutures may hold up to three times as many microorganisms as monofilaments (2). They also exhibit less resistance to passage through tissue than multifilament suture. Great care must be taken in handling and tying monofilament suture because crushing or crimping of the suture can weaken it and lead to the suture failing prematurely.

Calibre

Modern sutures are available in a range of diameters from #6 (heavy braided suture) to #11-0 (fine monofilament) (1). Strictly, this classification is dependent on the breaking strength of the suture, rather than purely on its size (3). In general the smaller the size of the suture, the less tensile strength it will have, though this will also be affected by the material and whether it is multifilament or monofilament.

A STITCH IN TIME

Alana Mitchell

Commonly used absorbable sutures

1. Multifilament

Vicryl ® (manufactured by Ethicon ®)
Polyglactin. Approximately 75% of tensile strength is retained at two weeks, with complete absorption by 56-70 days (1).

Dexon ® (manufactured by Covidien Syneture®)
Polyglactin. Provides wound support for 3 weeks, with complete absorption in 60-90 days (4).

Safil ® (manufactured by Braun ®)
Polyglycolic acid. 50% tensile strength is retained at 18 days, with complete absorption at 60-90 days (5).

It is worth noting that the different products made by different manufacturers share similar properties in terms of absorption rate and support provided. As a whole, these sutures are not appropriate when long term tissue support is required as they absorb too quickly. Instead, they are often used for general tissue approximation. There may be dyed or undyed varieties. Dyed may be easier to see, but undyed may be more suitable when suturing in superficial tissues and skin. Coated varieties may have slightly altered properties, such as more rapid absorption, or greater antimicrobial resistance.

2. Monofilament

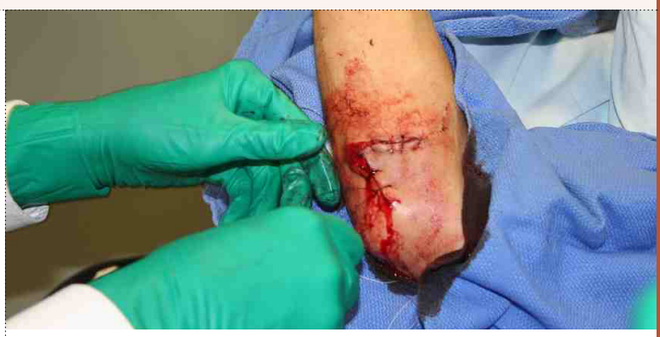
Monocryl ® (manufactured by Ethicon ®)
Poliglecaprone 25. This synthetic suture comes in dyed and undyed varieties. Importantly, these varieties have different properties as well as different appearances. For the undyed, 20 – 30% tensile strength is retained at two weeks, with complete absorption by 90 – 120 days. The dyed version takes longer to lose tensile strength and to absrob (1).

Monosyn® (manufactured by Braun ®)
Glyconate. Retains 50% of tensile strength at 14 days with complete absorption in 60-90 days (5).

Maxon ® (manufactured by Covidien Syneture®)
Polyglyconate. Supports wounds for six weeks, with complete absorption by 180 days (4)

PDS ® (manufactured by Ethicon ®)
Polydioxanone. Approximately 60% tensile strength remains at six weeks, with complete absorption by 180 – 210 days (1).

It is apparent that there are two broad categories here. Some products absorb more rapidly, with others providing longer term support. The specific uses of different absorbable monofilaments will vary between surgeons, but the general differences between them should be considered.



Commonly used non-absorbable suture materials

Silk
This is produced as a braided non-absorbable suture. Whilst it can be used for general soft tissue approximation, it does cause an acute inflammatory reaction in the tissue (1). It is often used to secure surgical drains.

Nylon
Examples of this non-absorbable monofilament include Ethilon®, Dafilon® and Dermalon®. As with silk, progressive degradation may occur over time, and so should be avoided where long-term tensile strength is required. It is widely used for soft tissue approximation (1,4,5).

Polypropylene
This is also used in nonabsorbable monofilaments, such as Prolene®, Premilene® and Surgipro®. In contrast to nylon, it is not affected by prolonged time in tissues. Consequently, it has a wide variety of potential uses (1,4,5).

Stainless steel
Steel can be used as a monofilament that will maintain tensile strength. As a result, it is used for abdominal wall closure, hernia repair, sternum closure and orthopaedic procedures (such as cerclage wiring fractures) (1). However, it is inelastic, and so will not allow tissues to swell with oedema (3).

Surgical Needles

The job of a needle is to carry the suture through tissue whilst causing as little unnecessary trauma as possible. The ideal needle would pass readily though tissue, be resistant to bending but without being brittle and of course sterile. Achieving this will require different needle designs for different applications. Nevertheless, most needles are made of surgical steel, and comprise three parts (1):

- Attachment end (or swage)
- Body
- Point

Most modern sutures come attached, or swaged, to an appropriate needle.

A STITCH IN TIME

Alana Mitchell

Body

Needles may be curved or straight. Some surgeons use straight needles to suture skin, holding the needle by hand. Large curved needles are also produced to be used by hand, for example when securing lines in a patient on the ward. Needles vary in the shape of the curve ranging from 1/4 circle, through 3/8 circle, 1/2 circle, to 5/8 circle. The needle is held on the body by the needle holder when suturing. The cross-section of the body often accounts for this function, for example, it may be rectangular.

Point

The point of the needle extends from the maximum cross-section of the body to the extreme tip. Needles are sometimes referred to by the shape of the point. The surgical trainee should be aware of several types. A major distinction is into cutting needles and round bodied needles.

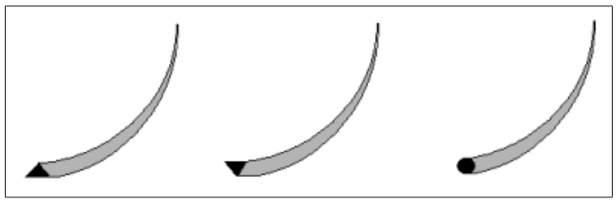


Figure 1: Conventional Cutting, Reverse Cutting and Round-bodied Needles

Types of needle

Cutting types

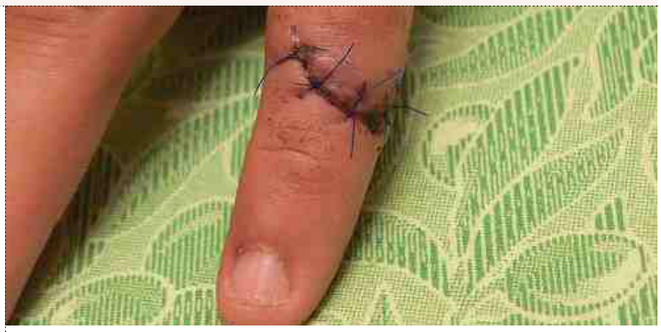
This group comprises two main types: conventional cutting and reverse cutting. These needles are used to suture tough material, such as skin. They are both triangular in cross section, with the apex facing inwards for conventional cutting and outwards for reverse cutting. Manufacturers suggest that reverse cutting needles are stronger and less susceptible to bending (1). Other texts also suggest that sutures placed with reverse cutting needles are less likely to "cut through" under tension (3). Reverse cutting needles may be useful for suturing tendon, fascia or ligaments (3).

Round bodied types

Round bodied needles are used in situations where tissue fibres split and so does not need to be cut, or when tissues are fragile. Furthermore, the tissue tends to close around the suture, which is ideal when a leak-proof seal is preferable, for example when suturing vessels or bowel (1). The exact tip shape varies, and includes blunt tip needles for suturing friable tissues, and to minimise the risk of needlestick injury (1).

Other types

There are also other types of needle that may be encountered in specialist settings. These include spatulated needles, which are used in ophthalmic and microsurgery, and avoid cutting through and cutting out, by eliminating the third blade of the needle (1). They are described in some texts as "side-cutting" as a result (3).



Other Wound Closure Options

Surgical trainees will also come across other useful options for apposing tissues. These include:

Cyanoacrylate

"Skin glue" can be used to quickly close well apposed skin wounds that are not under tension (3). It will eventually flake and peel off.

Staples

Staples can be used to close skin. They place the wound under less ischaemia than other methods (3). They require removal after an appropriate interval, just as interrupted non-absorbable sutures do.

Barbed sutures

These are basically absorbable monofilament sutures that have barbs placed along them, such that traditional knots are not required to secure the suture. Two examples are QuillTM SRS (by Angiotech®) (6) and V-Loc™ (by Covidien Syneture®) (7).

Paper sutures

Paper sutures can be used to close skin wounds themselves, or to support wounds already sutured (8). They can be applied quickly in a variety of settings, with little additional equipment required.

Choosing Between them!

As alluded to, certain sutures may be more appropriate than others in particular situations. Furthermore, there is not always one correct answer. For example, whilst some surgeons may close skin with interrupted nylon sutures, others may use absorbable braided sutures or glue or staples. The important point is to understand the capabilities and properties of the product you have chosen, to optimise the result for your patients.

It is worth familiarising yourself with the particular brands available in the theatres in which you work. Different trusts may have contracts with different suppliers. And, as with everything, it is always worth learning what your boss's preference is too...

A STITCH IN TIME

Alana Mitchell

Question Time

1. Which of the following are non-absorbable sutures?
A. Monocryl ®
B. Maxon ®
C. Dexon ®
D. Ethilon ®
E. Vicryl Rapide ®

2. Which of the following is the most appropriate to secure a vascular anastamosis?
A. Monocryl ®
B. Prolene ®
C. V-Loc ®
D. PDS ®
E. Cyanoacrylate glue

3. Which of the following would be most appropriate calibre of suture to close a wound on a child's face?
A. "0"
B. "2-0"
C. "4-0"
D. "6-0"
E. "10-0"

4. Which of the following sutures provides the longest duration of wound support?
A. Monosyn ®
B. PDS ®
C. Dexon ®
D. Vicryl ®
E. Safil ®

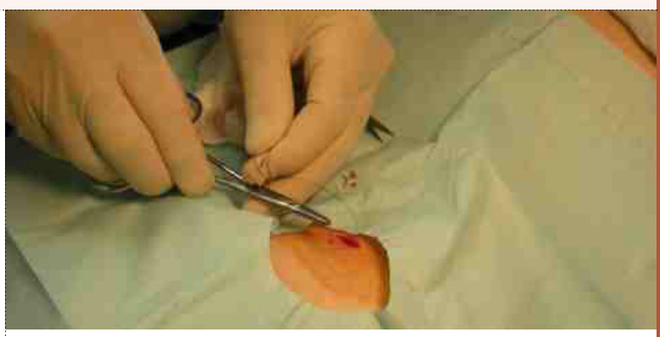
5. Which is the most appropriate needle for suturing skin?
A. Cutting
B. Round bodied
C. Spatulated
D. Blunt tip

Answers

Question 1: D, Ethilon ®. This is a nylon suture.

Question 2: B, Prolene ®. This is a stable non-absorbable suture, so will provide long term support.

Question 3: D, "6-0". A, B and C are too heavy, and E is reserved for microsurgery.



Question 4: Answer: B, PDS ®.

Question 5: A, Cutting.

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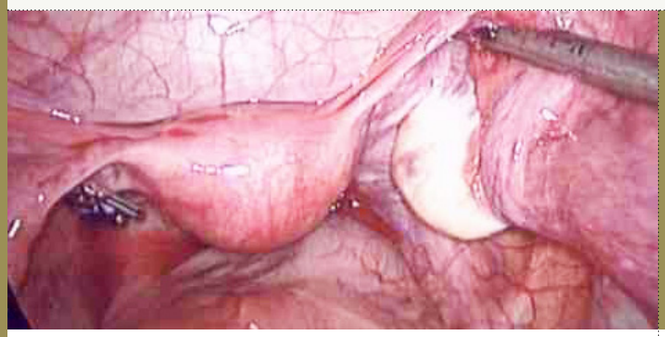
APPENDICECTOMY: STEP BY STEP PICTURE GUIDE. LAPAROSCOPIC SURGERY.

Kulbir Mann



Laparoscopic Appendicectomy.
Featured Specialty: General Surgery.

1.1 Aetiology and Pathology

Appendicitis is a common condition causing inflammation and infection of the appendix. It occurs more frequently in younger patients but also afflicts patients older than 30 years. Its pathogenesis is based upon persistent luminal obstruction causing secretions to develop into a collection thus increasing luminal pressure. The appendix wall becomes ischaemic leading to vascular thrombosis, gangrene and eventual perforation. This can either wall off with omentum and surrounding structures to cause an appendiceal mass or cause peritonitis. [1] The primary cause of appendicitis is mainly idiopathic but Table 1 shows less common causes.

Location	Cause
Intra-luminal	Foreign material, Parasites
Transmural	Viral (Adenovirus, cytomegalovirus) Bacterial (Yersinia, Mycobacteria) Parasitic (Strongyloides, Schistosoma)
	Inflammatory (Pseudomembranous colitis, Ulcerative Colitis, Crohn's disease)
	Vascular (Ischaemic, Angioma, Angiodysplasia)
	Neoplastic (Appendiceal carcinoma, carcinoid, lymphoma, psuedomyxoma & Caecal carcinoma)
Serosal	Gynaecologial (Salpingitis, Oophoritis, Endometriosis)
	Diverticular disease
	Auto-immune disease (PAN, RA)

Table 1; Rare causes of appendicitis [2]

1.2 Clinical Features and Diagnostic difficulties

The presentation of a patient with appendicitis has been well described and taught to all medical students, doctors and surgical trainees. This will not be focused upon here but there are common difficulties in diagnosis.

Young female patients that present with vague clinical signs and unsupportive blood results may require further investigation. A detailed gynaecological history should be sought and referral to gynaecology may be appropriate. We must consider torted ovarian cysts, pelvic inflammatory disease, ruptured ectopic pregnancy and mittelschmerz. Imaging to rule out ovarian disease may be helpful before an operation.

Patients with symptoms of diarrhoea, especially bloody, need investigating for inflammatory bowel disease and should not be operated upon. On rare occasions an operation may reveal terminal ileitis and the patient may proceed with conservative management or resection depending on the clinical picture. A gastroenterology opinion should be sought.

Elderly patients may present similarly with appendicitis but are more likely to form an appendiceal mass. Care should be taken to examine for this and may require imaging. Masses can be treated conservatively with antibiotics and observed. An interval appendicectomy may be indicated but is often at the discretion of the consultant surgeon. There should always be a concern of underlying carcinoma in these patients.

Pre-operative antibiotic usage is only reserved for patients who have clinical appendicitis, are waiting an operation and are systemically unwell. They will benefit from antibiotics prior to theatre. Otherwise antibiotics can obscure the clinical picture and hinder decisions to operate, especially in those patients who are unclear diagnostically.

APPENDICECTOMY: STEP BY STEP PICTURE GUIDE. LAPAROSCOPIC SURGERY.

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1.3 Management and Operative Indications

Investigations mainly revolve around ruling out differentials. Ultrasound and CT can be used for investigating a mass, acute gynaecological pathology and renal pathology.

The indication for an open appendicectomy is a clinical diagnosis of appendicitis. Contraindications include suspicion of other pathology that needs further investigation e.g. inflammatory bowel disease and gynaecological disease. If the patient has a mass clinically and upon imaging then a decision on conservative management should be discussed. If there is evidence of other disease then the patient should be referred appropriately.

Laparoscopy is often used in young female patients with vague clinical signs and is an essential investigation. Contraindications are few and include patients who have had previous operations with extensive adhesions, those with coagulopathies and those on radiation and immunosuppressive therapy. It is generally contraindicated in pregnant ladies in their first trimester. [3]

1.4 Consent

Laparoscopy is an exploratory procedure and consent revolves around explaining the operative management of appendicitis and also locating disease elsewhere that may require other speciality opinion. It is important to mention iatrogenic injury that may occur during port insertion or instrumentation. Conversion to an open procedure should always be discussed.

1.5 Operative Preparations

The patient is given a general anaesthetic and placed supine. The patient is placed supine and the equipment table and stack set are placed towards the patient's feet. The surgeon is on the left of the patient with the assistant. [3]

A laparoscopic operation requires the general set and a laparoscope. The stack provides further equipment for laparoscopy, which include monitors, a light source and an insufflator providing CO_2. An assistant familiar with laparoscopy should be guiding the camera and be aware of the surgeons intentions. [3]

Before patient preparation a patient safety check should occur between surgeons and theatre staff including the operation and patient allergies. The laparoscopy field is bordered by the xiphisternum and symphysis pubis inferiorly and superiorly and the anterior superior iliac spines laterally.

Wound infection is the commonest complications following appendicectomy and antibiotics should be given at induction of anaesthesia.

1.6 Laparoscopic Appendicectomy

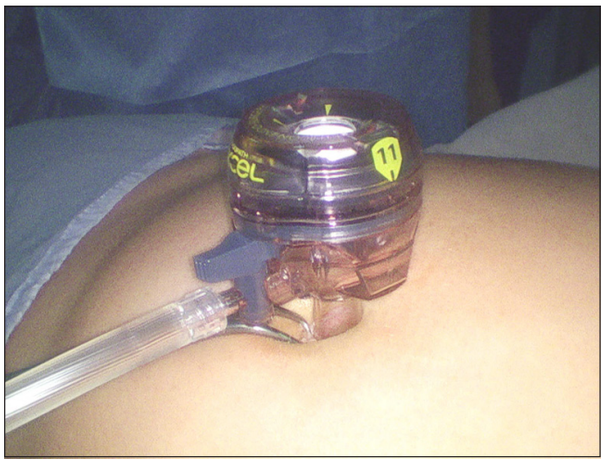


Figure 1; Camera 11mm port inserted

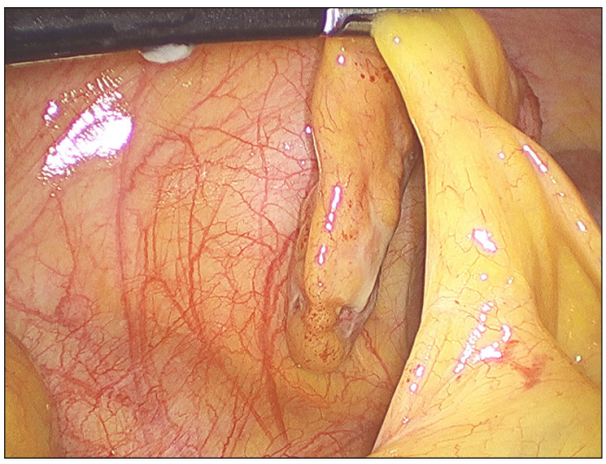


Figure 2; An inflammed appendix elevated by a grasper at the mesoappendix

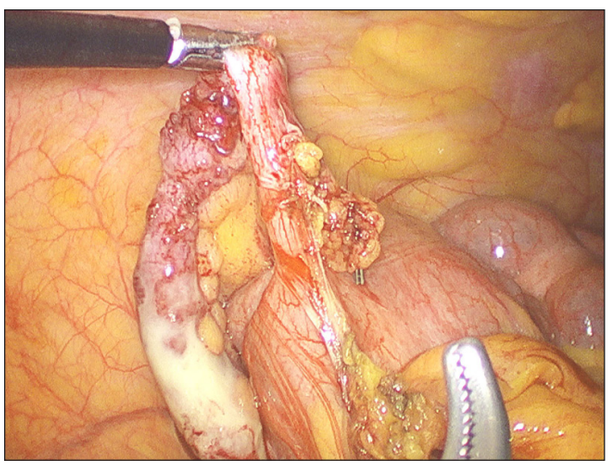


Figure 3; The mesoappendix divided showing the base of the appendix

APPENDICECTOMY: STEP BY STEP PICTURE GUIDE. LAPAROSCOPIC SURGERY.

Kulbir Mann

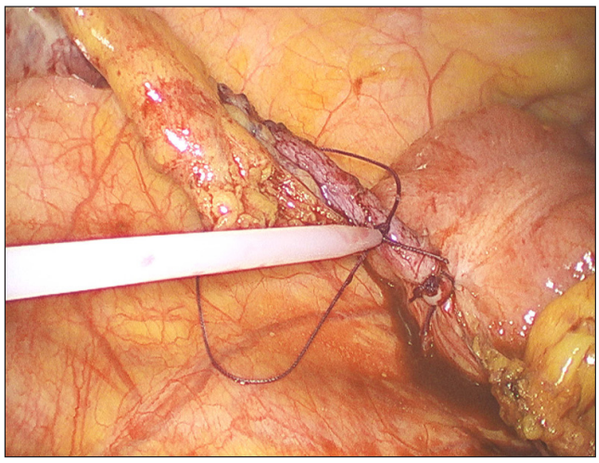


Figure 4; Application of the endoloop to the base of the appendix

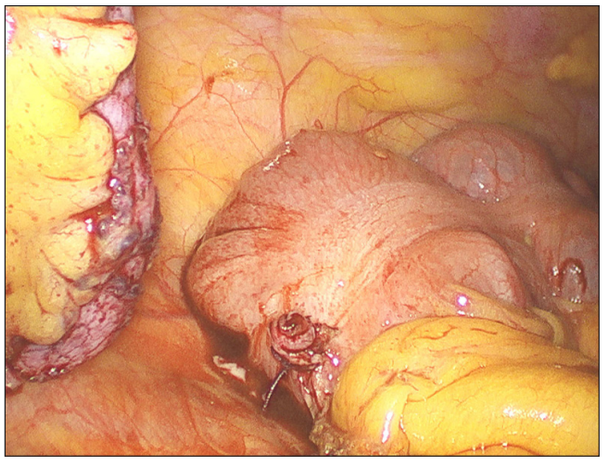


Figure 5; The resected appendix is seen to the left with the caecum to the right showing the sutured base.

The laparoscopic appendicectomy employs the fundamental basics of creating a pneumoperitoneum. Current recommendations state that it should be performed with an open technique using an open Hassan technique as opposed to the closed Veress needle approach. A small transverse or vertical infra-umbilical incision is made, for the 10mm port, and dissected to the linea alba. This is done with dissection scissors and the umbilical stalk and cicatrix can be the landmarks. The posterior rectus sheath layer is elevated and a 1-2cm incision is made through the peritoneum into the abdominal cavity. It is here a blunt trocar is placed and the cannula removed. The gas maximum pressure should be set to 12-15mmHg and the gas flow rate to 2-3L/min. The intra-abdominal pressure will be measured and should be slowly increasing. The flow rate can be increased when we are confident we have entered the abdominal cavity. One the pressure reaches the set level the laparoscope can be inserted. The scope should have a white balance carried out and be focused appropriately. [3]

The abdominal cavity is then thoroughly assessed by looking at all the viscera and looking for the appendix. This is limited without any further instrumentation and the 5mm accessory ports need placing. Local anaesthetic is administered perpendicular to the skin, into the abdominal cavity and visualising the needle tip with the laparoscope. With this method it is possible to visualise the inferior epigastric vessels and avoid them. A small transverse skin incision is made and the cannula inserted perpendicularly to the skin. A twisting motion should be used and the sharp end of the trocar visualised as it enters the cavity. Care should be taken to ensure there is no iatrogenic damage to viscera. The port positions for an appendicectomy are 1-2cm above the symphysis pubis and an optional left iliac fossa port. [3]

A non-traumatic grasper can be used to manipulate viscera to view hidden organs such as the ovaries. Manoeuvring the small bowel and the caecum should help to view the appendix. Gentle blunt dissection and diathermy can be used to free the appendix and visualise the mesoappendix. This needs to be ligated using hook diathermy or with a clip applicator. Once the base is freed an endoloop is used to place a suture around the base of the appendix and closed, ensuring a knot around the base. The endoloop is a very helpful suture applicator and requires experience to utilise properly. The appendix is pulled through the endoloop and the knot tied by placing the knot at the base of the appendix and tightening the suture. A further knot is placed distally and the appendix is resected between knots. The appendix is delivered using a Bert bag through one of the port sites. The abdominal cavity is then examined to ensure there is no residual bleeding. [3]

The ports are removed under direct visualisation. The central wound needs a direct closure of the rectus sheath to prevent port site hernias. This is done in varying ways but essentially two sutures are placed either side of the sheath using a J shaped needle and tied. The wounds are closed with subcuticular sutures. [3]

1.6.1 Tips

There are many cables and attachments involved in laparoscopy. It can become very entangled and fiddly, good organisation and position is required to ensure a comfortable surgical environment.

Dissecting down to the linea alba to insert the Hassan cannula is done in various ways by many surgeons. A helpful landmark is the umbilical stalk, which can be dissected, inverted and used as the insertion point. A small incision is made and clips placed either side to elevate the opening. This allows visualisation of the abdominal viscera, peritoneum or transversalis fascia, which will dictate whether further dissection is needed. The Hassan cannula can be inserted below the rectus sheath but not penetrating the peritoneum. Therefore inadvertent insufflation of the pre-peritoneal layer occurs and the cannula needs removing.

APPENDICECTOMY: STEP BY STEP PICTURE GUIDE. LAPAROSCOPIC SURGERY.

Kulbir Mann

Manipulating the laparoscope improves with experience. If there is poor focus or misting then cleaning and readjustment of the scope should be carried out. It is good practice to show the instruments entering the ports, moving towards the operating field to prevent visceral damage. A 0o laparoscope is simple to use and is pointed towards the operating field. The 30o scope can be used to visualise depths but requires practice to be able to look around structures by rotating the camera angle.

Port placement is essential to an efficient dissection because of the angles required to reach the RIF. Generally directing the port perpendicular or slightly angled towards the RIF is easiest as the instruments are naturally aimed towards the RIF. These may require re-insertion.

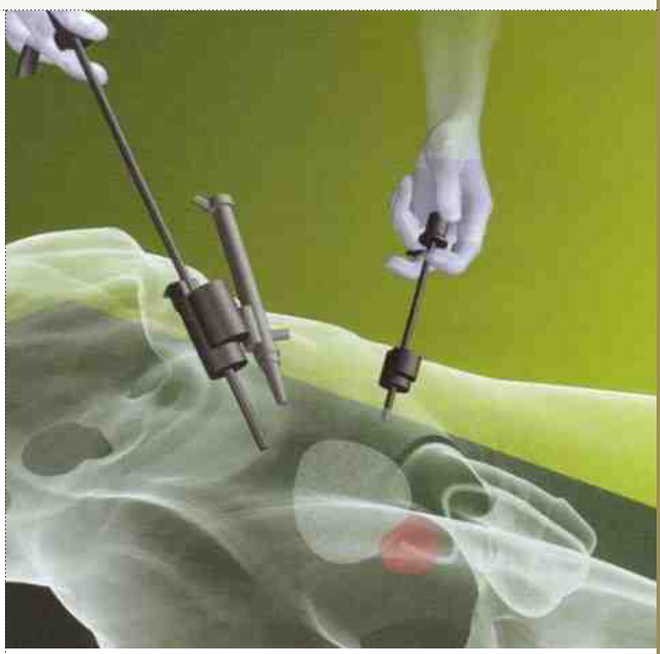
Bleeding is a common complication and there are many ways in which is it is controlled laparoscopically. The camera gives a zoomed view and bleeding can appear to be of greater severity than it actually is. However it still requires controlling and initial measures include pressure using ribbon gauze, hook diathermy and diathermy attached to a grasper. It is rare that a suture is required to controlled bleeding in laparoscopy.

The appendix may be fixed or lying retrocaecally and be difficult to visualise or mobilise. Camera ports can be changed and a 10mm size and the camera placed suprapubically. Dissection may be technically impossible, especially if planes are difficult to delineate. This is likely to cause bleeding which may prove difficult to control. The decision to open is a difficult one. It is important that as trainees gain in experience they are aware of their limitations. It is important to ask for senior assistance if there is too much difficulty. [3]

The appendix may be normal. Laparoscopy is the best diagnostic tool to investigate the cause of the clinical picture. If there is no other cause found then the appendix should be removed to rule out early appendicitis and to eliminate appendicitis as a cause of recurrent symptoms. The small bowel should be handled with graspers and “walked” to two feet to rule out a Meckel’s diverticulum.

1.7 The Operation note

The classical operation note consisting of incision, findings and procedure, summarise well the salient points that need recording. The operation note need to inform medical professionals what was found and what was done in order to manage the patient post operatively. The technique of pneumoperitoneum should be documented and a diagram of port site locations and their sizes. The findings are based around the presence of free fluid, the status of the appendix and how much dissection was required. Post procedure instructions are based upon feeding the patient, estimated discharge time, use of antibiotics, and follow up.



1.8 Post Operative Management and Complications

A patient with an inflamed appendix with little dissection and easy resection can be allowed to eat and drink as tolerated. Antibiotics are often given intravenously for three postoperative doses. Mobilisation should be encouraged and once the pain is controlled the patient is safe for discharge.

Should a patient have a perforation and a contaminated peritoneum, recovery will be slower. Patients should begin on clear fluids and be observed carefully for developing sepsis. Antibiotics are usually given intravenously for five days. There is little variation with laparoscopic appendicectomy management but they generally have better pain control and are discharged earlier.

Complications largely consist of bleeding, wound infections and collections. Bleeding is managed generally by immediate resuscitation and severity assessed with regards to re-operation, it may be occurring intra-peritoneally or superficially. Wound infections can range from cellulitis to deep infective collections. Collections may present with a delayed recovery, an ileus, general malaise or development of fever. These may be amenable to surgical or percutaneous drainage highlighting that imaging is key to its treatment. [3]

The degree of appendicitis and contamination of the peritoneum should dictate the level of care required post operatively to prevent sepsis and complications. Careful observation is essential.

APPENDICECTOMY: STEP BY STEP PICTURE GUIDE. LAPAROSCOPIC SURGERY.

Kulbir Mann

Questions MCQs

1. A 15 year old boy is admitted to A&E with RIF pain, fever, nausea, vomiting. What is his most likely abnormal investigation finding?
A) R sided gas shadows on AXR
B) Microscopic haematuria and nitrites on dipstick
C) Leucocytosis
D) Anaemia

2. An 80 yr old lady is admitted with right sided abdominal pain with marked tenderness on examination with raised haematological inflammatory markers. What is her least likely differential?
A) Appendiceal Mass
B) Diverticulitis
C) Pyelonephritis
D) Appendicitis

3. The appendix is least likely to be located where?
A) Retrocaecal
B) Pelvic
C) Postileal
D) Retroileal

4. Most Common malignancy of appendix is?
A) Mixed Cellularity
B) Adenocarcinoma
C) Squamous Cell carcinoma
D) Carcinoid Tumor

5. Which is the least likely pathogenesis statement about appendicitis?
A) Appendicitis results from obstruction of the lumen of appendix.
B) Gangrenous appendicitis results from arterial and venous obstruction
C) Radiation of pain from the umbilical region to the RIF shifts once luminal obstruction occurs and infection sets in.
D) Perforation of appendix increases the risk of death.

Answers

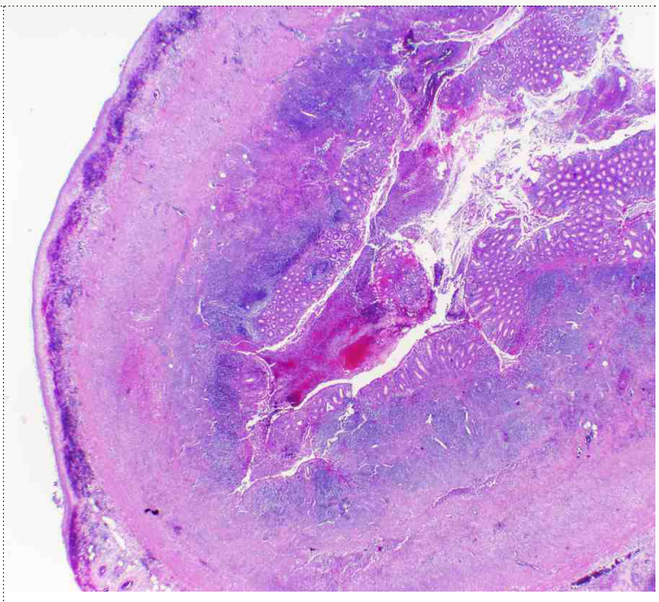
Question 1. C. Leucocytosis, the others are not indicative of appendicitis

Question 2. D. Appendicitis, elderly patients are more likely to present with a mass or other pathology. Though appendicitis is still found in the elderly

Question 3. D. Retroileal appendix is seen very rarely. Most common is retrocaecal

Question 4. D. Carcinoid is the most common tumour of the appendix, thought adenocarcinoma is believed to be approaching similar levels.

Question 5. C. Radiation generally occurs with peritoneal irritation.



Viva questions

1. What clinical features would you expect and aim to elicit in a patient with appendicitis?

2. What incision would you make for an open appendicectomy and what layers would you expect to encounter?

3. How would you induce a pneumoperitoneum for laparoscopy?

4. How would you proceed in a laparoscopic appendicectomy?

5. Compare the indications, advantages and disadvantages of the open and laparoscopic procedure.

6. In the situation of a macroscopically normal looking appendix, how would you proceed?

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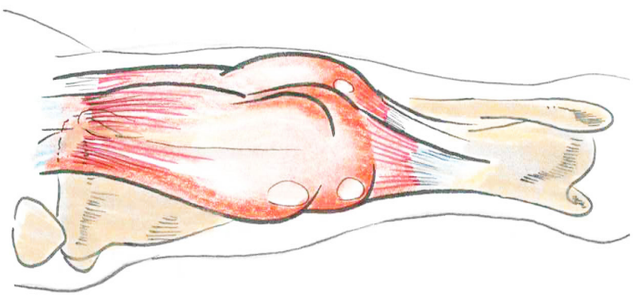
ACUTE COMPARTMENT SYNDROME

Conal Quah, Paul Dearden, V-Liem Soon and Giresh Swamy

Acute compartment syndrome of the lower limb.
Surgical Specialties: General Surgery* Trauma & Orthopaedic Surgery.



Abstract

Acute compartment syndrome is an acute limb threatening emergency, which occurs most commonly following fractures of the lower limb. The key to diagnosis relies on the surgeon having a high index of suspicion. Measurements of intracompartmental pressure, although a useful adjunct in certain scenarios, should not be a substitute for accurate history and clinical examination. In our review, we outline the aetiology, pathophysiology, diagnosis and management of acute compartment syndrome of the lower limb.

Case Vignette

Mr Charles, a 24 year old gentleman, was admitted yesterday following an injury whilst playing football. He is normally fit and well. He sustained a Grade I open fracture of his right tibia and fibula and underwent intramedullary nailing of his tibia earlier today. He was first on the trauma list, but his surgery was slightly prolonged as it was a junior registrar who was the operating surgeon. He returned to the ward at about 2pm and had been reasonably comfortable since then.

You, as the orthopaedic ST1 on-call, have been asked to see him 6 hours later as the nurses are concerned with his level of pain. His pain is 'excruciating' and 'worse than the break itself'. He had been given 10mg of IV morphine half an hour ago and his pain is still the same. His observations show that he is tachycardic and hypertensive. On your examination, you find that his toes are warm, well perfused and movements are limited by pain in his leg. He screams when you passively flex and extend his hallux. You give him a further 10mg of IV morphine, split his dressings and elevate his leg to heart level. Unfortunately, all measures prove unsuccessful in relieving his pain.

Suspecting acute compartment syndrome, you quickly phone the on-call consultant, who is just about to leave for home. After reviewing the patient, he agrees with your diagnosis and arrangements are made for an urgent fasciotomy. Mr Charles undergoes four compartment fasciotomies using the two incision technique. Your consultant points out in theatre that the compartments were tense, but no necrotic muscle was seen and, although grumpy he had to stay late, commends you for prompt diagnosis. The wounds are left open and following re-inspection 48 hours later, delayed primary closure is achieved.

Introduction

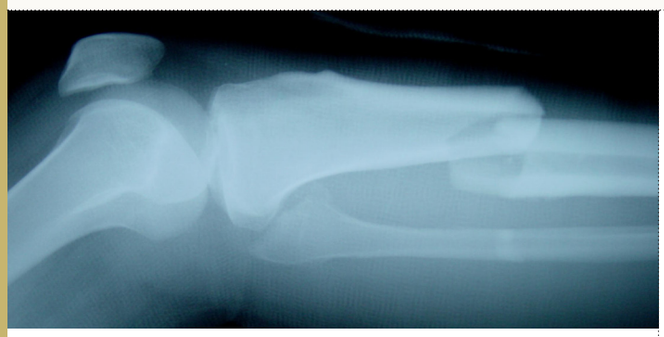
Acute compartment syndrome is a condition in which there is an increase pressure within a myofascial compartment leading to reduced capillary blood flow below the threshold for local tissue perfusion. Compartment syndrome was initially described by Malgaigne but it was Volkmann who first made reference to it back in 1881.[1] It has been described by Matsen as 'a condition in which increased pressure within a limited space compromises the circulation and function of the tissues within that space'.[2] Typically, limbs are more susceptible to this condition; however, it has also been described in the abdomen.[3] Acute compartment syndrome is a limb threatening surgical emergency, and if left untreated, becomes potentially life threatening. Other complications of compartment syndrome include ischaemic contractures, permanent neurological deficit, amputation and renal failure. The key to prevent any delay in diagnosis is to have a high index of suspicion.

Epidemiology

Fractures account for the majority of compartment syndromes observed. McQueen et al. reported in their series of 164 patients with acute compartment syndrome, the primary condition was a fracture in 69%.[4] The incidence after closed tibial fractures ranges from one to 29%, and for open fractures between one and ten percent.[5] Young men appear to have a higher incidence of compartment syndrome, and a possible explanation for this could be that they have relatively larger muscle volumes encased in tight compartments. Conversely, the elderly population have more hypotrophic muscles that have relatively more space to expand in their compartments. Another factor could be that young men are more likely to sustain high energy injuries. The incidence of compartment syndrome following reperfusion injury ranges between 0 and 21%.[5]

ACUTE COMPARTMENT SYNDROME

Conal Quah, Paul Dearden, V-Liem Soon and Giresh Swamy



Aetiology

Compartment syndrome can be either due to increased compartment content, reduced compartment size or increased capillary permeability (Table 1).

Increased Compartment Content	Reduced Compartment Size	Increased Capillary Permeability
Trauma: - Fractures - Crush injury	Iatrogenic: - Casts - Dressings - Pneumatic antishock garment	Reperfusion injury
Bleeding: - Vascular injury - Bleeding disorders - Anticoagulant Therapy	Localised external pressure: - Rubble - Prolonged limb compression	Trauma
Thrombosis	Excessive traction of fractures	Burns
Embolisation	Closure of fascial defects	Overuse of muscle
		Orthopaedic surgery

Table 1: Causes of acute compartment syndrome (adapted from Matsen[6] and Tollens[7])

Compartment syndrome occurs most commonly following trauma, causing fractures and soft tissue injuries. Fractures of the tibial shaft (36%) and of the forearm (18%) are the most common fractures causing acute compartment syndrome of a limb.[4] It was suggested more than thirty years ago, that compartment syndrome was not a complication of open fractures, with the theory that there is spontaneous decompression of the compartments at the time of the injury.[8] However, studies have shown that this is not the case.[9-11]

Soft tissue injury without any fracture is the second most common cause of compartment syndrome with an estimated 23% of cases attributed to it.[12] There is a greater risk if there is an associated coagulopathy. Both low and high energy injuries alike and prolonged limb compression secondary to altered consciousness from alcohol or drug abuse can result in compartment syndrome. Additionally, positioning of a patient in theatre can result in direct pressure on the limbs. The nonoperated leg that is elevated and positioned in a hemilithotomy position to provide adequate access for imaging, for example in intramedullary nailing procedures, increases the risk of developing acute compartment syndrome.

Pathophysiology

Adequate tissue perfusion is essential for cell viability. The pressure in the microcirculation determines the adequacy of nutrient blood flow in the capillaries which is opposed by the tissue and venous pressures. In acute compartment syndrome, the increase in pressure within the myofascial compartment will result in a reduced capillary blood flow below the threshold for local tissue perfusion. The circulatory compromise that results from the increased compartment pressures will eventually lead to muscle ischaemia and necrosis if compensatory mechanisms such as vasodilatation, development of collateral vessels and anaerobic metabolism are unable to meet the metabolic demands of the tissues. There are two complementary theories that have been proposed to explain the pathophysiology of acute compartment syndrome:

a) "Arterio-venous gradient theory" – The increase in venous pressure and/or an increase in capillary resistance will result in reduced capillary blood flow. When tissue perfusion pressure rises, the thin walled veins will collapse. Continued blood flow from the capillaries will re-establish the patency of the veins as the venous pressure exceeds the tissue pressure. This will eventually result in a reduced blood flow as the increased venous pressure will reduce the arterio-venous gradient and therefore no longer meet the metabolic demands of the tissue.[5, 13-15]

b) "Ischaemia-reperfusion syndrome" - Following decompression of a compartment syndrome or revascularization following an embolic event, the release of factors such as leucotrienes, tumour necrosis factors, free radicals and others mediators can cause vasodilatation and vascular leakage that will increase the compartment pressure. This will then result in the arterio-venous theory taking over and a vicious downward spiral.[5, 15-16]

Diagnosis

The prompt recognition of raised ICP is of upmost clinical importance before cellular perfusion is compromised. A thorough history and examination are paramount, but are assisted by the use of a range of pressure monitoring devices to increase diagnostic accuracy.

Certain clinical features can be recognised in the patient with rising intracompartmental pressure (ICP) secondary to any pathology: the so called *6 Ps:*

- **Pain out of proportion*** to the initial stimulus following adequate mild analgesia.
- **Pain on passive stretching*** of the muscle groups within the affected fascial compartment is perhaps the most useful and reproducible early sign of raised ICP. However, on occasion, it may be problematic to distinguish between superficial and deep pain.
- **Pressure** - Raised ICP may be appreciable at the bedside as tense, shiny overlying tissues associated with deep tissue oedema.
- **Paraesthesia** may be found on examination and is the result of cellular hypoperfusion injury to which nervous tissue is the most sensitive. If left unchecked, paraesthesia will progress to anaesthesia in the distribution of the affected nerve(s).

ACUTE COMPARTMENT SYNDROME

Conal Quah, Paul Dearden, V-Liem Soon and Giresh Swamy

• **Pulselessness** in the affected limb, after exclusion of arterial trauma, is a late sign resulting from the intracompartmental pressure rising to the level of the mean arterial pressure and compromising/occluding the flow in main axial arteries.
• **Paralysis** may also be a late sign, although it may be due to pain on muscle contraction. Reduced neuronal function and muscle cell death and cannot be attributed to raised ICP unless associated nerve injury has been excluded.

**Critical clinical signs*

Clinical diagnosis of compartment syndrome requires a cooperative, alert patient who is able to comprehend and carry out simple instructions. It is, therefore, more difficult in sedated or disorientated patients, in children or in those with regional anaesthesia present. For this reason, direct and indirect methods of compartment pressure measurement have been developed to provide objective, quantitative evidence to guide treatment.

Critical Pressure

Pressure of tissues within a closed myofascial compartment is normally 0-10mmHg. The ICP must be correlated to the patient's diastolic blood pressure when making the diagnosis of acute compartment syndrome. It can be recommended that fasciotomy is indicated with ICPs of 30-50mmHg. A lower cut off of 30mmHg has been commonly used as, above this level, fascial compliance grossly decreases. At pressures over 30mmHg capillary perfusion is also insufficient to maintain blood flow [5, 15].

Diastolic blood pressure minus the ICP is known as the delta pressure (Δp) is clinically used together with clinical signs/symptoms in diagnosing compartment syndrome. Critical Δp's have been demonstrated at 10-35mmHg [4, 6]. Commonly, a Δp of 30mmHg is used to avoid unnecessary fasciotomy in patients with subclinical raised ICP as no clinical consequences are seen in Δp's greater than 30mmHg [4].

Transient increases in ICP or Δp are not necessarily diagnostic of impending tissue necrosis secondary to compartment syndrome. Short increases in ICP can be seen during tibial intramedullary nailing but these are not always associated with signs of compartment syndrome [4].The degree of muscle damage is related to the time duration at which a maximal ICP is maintained. If left unchecked for more than 12 hours, permanent muscle necrosis and residual disability may occur. If surgical decompression is undertaken within 6 hours a full functional recovery can be expected.

Non-invasive Investigations

The use of pulse oximetry in compartment syndrome assessment is highly inaccurate and misleading and should never be relied upon in the setting of raised ICP as distal skin perfusion and oxygenation can be normal in a limb distal to an affected compartment[17]. Ultrasonography, CT and MRI are capable of identifying structural change within a compartment but as yet are not accurately correlated to intracompartmental pressure values[18-20].

Also, the availability of scanning facilities in many units limits rapid access to these investigations, making their use as a diagnostic tool to identify candidates for fasciotomy impractical. Near-infrared spectroscopy (NIRS) has been used to demonstrate change in tissue oxygen saturations but requires baseline values for clinical use. It is also highly position-sensitive and can be inaccurate when assessing deeper tissues. Consequently, NIRS is not at present a clinically useful[21]. Early alteration in neuronal function may be evaluated using Electromyography (EMG), somatosensory evoked potentials and vibration sense testing[22-24].

Although these methods appear highly sensitive in picking up neuronal compromise secondary to raised ICP, to date they have not gained widespread clinical support. At present, there is no non-invasive technique that is readily available which provides comparable diagnostic accuracy to invasive methods. Future developments in non-invasive diagnostic techniques in ICP monitoring may include mechanical impedance and measurement of surface hardness correlated to compartment pressure but both require further clinical assessment[25-26].

Invasive Investigations

Traditional methods of ICP measurement involve the use of basic equipment readily available in most medical units. Simple or side-ported needles, a length of plastic tubing containing saline and air attached to a mercury manometer can be assembled easily to provide direct measurement of pressure. This method however, involves injection of saline into the affected compartment, which may well increase the pressure, and the resulting cellular injury. Also, simple needle techniques tend to give consistently higher readings than other methods, possibly leading to increased false positive diagnoses, and side ported needles cannot be used for continuous monitoring[27].More accurate than needle pressure measurement systems are the 'wick' and 'slit' catheter models[28]. Both wick and slit catheters involve the use of a length of saline-filled polyethylene tubing connected to a pressure transducer, allowing continuous pressure monitoring. The limitation of wick and slit catheter systems is that the tip of the catheter can become blocked with blood leading to inaccurate measurements. Solid-state transducer intracompartment catheter (STIC) systems have been developed to overcome the potential draw backs of other invasive techniques[29].

They involve a multi-perforated polyethylene tip over the STIC connected to a pressurized constant infusion system and can be used for monitoring over periods up to 16 hours. Transducer-tipped fibre optic systems are expensive and require the insertion of a 2.1mm sheath into the affected compartment. They do, however, provide distinct advantages over the previously described invasive techniques as they do not involve injection of fluid and hence are free from hydrostatic pressure artefacts[30].

ACUTE COMPARTMENT SYNDROME

Conal Quah, Paul Dearden, V-Liem Soon and Giresh Swamy

Management

The only way to treat established acute compartment syndrome is by surgical decompression. However, restoring circulating blood volume and oxygenation can help improve outcome. Once the diagnosis of compartment syndrome has been considered, there are a few first line measures that must be performed to reduce further cell hypoxia which can avoid unnecessary surgery (table 2).

First Line Management For Suspected Acute Compartment Syndrome
Step 1: Remove circumferential constricting bandage or cast
Step 2: Avoid hypotension
Step 3: Limb elevation at the level of the heart
Step 4: Supplementary oxygenation
Step 5: Inform Seniors!

Table 2: The step by step first line management for suspected acute compartment syndrome

Firstly, removing any circumferential constricting bandages or casts will help reduce external compression and allow for some increase in compartment size. Avoiding hypotension is also important as this will reduce perfusion pressure which will in turn facilitate tissue ischaemia. The limb should be elevated at heart height as further elevation can reduce the arterio-venous pressure gradient that will result in decreased capillary blood flow. Finally, supplementary oxygen therapy must be prescribed to optimize tissue saturation[13, 14, 31].

Fasciotomy decompressing all four compartments is the only established and reliable treatment for acute compartment syndrome[12,32]. The two incision technique is the most commonly used technique in clinical practice and has been recommended by the British Orthopaedic Association as it provides optimal access to all four compartments[33]. The 15-20cm anterolateral longitudinal incision is placed between the fibula shaft and tibial crest. This allows access to the anterior and lateral compartments of the leg. The intramuscular septum is identified after undermining the skin by a short longitudinal incision over the muscle bellies (image 1).

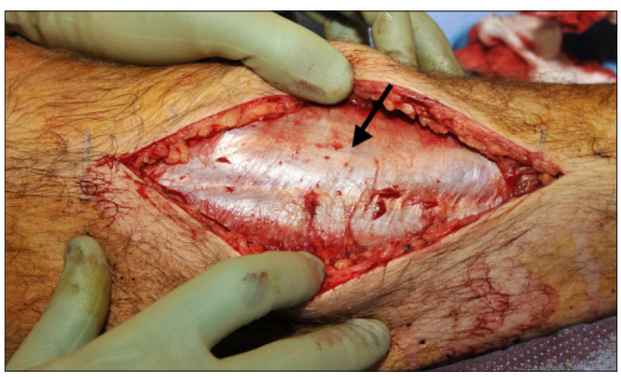


Image 1: The intramuscular septum is identified after incising the skin and subcutaneous tissue

The anterior compartment is decompressed by extending the first incision in the fascia throughout the leg. Access to the lateral compartment is gained by following the undersurface of the anterior compartment laterally to find the intermuscular septum of the lateral compartment which is then incised[5, 31, 34] (image 2).

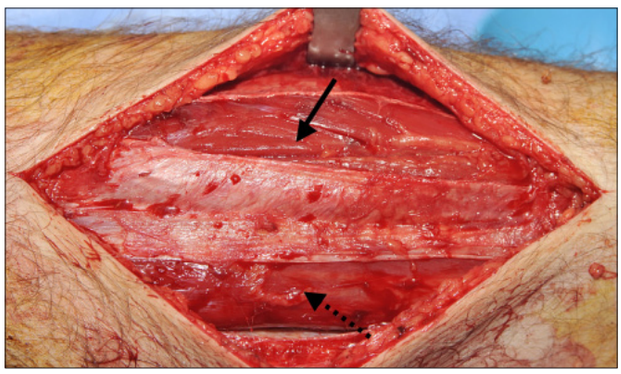


Image 2: Decompressed anterior (straight arrow) and lateral compartments (dashed arrow)

The superficial peroneal nerve must be protected along its course especially adjacent to the septum where it crosses the junction of the middle and distal thirds of the leg. The 15-20cm posteromedial incision is made 2cm posterior to the medial subcutaneous border of the tibia (image 3).

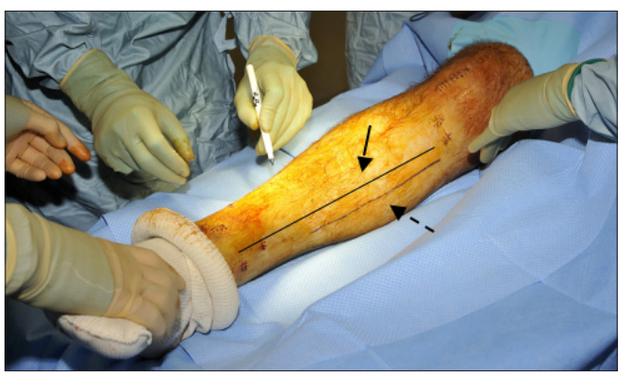


Image 3: The 15-20cm posteromedial incision (dashed arrow) is made 2 cm posterior to the medial subcutaneous border of the tibia (straight arrow).

ACUTE COMPARTMENT SYNDROME

Conal Quah, Paul Dearden, V-Liem Soon and Giresh Swamy

The fascia of the superficial posterior compartment is incised first longitudinally, exposing the gastrocnemius and soleus muscle (image 4).

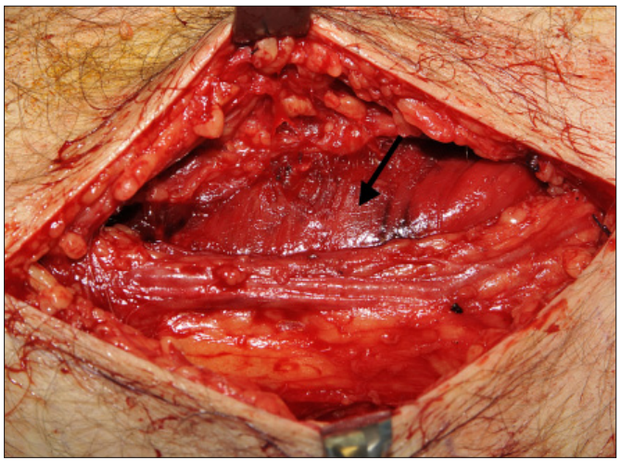


Image 4: Decompressed posterior compartments

The saphaenous nerve and vessel should be retracted anteriorly to avoid damage. The gastrocnemius is then retracted posteriorly from the tibia to expose the fascia of the deep posterior compartment which is then incised.

The other technique that has been used is a single lateral incision with or without fibulectomy. The skin incision is made directly over the fibula which allows easy access to the anterior and lateral compartments. Retracting the peroneal muscles anteriorly will allow exposure to the posterior intermuscular septum which is then incised to decompress the superficial posterior compartment. The deep posterior compartment is then released directly behind the fibula after dividing the origins of soleus from the fibula[5, 34]. Fibulectomy to expose all four compartments is not encouraged as it not only risks damage to the common peroneal nerve but also removes potentially stabilizing influence of the fibula on the tibial fracture[34]. Although effective, the advantage of the double incision technique is that it is faster as the incisions are all superficial and relatively safer than the single incision technique[35]. It can also be performed under local anaesthesia even at the bedside, however, the need for two separate incisions remains a disadvantage.

The muscle viability should be assessed based on the 4 Cs: color, consistency, contractility and capacity to bleed. All dead muscle should be debrided and the wound left open with sterile dressings applied. A second inspection in theatre should be scheduled 24-48 hours later. The fasciotomy wounds should be closed only if the muscles appear to be healthy at the second inspection. Delayed closure can be attempted at this stage only if there is no tension on the skin edge. However, on most occasions due to skin retraction and oedema, the skin edges cannot be approximated and a split thickness skin graft is required. This leaves a thin, insensate, and often poor cosmetic result. The shoe lace technique has been used with increasing frequency to gradually tighten the skin edges and achieve cover in 10 days. The sialastic vessel loop is applied across the wound with staples in a shoelace fashion thereby preventing skin retraction[36] (image 5).

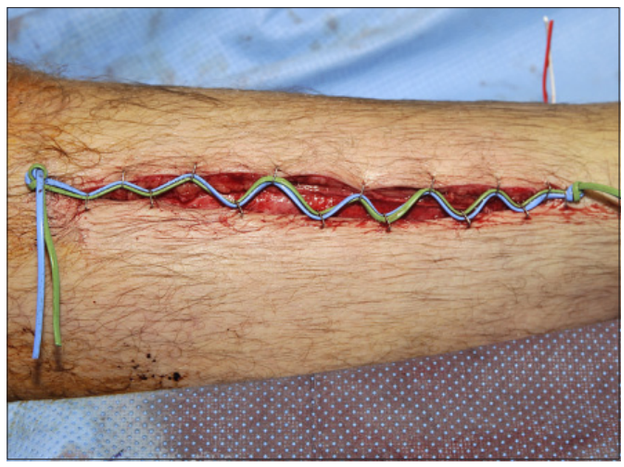


Image 5: Shoelace technique for closure to prevent skin retraction. Care on tension must be taken to ensure compartment syndrome is not recreated.

The Suture Tension Adjustment Real (STAR), is a mechanical method for gradual wound closure that is effective in 2-4 days. This technique involves placing the STAR units parallel to the wounds and then gradually tightened at the bedside[37].

ACUTE COMPARTMENT SYNDROME

Conal Quah, Paul Dearden, V-Liem Soon and Giresh Swamy

Conclusion

Early diagnosis and treatment of acute compartment syndrome is paramount to avoid devastating consequences. A high index of suspicion along with history and clinical examination in the conscious cooperative patient is often sufficient to make the diagnosis. Urgent fasciotomy is the only effective treatment for acute compartment syndrome.

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ACUTE COMPARTMENT SYNDROME

Conal Quah, Paul Dearden, V-Liem Soon and Giresh Swamy

Acute compartment syndrome of the lower limb.
Surgical Specialties: General Surgery* Trauma & Orthopaedic Surgery.

Questions

1. Acute compartment syndrome
a. Occurs only in closed fractures.
b. Can occur with no apparent trauma.
c. Less commonly occurs in young males.
d. Can often be diagnosed on clinical grounds.
e. Results from an imbalance between perfusion and intracompartmental pressures.

2. When managing a patient with suspected acute compartment syndrome, one should
a. Provide supplementary oxygen.
b. Remove any circumferential dressings or plaster.
c. Contact the anaesthetist to perform a nerve block for analgesia.
d. Elevate the limb to above heart level in an attempt to reduce swelling.
e. Inform a senior colleague at the next trauma meeting.

3. The diagnosis of acute compartment syndrome in the lower limb
a. Is associated with a rise in serum creatine kinase.
b. Is confirmed with a delta pressure greater than 30 mmHg.
c. Can be made using non-invasive investigations such as EMG.
d. Should be reconsidered when there is no paraesthesiae together with a warm foot.
e. Can be difficult in selected patient groups.

4. Regarding fasciotomies of the leg
a. The British Orthopaedic Association recommends the single incision technique.
b. Fibulectomy should always be performed to allow for further decompression.
c. Muscle viability should be assessed using the 4 C's.
d. The saphenous nerve is at risk when using the anterolateral approach.
e. May require input from the plastics team at a later stage.

5. All of the following statements regarding acute compartment syndrome are correct except
a. Acute compartment syndrome of the lower limb most commonly occurs following tibial diaphysis fractures.
b. Delayed primary closure of fasciotomy wounds should only be attempted after at least 48 hours.
c. The diastolic blood pressure is of greater importance than the systolic blood pressure.
d. A intracompartmental pressure of less than 30 mmHg is diagnostic.
e. A full functional recovery can be expected if fasciotomy undertaken within 6 hours.

Answers

Q1.	a. F	b. T	c. F	d. T	e. T
Q2.	a. T	b. T	c. F	d. F	e. F
Q3.	a. T	b. F	c. F	d. F	e. T
Q4.	a. F	b. F	c. T	d. F	e. T
Q5.	D				

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THE RECONSTRUCTIVE LADDER

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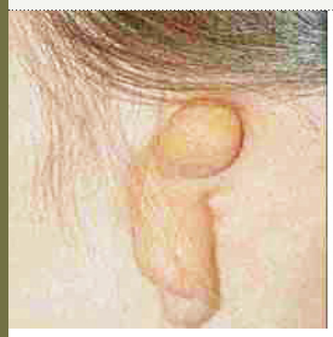
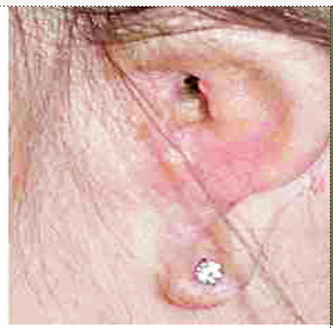




Plastic Surgery:
The Reconstructive Specialty.
Plastic and Reconstructive Surgery.

Plastic surgery is a specialised field, which does not deal with a specific organ site – it is a specialty based on the application of surgical principles throughout the body. The vast majority of workload lies in providing reconstructive input in collaboration with other specialties. For example, plastic surgeons work with orthopaedic surgeons for the management of open fractures, with dermatologists for skin cancer reconstruction, and with general surgeons and gynaecologists following cancer resection. All surgeons should have a basic understanding of the management of wounds. We need a systematic approach to determine the most appropriate option.

The Reconstructive Ladder

Traditionally, plastic surgeons have used the analogy of a ladder to organize the options available for wound management, with each reconstruction type forming a "rung". With each step up the ladder, the reconstructive option becomes more complex. It was traditionally considered desirable to perform the simplest reconstruction possible to achieve a good result. The rungs of the ladder described in popular plastic surgery textbooks (1, 2) include:

1. Secondary intention

No attempt is made to close the wound. Healing occurs by epithelialisation from the edges and wound contraction, eventually achieving closure. This relies on a healthy wound bed, with meticulous review and adjustment of dressings as required. Consequently, it may be very labour-intensive, and is not simply "the easy option". It is useful for example, in elderly patients and those with significant comorbidities.

2. Direct closure

The edges are brought together surgically by means of sutures, staples, glue, steristrips, etc. The wound heals by primary intention, with the eventual optimal outcome for this type of closure being a thin line scar.

3. Split thickness and Full thickness skin graft

Grafts

A graft is a piece of tissue that is transferred from one site to another without its own blood supply. In contrast, a flap carries its own blood supply. Different tissues can be used as grafts including skin, bone, nerve, and tendon. We will focus on skin grafting as this is a common wound management option.

A piece of skin is harvested from one part of the body and used to cover another, recipient, site. Skin has epidermis and dermis. Skin grafts always include epidermis, but also a varying amount dermis. If the skin graft contains epidermis and all of the dermis it is called a "Full thickness" skin graft. If it only has part of the dermis with the epidermis, it is called a "Split thickness" skin graft. For full thickness grafts, the donor site is typically down to fat and has to be closed directly, whereas for split thickness grafts, the donor site is like a graze, and is left to heal. Therefore, the amount of full thickness graft that can be taken is limited by the defect that will be created, but split thickness graft can be harvested form most body sites.

As skin grafts do not possess their own blood supply, they must undergo a process of "take" to heal in the recipient site. It is important that they are applied onto a graftable surface to facilitate this. Such surfaces include fat, muscle, granulation tissue, paratenon, perichondrium, periosteum and medullary bone. Exposed tendon, cartilage and cortical bone are generally not graftable surfaces. An unsuitable surface or the presence of infection or haematoma under the skin graft may increase the risk of graft loss.

Split skin grafts may be meshed, creating a lattice-shape. This allows exudate to pass through the mesh, may improve how the graft contouring to the bed, and may allow a graft to be used to cover a larger surface area.

As full thickness grafts have more dermis requiring revascularisation, they may be at higher risk of not taking. However, they undergo less contracture compared to split skin grafts, which thus may have more scarring issues in the longer term.

4. Local flap

Flaps

As discussed above, a flap carries its own blood supply. They can be classified on the basis of how near they are to the wound, as we will discuss here, but also by how they are moved, and what tissue they are composed of. Flaps may be moved via transposition, rotation, advancement, or as free flaps. The flap may constitute skin and subcutaneous tissue, fascia, muscle or bone, or combinations of these e.g. fasciocutaneous flaps, myocutaneous flaps, osteomyocutaneous flaps, etc.

THE RECONSTRUCTIVE LADDER

Jeremy Rodrigues and Reena Agarwal

In a local flap, the tissue is moved from an area adjacent to the wound to fill it. A "secondary defect" may arise where the tissue has been moved from, which may be managed with direct closure or skin grafting.

5. Regional flap

The flap is moved from another area of the body to cover a wound, but without detaching it from its blood supply. An example is moving latissimus dorsi from the back through the axilla to the anterior chest wall to reconstruct a breast after mastectomy.

6. Free flap

Here the flap, with its blood supply, is completely detached, moved, and plumbed in to a recipient blood vessel in or near the wound, for example latissimus dorsi muscle may be taken with its blood supply (thoracodorsal artery and vein) to cover open tibial fractures by anastamosing thee vessels to vessels in the lower leg, e.g. the posterior tibial vessels.

Other Options

In addition to the traditional options, there are other reconstructive possibilities that surgical trainees may come across:

Vacuum therapy

In recent years, vacuum therapy has become a popular adjunct to reconstruction. It may be used to achieve wound closure, or to convert a non-graftable wound to a graftable one. It involves a collapsible dressing on the wound attached via tubing to a suction machine, with a dressing applied over the top to achieve a seal. Negative pressure has beneficial effects on the wound that stimulate the development of granulation tissue. This may be used until the wound heals to a point where other dressings can be used, or may be used for a period and then the wound can be reconstructed with either a skin graft or a flap.

Tissue Expansion

Here, an implant consisting of a bladder and a port are inserted under tissue and gradually inflated at intervals. This is often used to increase skin area, but can used under other tissues too. The process increases the area of tissue available for reconstruction. Expansion is performed at intervals to prevent ischaemia of the tissue occurring. It is used to provide flaps for reconstruction on the scalp in particular, where it is cosmetically important to reconstruct with hair-bearing skin. Tissue expansion is also used frequently in breast surgery.

Dermal Substitutes

There are a range of biosynthetic products that may be used in reconstructive surgery. These products are used to provide a framework for healing, into which native cells can migrate. Trainees in plastic surgery may come across different types. Common varieties in the UK include Integra® and Matriderm®. Integra® comprises two layers: a deep layer of collagen and glycosaminoglycan, and a temporary outer layer of silicone (3). The outer layer is removed and the healing wound bed then skin grafted at a later date.

In contrast, Matriderm® is a collagen elastic matrix which can be skin grafted onto at the same time as application (4).

Keratinocyte Culture and ReCell®

Services may exist to provide keratinocyte culture. Here, a biopsy of skin is taken from the patient and a solution of keratinocytes is grown in vitro, which can later be applied to wounds.

ReCell® is a system that converts a skin sample into a spray that may be applied to a larger area during the same procedure (5).

Fat transfer

Liposuction is a common procedure in plastic surgery. The fat obtained in such a way can be used to provide volume reconstruction, by injecting it elsewhere in the body. Many slants on this technique are described, but a commonly used one is that described by Coleman (6).

The process involves harvesting fat in a similar fashion to liposuction. The fat is then centrifuged, and the oily supernatant discarded (this is formed mainly from ruptured fat cells) and the precipitate then infiltrated in small aliquots where required (6). Fat transfer has various potential applications, and is sometimes used in breast surgery, though this is not without controversy and debate (8). Besides fat transfer, there are also a range of synthetic fillers available on the market.

Climb the ladder...or just take the elevator?

Whilst the reconstructive ladder may useful for the surgical trainee to use to organise their thoughts, in practice, the trainee may feel that decision-making in plastic surgery units seems to follow other approaches.

Besides the reconstructive ladder, other theories of how to approach reconstruction have been proposed. These include Gottlieb and Krieger's reconstructive elevator (7). Their concept is that the simplest option is not always optimal, and that sometimes it is preferable to "take the elevator" up to a more suitable level (8).

Alternatively, Mathes and Nahai suggested the reconstructive triangle in which flaps, microsurgery or skin expansion are used to achieve the best outcome (9).

The above options are the starting point for the trainee surgeon on which to build a logical approach to wound management. We will discuss particular options in more detail in the forthcoming issues.

THE RECONSTRUCTIVE LADDER

Jeremy Rodrigues and Reena Agarwal

Consolidation

Now we can consolidate what we have discussed by considering a simple example that may present to the Emergency Department and be seen by a junior trainee:

A sixty year old man presents with a traumatic wound of 2cm in diameter on the forehead. How would you assess this?

Assessment

- Size
- Site
- Depth/Base
- Aesthetic appearance

Options

Assuming that there is no underlying bony injury or haematoma, and that the base consists of soft tissue covering the skull without gross contamination, what are the reconstructive options to achieve healing?

1. Direct closure
This wound is too large to close directly.

2. Secondary intention
It could be left to heal with dressings. This gives acceptable scarring in the elderly, but may not give an optimal aesthetic result in a younger patient (scarring is often less severe in the elderly).

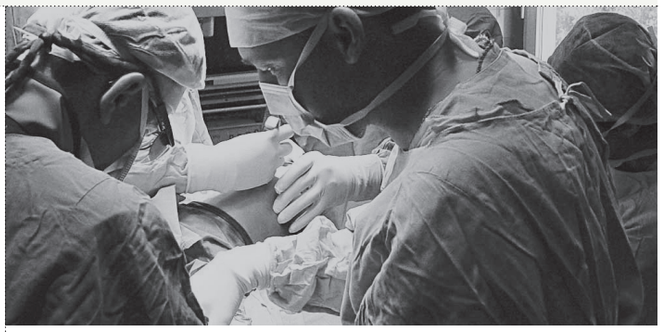
3. Split skin graft
This is possible, but again will not give a good cosmetic result. The graft is thin, and so may leave a "crater"-like appearance. As discussed above, it will contract over time as well.

4. Full thickness skin graft
This is like a "patch" of skin. This will give a better cosmetic result and is easy to perform. Full thickness graft can be harvested from sites in the head and neck area with skin laxity, such as in the supraclavicular fossa, or the postauricular area.

5. Local flap
This will rely on surrounding skin laxity, but may be an option.

6. Regional and free flaps
Not suitable options in this case. As we have discussed, the simplest suitable option is preferred, and closing a defect of this size does not require moving tissue in these ways.

In this simple summary case, we can see how a systematic approach of "climbing" the reconstructive ladder aids the choice of an appropriate reconstruction.



Test Yourself

1. Which of the following is not correct?
A. Split thickness skin grafts usually take on exposed tendon
B. Split thickness skin grafts do not usually take on exposed cartilage
C. Full thickness skin grafts usually take on exposed muscle
D. Meshed split thickness skin grafts usually take on exposed fat
E. Split thickness skin grafts usually take on exposed paratenon

2. You are inflating a skin tissue expander in the plastic surgery clinic. You inject 50ml of normal saline into the port, and the overlying skin being expanded becomes extremely tight and white, with no capillary refill. The patient complains that it feels very tight. You should:
A. Remove some of the volume of saline injected until colour returns in the skin, and discuss with your senior before continuing.
B. Inject another 100ml to ensure maximum expansion is achieved.
C. Book the patient for theatre to remove the expander and perform the reconstruction, as maximum expansion must have been achieved and the expansion is now complete.
D. Cover the expanded skin with a dressing to hide it until the capillary refill returns.
E. Discharge the patient with strong analgesia and a follow up appointment in two days time to reassess the skin.

For questions 4 and 5, select the most appropriate reconstruction from the following:

- Allow to heal by secondary intention
- Achieve primary closure by suturing the edges together
- Apply a meshed split thickness skin graft
- Apply a sheet split thickness skin graft
- Perform a free flap

3. In the following situation, is the final statement true or false?
You are raising a flap with your consultant. She clips and divides the artery and vein supplying the flap to move it elsewhere on the body to a distant recipient site where she plans to anastamose the vessels to ones in the recipient site. As the flap has been detached from the body's circulation, it has become a graft.

THE RECONSTRUCTIVE LADDER

Jeremy Rodrigues and Reena Agarwal

4. An otherwise fit 25 year old man has a motorbike accident. He has been managed overnight in A&E and his only injury is a Gustilo IIIB open tibial fracture. He is taken to theatre the following morning for a joint procedure between orthopaedics and plastics. You accompany your consultant plastic surgeon. The orthopaedic surgeons debride and wash the wound and perform an intramedullary nail for fixation.

You assess the wound with the consultant. There is a 15cm x 6cm defect. The wound is clean and adequately debrided. The edges will not appose, even under tension. In the base of the wound is exposed tibia, with periosteal stripping leaving several centimetres of exposed bone. The fracture site is exposed. Your consultant asks what reconstruction you think is most appropriate.

5. A 60 year man has been burned in a house fire. He has a 15% total body surface area full thickness burn mainly affecting his chest and abdomen. His lower limbs are spared. After treatment in ICU, he is taken to theatre and all of the burn debrided. The wounds are clean and bleeding. The base of the wound is fat.

Answer

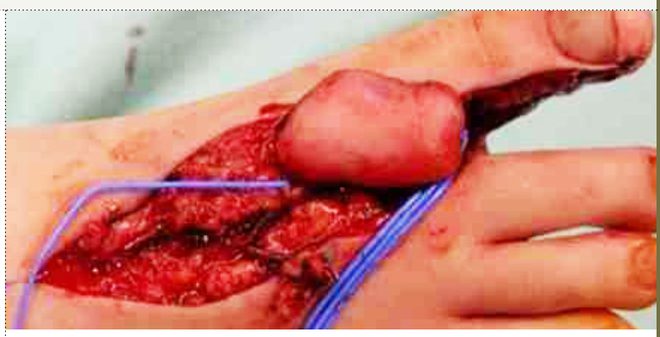
Question 1:
A (Skin grafts usually take on paratenon, but not on exposed tendon)

Question 2:
A. The skin has become ischaemic. Volume can be removed through the injection port. Junior trainees should seek senior input before re-expanding. Leaving the skin in this condition (D and E) leaves it at risk of necrosis. Injecting more saline will worsen the situation. The reconstruction is not necessarily complete. The expansion is not necessarily completed, more gradual expansion may still be possible, and may still be required for the reconstruction.

Question 3:
false. Although the vessels supplying the flap have been divided, the vessels within the flap are still intact, and will resume perfusing the flap when anastamosed at the recipient site. The pedicled flap has become a free flap, not a graft.

Question 4:
Perform a free flap. This wound should not be left to heal by secondary intention, and will not close primarily. Skin grafts will not take on exposed bone. Flap cover is required, either pedicled or free. A free flap is the only option in the list provided.

Question 5:
Apply a meshed split thickness skin graft. This area needs cover and will clearly not close directly. Fat will take skin grafts. A meshed skin graft will allow for expansion of the graft, whereas a sheet graft would require harvest from a very large donor site, and so is less appropriate to cover these bodily sites. A free flap is not necessary, and would have to be extremely large indeed if it were to cover this defect.



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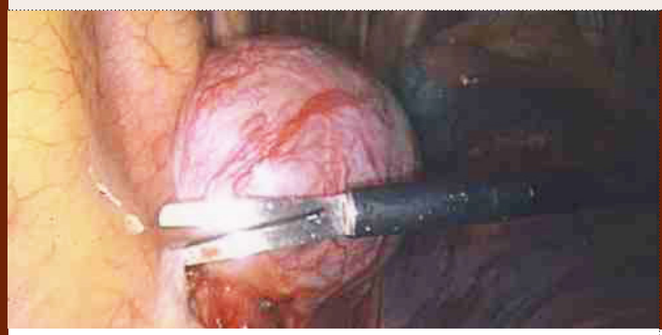
CERVICAL MEDIASTINOSCOPY – STEP BY STEP GUIDE

Ayo Meduoye and Laura Harrison



Video Assisted Cervical Mediastinoscopy.
Cardiothoracic Surgery.

History and pathology

Cervical mediastinoscopy is a surgical procedure most frequently used to obtain material for histopathological diagnosis of mediastinal masses and to stage bronchogenic carcinoma. It can be performed with minimal mortality and morbidity in the hands of experienced personnel, with mortality estimated at 0-0.5%, and morbidity at a frequency of 1-4% . Mediastinoscopy was first recommended by Carlens in 1959 as an invasive endoscopic technique to evaluate the mediastinum. Daycase procedure is routinely possible. Controversy still exists as to whether this procedure should be used routinely or selectively in staging a patient with bronchogenic carcinoma . It has been, and will continue to be, a useful technique in the evaluation of the mediastinum.

Indications and contraindications

Patients with resectable primary lung cancer are chosen for video-assisted mediastinoscopy when at least one of three clinical indicators is present: 1) CT/PET evidence of mediastinal lymphadenopathy, 2) elevated levels of serum tumor markers, or 3) diameters of primary cancers > 2 - 3 cm.

All patients being considered for radical resection of malignant pleural mesothelioma should preferentially undergo preoperative cervical mediastinoscopy, irrespective of radiological findings .

The procedure is usually immediately preceded by bronchoscopy, which may make mediastinoscopy unnecessary (ie, if there is evidence of tumour invasion in the endobronchial tree).

The presence of aneurysm of the aortic arch or innominate artery are considered contraindications.

Gaining informed consent/explaining procedure to patient

Patients should be informed that they will be given general anaesthesia so that they will be asleep and not feel any pain throughout the procedure. A tube (endotracheal tube) is then placed in the mouth to help breathing. An incision is made in the lower neck. A mediastinoscope is inserted through this incision into the chest. Tissue samples are taken from the lymph nodes surrounding the airways. The scope is then taken out. The opening is closed with stitches. There will be some tenderness at the site of the procedure afterward. Patients may have a sore throat after the procedure.

Possible complications are pain, bleeding, infection, perforation (oesophagus/ trachea), pneumothorax, lung injury, median sternotomy, left recurrent laryngeal nerve injury (hoarse voice), death 0-0.5%.

Equipment required

Mediastinoscopy tray including cupped mediastinoscopy forceps, Suction electrocautery, Biopsy forceps, Long dissector, Pledgets, Video mediastinoscope and stack, Rigid and flexible bronchoscope, Doughnut, Sandbag, Diathermy machine settings at 30 coag only, light source, median sternotomy tray. 20 ml syringe with saline to flush (+/- adrenaline for arresting bleeding), green needle. Surgicel® or other oxidised cellulose polymer haemostatic agent. Draping / sterile field preparation

Drape as median sternotomy

- 2-ply head towel
- Surgeon at head end
- Scrub nurse on patient's right
- Video monitor at feet end
- Top lights off once mediastinoscope is in place

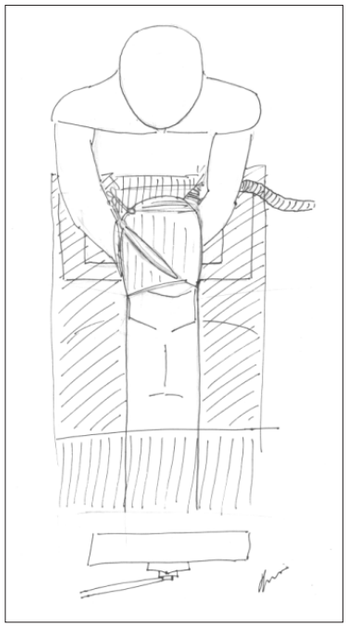


Figure 1

CERVICAL MEDIASTINOSCOPY – STEP BY STEP GUIDE

Ayo Meduoye and Laura Harrison

Patient positioning and relevant anaesthetic points

Supine, sandbag under shoulders, doughnut under head, neck extended, standard general anaesthesia without single-lung ventilation. Local anaesthesia approach is possible but not ideal . ET tube fixed to patient's left to avoid path of the mediastinoscope.

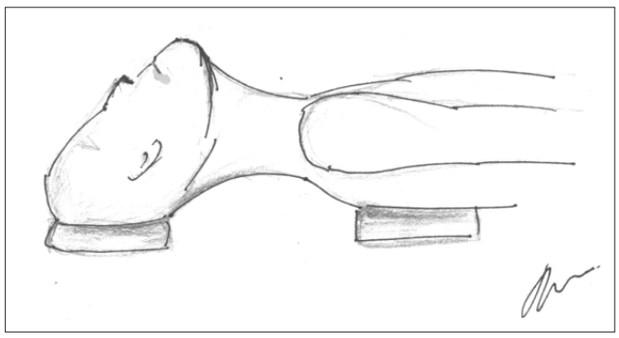


Figure 2

Procedure

Transverse 3cm incision (or gently 'smiling' incision) is made between the anterior borders of sternocleidomastoid, 1cm above the suprasternal notch.

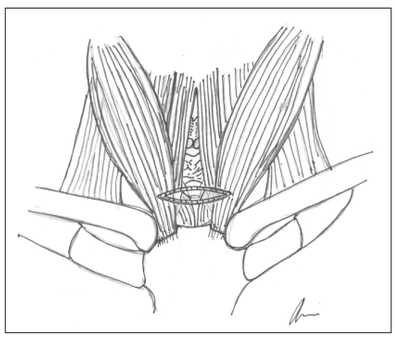


Figure 3

Dissection with electrocautery through platysma and down to strap muscles (sternohyoid and sternothyroid). Significant bleeding indicates deviation from the midline, re-orientate.

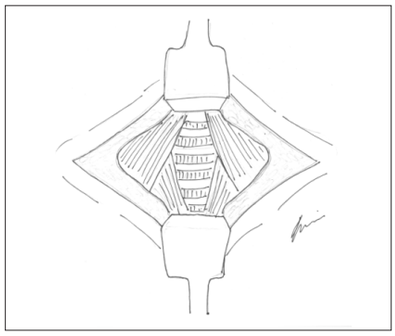


Figure 4

Retract strap muscles superiorly and with pressure applied in a posterior direction, exposing and defining the trachea in the midline. Elevate and open pretracheal fascia.

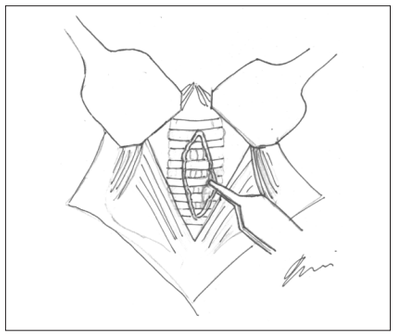


Figure 5

Blunt dissection with index finger to define plane. This step is critical. Trachea is palpable posteriorly, innominate artery is palpable anteriorly. Blunt dissection to left and right of trachea to break down pretracheal fascia.

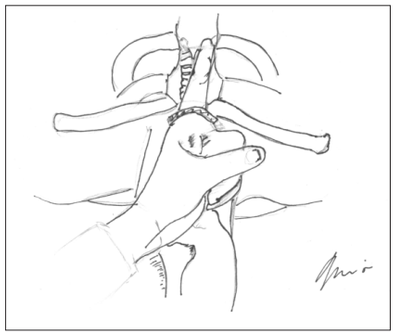
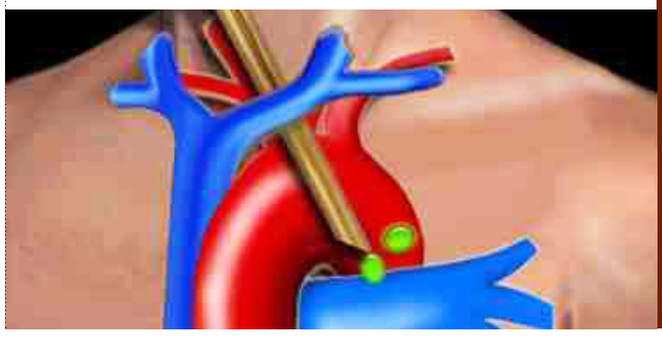


Figure 6

Mediastinoscope is now gently introduced below the pretracheal fascia. Dissection is now undertaken with suction (+/-electrocautery). Suspicious structures should be punctured with an aspiration needle before biopsy is undertaken to ensure that it is a lymph node and not a blood vessel. Electrocautery should be used sparingly (if at all) on the left side to avoid left recurrent laryngeal nerve damage. Biopsied lymph nodes are labelled by station and sent for cytology in separate pots. Lymph node anatomy is as follows;



CERVICAL MEDIASTINOSCOPY – STEP BY STEP GUIDE

Ayo Meduoye and Laura Harrison

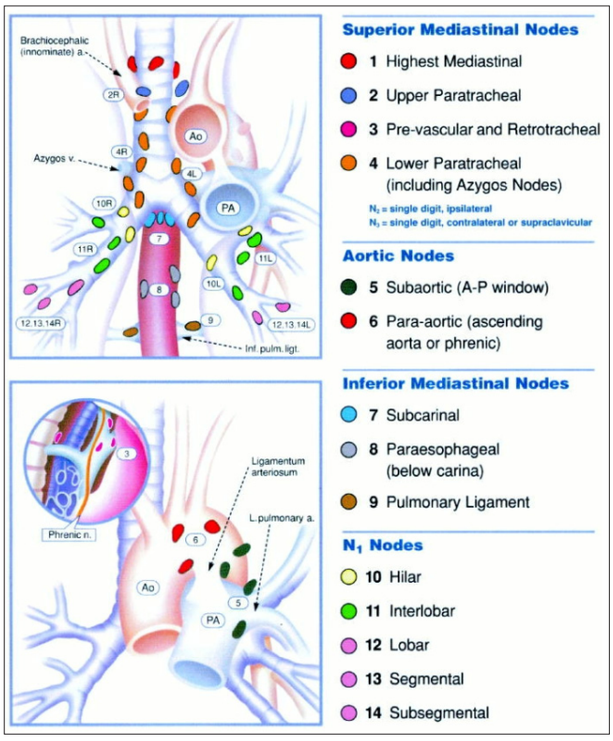
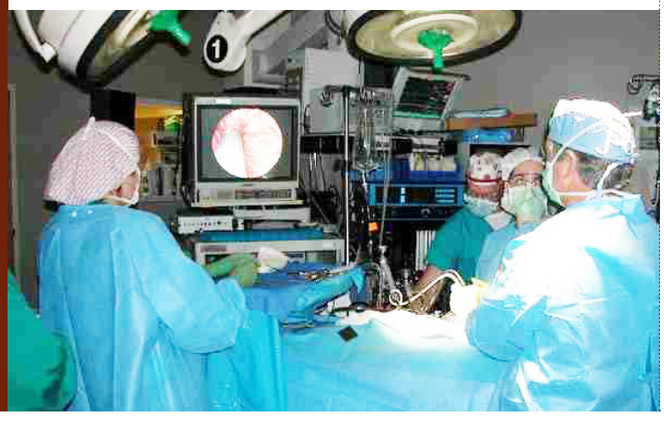


Figure 7

Diagram adapted with thanks from Cancer Medicine, 6th Edition, Holland & Frei et al.

If mediastinoscopy is being undertaken for lung cancer staging, most surgeons will begin sampling on the contralateral side to the lesion. Nodes are assessed by the naked eye and can be sent for frozen section (rapid intra-operative histopathological analysis). Positive contralateral nodes will lead to an N3 diagnosis, and such a patient is not a surgical candidate, regardless of the involvement of ipsilateral nodes.

Closure is performed in three layers and there should be no hesitation to close over a small gauge redivac drain if haemostasis is unsatisfactory.



Documentation of Procedure

- Date/Time
- Procedure
- Indication
- Surgeon/Anaesthetist
- Anaesthetic

I(Incision):

Midline, 3cm transverse incision 1cm above suprasternal notch, pretracheal fascia identified and opened, blunt dissection, mediastinoscope inserted.

F(Findings):

Macroscopically abnormal lymphoid tissue identified at stations eg; 2R, 4L etc.

P(Procedure):

Blunt dissection with suction electrocautery, Lymph nodes verified by aspiration needle puncture, biopsies taken and labelled by station, sent for frozen section/ formal histopathology.

C(Closure):

- Haemostasis
- Redivac® drain (if indicated)
- Pleural drain to underwater seal/Portex® bag (if indicated)
- Interrupted absorbable sutures to close platysma/subcutaneous tissue/dermis
- Continuous non-absorbable subcuticular suture to close skin (cosmesis is important esp. given location of incision)
- Anti-microbial preparation applied to wound (eg: Betadine®)
- Dressing

Managing complications

- Minor bleeding: electrocautery, local pressure, saline wash with adrenaline, Surgicel®, close over Redivac® drain.
- Major bleeding: pack, anterior thoracotomy or median sternotomy depending on source of bleeding and location of tumour, primary resection of tumour at this stage may be applicable.
- Tracheal rupture: Can occur in patients with mediastinal fibrosis. Defect is covered with oxidised adhesive.
- Pneumothorax/lung injury: closure with pleural drain connected to underwater seal either via cervical incision or via chest wall. Admission may be indicated.
- Local wound infection: Cellulitis should be managed aggressively, initially with intravenous antibiotics, as the risk of mediastinitis is significant.

Tumour seeding: has been reported in the path of the mediastinoscope .

CERVICAL MEDIASTINOSCOPY – STEP BY STEP GUIDE

Ayo Meduoye and Laura Harrison

Multiple Choice Questions

1) Aneurysm of this vessel is a contraindication to performing cervical mediastinoscopy;

a)thoracic aorta
b)aortic arch
c)pulmonary trunk
d)carotid artery

2) In patients with bronchogenic carcinoma, lymph node sampling should commence on which side, respective to the lesion;

a)ipsilateral
b)contralateral
c)either side, it's not relevant

3) During cervical mediastinoscopy , this nerve lies in close proximity to the dissection;

a)left recurrent laryngeal nerve
b)right recurrent laryngeal nerve
c)phrenic nerve
d)ansa cervicalis

4) In preparation for cervical mediastinoscopy, patients should be draped for;

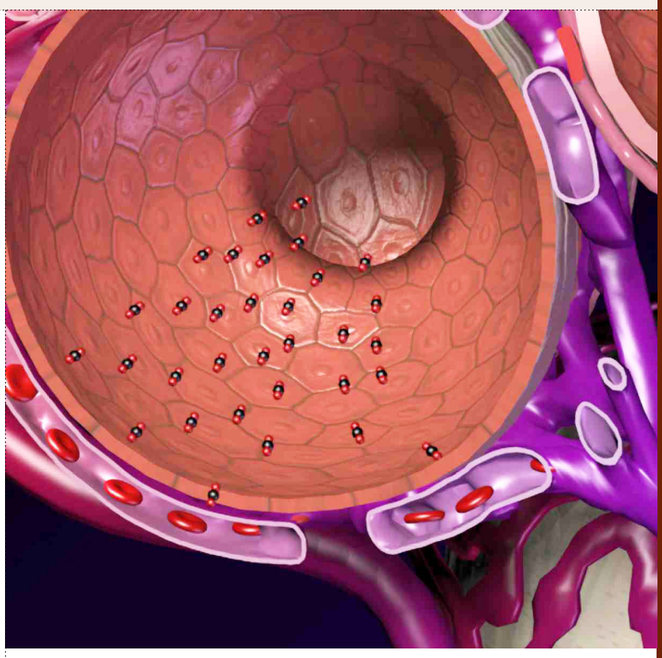
a)left thoracotomy
b)formal neck dissection
c)median sternotomy

5) Mortality rates for cervical mediastinoscopy are in the region of;

a)0-1%
b)1-2%
c)2.5-5%

MCQ answers

1) b **2)** b **3)** a **4)** c **5)** a



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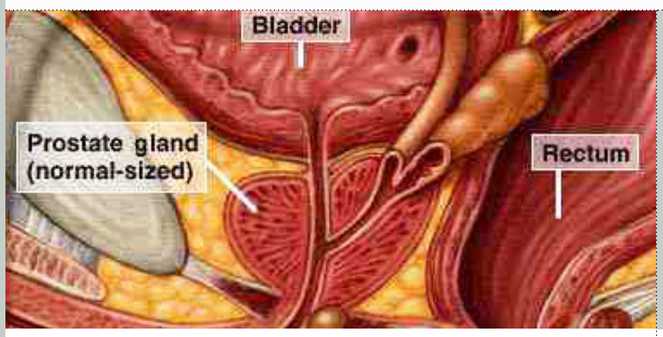
BOO (BLADDER OUTLET OBSTRUCTION)

Rohit Chaturvedi



Bladder Outlet Obstruction. Urology.

Case Vignette

A 57 year old gentleman presents to the outpatient department complaining of poor stream and nocturnal incontinence which has been deteriorating. On examination he has a painless large suprapubic mass extending to the umbilicus which is dull to percussion. A recent serum creatinine as performed by the general practitioner is within normal limits.

Case Explanation

The patient in the case study has low pressure chronic urinary retention, explaining the normal creatinine. The nocturnal incontinence is overflow incontinence. His management requires urethral catheterisation in the short term. This should be performed as an inpatient so to observe for a post-obstructive diuresis and decompression haematuria. A residual volume should be measured and hourly urine output. If a large volume (greater than 1 litre) is present a trial without catheter should not be performed and the patient may be left with a catheter in-situ or taught clean intermittent self catheterisation prior to evaluation with urodynamics.

Introduction

Bladder Outlet Obstruction (BOO) encompasses a number of aetiologies, with a large spectrum of presentations. The aetiologies of BOO in men and women are largely gender specific and considered by many two separate topics.

The term BOO refers to the relationship between detrusor pressure on voiding and urinary flow and therefore defined as high pressure, low flow. Therefore by definition BOO is a diagnosis of pressure / flow studies and it follows that this is the gold standard investigation, however, a diagnosis can be inferred from a free flow rate.

Patients with BOO are primarily treated based on symptoms rather than for prevention of future complications. BOO gradually induces numerous morphological and physiological changes in bladder and urinary tract. It leads to the hypertrophy of smooth muscle cells and development of connective tissue in-between, observed as trabeculae, which thickens the bladder wall (1). Such pathological consequences may result in a poorly compliant bladder and thus increases pressure within the bladder and kidney (2). If BOO is not corrected in time it may result in permanent damage to renal function, however, the vast majority of patients do not develop any complications.

Causes of BOO

The BOO spectrum may broadly be divided into anatomical and functional causes (see table 1).

Regardless of aetiology BOO results in resistance within the urinary tract at any location from the bladder neck to urethral meatus. This resulting resistance within the bladder renders its action unpredictable. Patients describe symptoms of an obstructive, irritative or mixed picture, but this is unlikely to relate to the aetiology or degree of obstruction. For instance, patients encountering disabling symptoms may have minimal objective findings, whereas complete decompensation of the lower urinary tract may be found in an asymptomatic patient (3).

Causes of BOO	
Iatrogenic	Incontinence procedures (i.e. Slings) Post pelvic surgery Post urethral dialatation
Anatomical	Pelvic prolapsed Retroverted uterus Ovarian cyst Urethral valves Meatal stenosis
Infective	Urethritis Stricture Cystitis
Neoplastic	Benign Prostatic Enlargement Urethral carcinoma Vaginal carcinoma Cervical arcinoma Prostate carcinoma Bladder carcinoma
Trauma	Insertion foreign body Stricture
Neurological	Spina Bifida Multiple Sclerosis Fowlers syndrome
Metabolic	Urethral stones Bladder neck stone

Table 1

BOO (BLADDER OUTLET OBSTRUCTION)

Rohit Chaturvedi

Patient Evaluation

The assessment of a patient presenting with signs or symptoms suggestive of BOO should be divided into subjective and objective, therefore should always start with a detailed history and examination. The prevalence of urinary symptoms increases with age (4), however symptoms are neither cause nor gender specific. It is for this reason the term prostatism was replaced with the more favourable Lower Urinary Tract Symptoms (LUTS) (5). This term does not infer a pathological cause and is non-specific to gender as symptoms previously described as prostatism were found to exist within populations of elderly women (6). LUTS are not specific to BOO and may be suggestive of numerous causes.

The symptoms encompassed by LUTS are split in two, that relating to filling symptoms and voiding symptoms. Filling symptoms are those of hesitancy, sensation of incomplete bladder emptying, poor stream, and post void dribbling. Voiding symptoms encompass urgency, frequency, dysuria, and nocturia.

In the evaluation of symptoms the best way to assess symptom severity is using a validated symptom score such as the International Prostate Symptom Score (IPSS). The IPSS score system is derived from the American Urological Association (AUA) 7 scoring system with the addition of a quality of life score. Using this tool the severity of symptoms may be assessed and changes in symptoms may be monitored over time or following an implementation in management.

In the history it is important to consider other causes of LUTS (e.g. pelvic surgery, cardiac conditions, neurological conditions, diabetes) and red flags such as haematuria will need to be investigated in their own right.

During patient examination the abdomen will need to be assessed for distension and a digital rectal examination (DRE) to assess the prostate in males and a per-vaginal (PV) examination in females may also be warranted. The role of DRE, for the prostate, is to assess for nodules and abnormalities in the contour, which may suggest malignant pathology as well as provide rough estimate of size. Surprisingly size is not an indication by which to treat a patient, however should operative intervention be considered, trans-rectal ultrasound (TRUS) evaluation would be required as prostates greater than 100g would more likely require an open procedure. The DRE is also an important tool in the assessment of anal tone, which may suggest sacral nerve pathology. Inspection of the external genitalia may reveal meatal stenosis, phimosis or prolapse of pelvic organs.

Unfortunately subjective assessment correlates poorly with objective and aetiological cause. In order to complete a full patient assessment, objective means are required which aim to quantify the degree of obstruction and determine pathological cause so that a patient specific management plan may be formed.

Investigations

Investigations should start with simple tests. A urine dip and mid-stream urine (MSU) provide invaluable information, e.g. picking up haematuria, glycosuria or urinary tract infections which may require a different path of investigation separate to BOO. Biochemical evaluation of renal function is important, a serum creatinine is not only useful in establishing a baseline but also in detection of renal failure due to high pressure urinary retention.

The use of PSA testing in men with symptoms suggestive of BOO is not routine and tends to be performed when indicated. Indications that may prompt this investigation include suspicion of prostate cancer based upon history and examination, plus the European Association of Urology (EAU) recommends the use of PSA testing where the diagnosis of prostate cancer will change the decisions made on therapeutic option (7). In either case PSA testing comes with a lot of responsibility and patients should be adequately counselled as various factors such as cancer, BPH, infection, trauma, and age, might influence serum PSA levels. Patients need to be aware that though sensitive for prostate cancer PSA testing is not specific.

A very inexpensive and effective tool in objective assessment of patients presenting with frequency and/or nocturia is the use of frequency-volume (FV) charts (8) (which may also be referred to urine diaries). They may be used to exclude nocturnal polyuria (9,10), which may be due to causes which require simple conservative measures. Polyuria secondary to excessive fluid intake is another diagnosis that can easily be picked up with the use of a fluid volume chart. Neither of the mentioned conditions may be treated surgically.

Radiological imaging is often not required unless there is suspicion of high pressure urinary retention resulting in renal dysfunction. The image modality of choice in this situation would be ultrasound of kidneys, ureters, and bladder (US KUB). Elevated serum creatinine and large post void residual volumes (PVRV) would raise suspicion. Koch et al assessed the outcome of 556 consecutive patients with BPH and found that 0.8 % of patients with a creatinine < 115 had upper tract dilatation whereas 33% of those with a creatinine > 130 showed evidence of upper tract dilatation (11). At the time of US KUB it is also possible to perform flow rate (FR) studies (Figs. 1 and 2) and measure PMRV however these investigations may occur in the outpatient department. FR studies are a useful tool in objectifying the initial subjective assessment of flow. When interpreting urinary FR one must always consider the volume voided as values which are too high or low are not representative of the true FR. Unlike urodynamic studies (UDS) a FR will not distinguish between BOO and a poorly contractile bladder and as such it has no role in the prediction of outcome following surgical intervention. PMRV should be taken following a flow rate or micturation as it may provide some use in identifying patients in whom conservative management of chronic retention may be an option.

BOO (BLADDER OUTLET OBSTRUCTION)

Rohit Chaturvedi

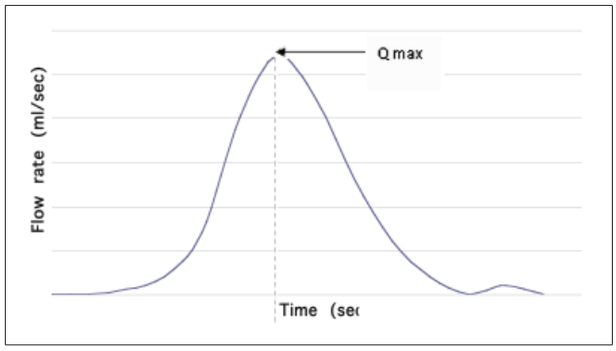


Fig. 1 - Normal Flow Rate Chart

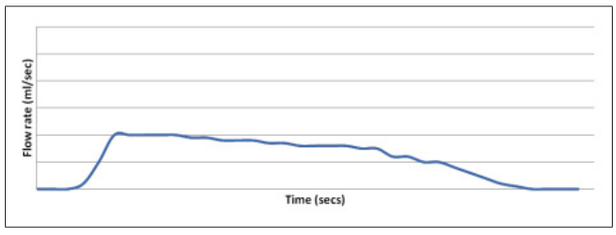


Fig. 2 - BPO

UDS remains the gold standard in the diagnosis of BOO, unsurprising when you consider BOO by definition is a urodynamical diagnosis, and distinguishing the various aetiologies giving rise to LUTS. It can distinguish the difference between BOO and a poorly contractile bladder. Such information can be invaluable in preoperative assessment of a patient as it allows for adequate counselling of patients on likely post operative outcomes so the patient may make a well informed choice.

Diagnostic endoscopy of the lower urinary tract provides visual assessment of the urethra from the meatus to the bladder. It currently provides the best assessment of urethral strictures identifying location and magnitude.

Following investigation any management plan formulated is a complex interplay between the subjective symptoms experienced by a patient and the objective findings from investigations. Patients who have little in the way of symptoms may not wish for intervention based on "numbers" and vice versa. For the purpose of this article we will briefly discuss the management of the most common causes of BOO.

BOO Manifestations

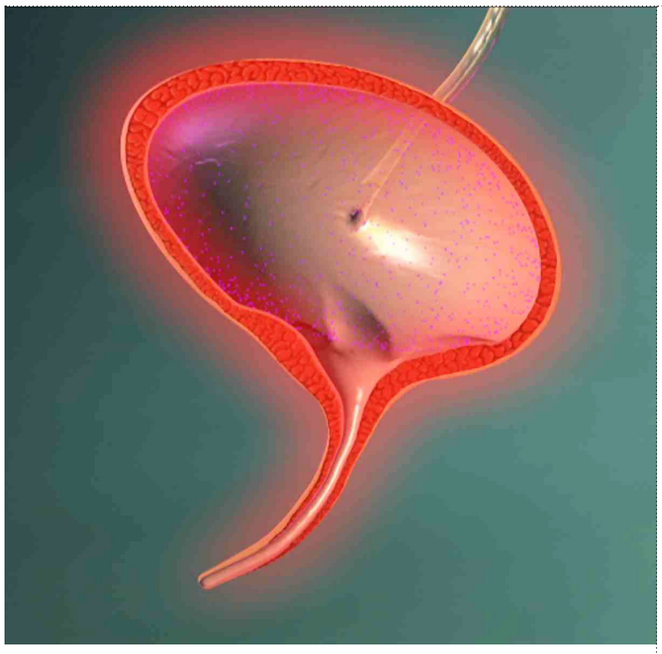
Junior members of the Urological or general surgical team, who have greater exposure to acute services than the outpatient department, will see BOO manifest itself as urinary retention.

Patients in acute urinary retention (AUR) classically present with severe abdominal pain and a history of failure to void urine despite a strong desire, with resolution upon catheterisation. It is not uncommon to hear the story of large intake of fluid and a car journey resulting in bladder overdistention secondary to delayed voiding thus impaired detrusor contractility (beware the FR test with large volumes passed). Such events are either precipitated (pAUR) or spontaneous (sAUR) and categorising AUR in these subsets is important in assessing those who will likely suffer a repeat episode. pAUR is less likely to recur once the precipitating event has been dealt with. Precipitating events may include anaesthesia, pelvic surgery, constipation, clot retention, urinary tract infection, or drugs (anticholinergics or sympathomimetic medications). sAUR however is much more likely to recur and diagnosis along with accompanying definitive management is required within this group. It is known for AUR to occur without pain in acute on chronic urinary retention, however think of neurological pathology in this group. Prompt assessment and examination including appropriate examination of sacral nerves is required.

Post-operative urinary retention is common and often multifactorial, it is not simply a case of pre-existing BOO being highlighted (13). Anaesthetic and its associated drugs may have local as well as central actions. Other factors following surgery may include pain, constipation, immobility, instrumentation to lower urinary tract, infection, and neuropraxia. In such scenarios catheterisation may be required in the short term and often normal voiding will resume following resolution of precipitating factors and a trial without catheter (TWOC), if not a period of clean intermittent self-catheterisation (CISC) may be required, especially in those who may have suffered neurological injury. If symptoms persist urological assessment would be indicated.

BOO (BLADDER OUTLET OBSTRUCTION)

Rohit Chaturvedi



Patients may incompletely empty their bladder. As they are managing to void spontaneously they remain unaware of the gradual distension of their bladder and develop chronic urinary retention. This may cause backpressure resulting in dilated upper urinary tracts and loss of renal function, high pressure chronic urinary retention (HPCUR). The first presentation may be due to the incidental finding of deterioration in renal function, overflow incontinence manifesting as bedwetting or acute on CUR. Upon catheterisation of these patients large volumes of urine will be drained (often greater than 1000ml). HPCUR is confirmed by imaging of the upper tracts however in a patient with a large residual volume (RV) and elevated serum creatinine HPCUR should be assumed and therefore these patients require admission for urine input : output monitoring due to the high risk of diuresis. This group of patients require cautious fluid management; over aggressive replacement of fluids will drive the diuresis and therefore the patient should be checked for postural hypotension. A TWOC is generally considered inappropriate in this group and if considered should involve the teaching of CISC until a long-term solution is available.

When asked to catheterise a patient always ask "is this appropriate?" (Table 2) If the patient is not in pain there is no need to rush in with a catheter. History and examination is key, if available perform a bladder scan. As a general rule if a patient is pain free, with a stable non-compromised renal function and free of infection they most likely do not need a catheter as they likely represent the common low pressure chronic urinary retention (LPCUR) group. Remember many patients attending the urology outpatient department will have high residual volumes and distended bladders but few will get admitted or catheterised acutely. Recurrent infections in LPCUR will require intervention to aid bladder emptying, in the short term this may involve CISC however in the population unable to do this a low dose prophylactic antibiotic may be considered. Further management of this group should be considered following urodynamics to assess bladder compliance. When in doubt call for help or advice.

Retention	Acute	Low P Chronic	High P Chronic	Post Op	Neurological
Pain	Y	N	N	Y/N	N
RV	<1L	<1L	<1L	<1L	<1L
Creat	N	N	H	N	N
Rx	TWOC/ drugs TURP	May consider ISC	ISC Watch for diuresis	TWOC / ISC	The cause
	Long term catheter				

Table 2 Management of urinary retention:

A rough guide to treatment:

As previously stated a definitive treatment may only take place once there is a diagnosis and quality of life is taken into consideration.

BPH is the most common cause of BOO in men. In patients with uncomplicated BOO and adequate quality of life as per IPSS no intervention is indicated and a period of "watchful waiting" with monitoring is a good starting point. The next step is medical therapy using alpha-blockers, which act on prostatic smooth muscle involved in the dynamic component of BPO, or/and 5 alpha-reductase inhibitors which inhibit the conversion of testosterone to dihydrotestosterone and reduce volume effects often referred to as the static component. Should this fail or not be appropriate the choice lies between conservative (urethral /suprapubic catheter or CISC) and operative management (Trans Urethral Resection of Prostate (TURP) or open prostatectomy).(Fig3)

Urethral stricture disease involves the formation of scar tissue within the urethra. Initial temptation to dilate the urethra should be avoided so a short stricture is not converted to a longer, denser stricture requiring complicated reconstruction. Pending investigation the options are dilatation, optical urethrotomy or excision and reconstruction.

Physical causes of urethral obstruction such as prolapsing pelvic organ or post incontinence surgery will necessitate repair where appropriate otherwise conservative management. Where there is no physical obstruction, such as in neurological disorders, the options lie with CISC or in-dwelling catheters. In some cases this may not be possible and consideration of a surgical procedure to form a catheterisable stoma, Mitrofanoff procedure, may be warranted.

BOO (BLADDER OUTLET OBSTRUCTION)

Rohit Chaturvedi

In young, pre-menopausal women the diagnosis of Fowlers syndrome, a primary disorder of urethral sphincter relaxation, may be considered. This disorder tends to affect women in their third decade and is associated with polycystic ovaries in 50 % of cases. Often this group of patients struggle with CISC as they find upon removal of the catheter they experience discomfort or the sensation the catheter is stuck. A standard BOO work-up of investigation can indicate the diagnosis but ideally sphincter electromyogram (EMG) is required. Treatment is often conservative with either a no intervention or CISC approach. In those with severe symptoms consideration of sacral neuromodulation may be required.

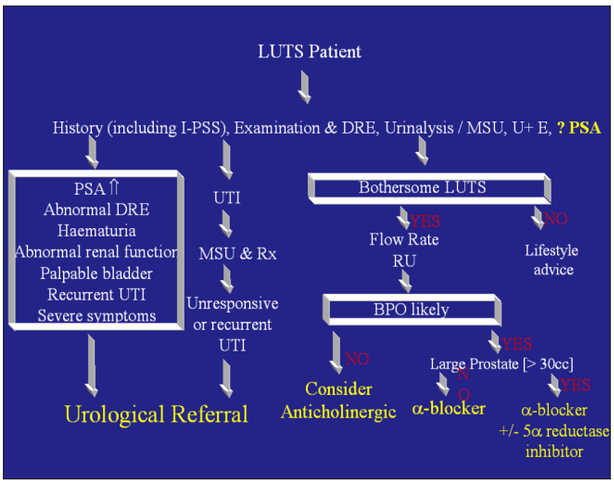


Fig 3

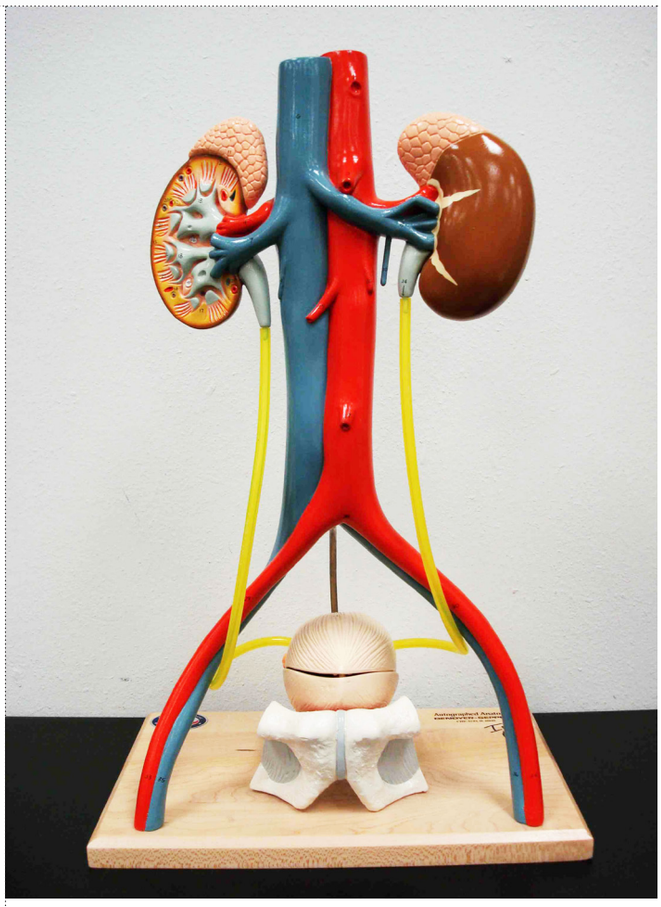
In Summary

The topic of BOO is diverse, crossing over with multiple other topics in Urology. Most diagnosis and management plans can be formed with a logical approach to the topic, encompassing a detailed history with examination and appropriate investigations. Avoid the pitfalls of assuming all patients with voiding difficulty have BOO and where there is a complicated picture or confusion proceed to Urodynamics to aid treatment and management.

Urology Questions (BOO)

1. A 55 year old gentleman presents with difficulties passing urine. Which is not an irritative lower urinary tract symptom?

a) frequency
b) nocturia
c) urgency
d) hesitancy
e) urge incontinance



2. Drugs in bladder outflow obstruction: For each of the pharmacological agents described below, select the appropriate mode of action. Each option may be used once, more than once or not at all.

Pharmacological agents:
a) Tamsulosin
b) Finasteride
c) Oxybutynin
d) Tolteridine
e) Goserelin

Mode of action:
1) LHRH analogue
2) Anticholinergic
3) Alphablocker
4) PDE5 inhibitor
5) 5 alpha reductase inhibitor

3.
A. Benign Prostatic Hypertrophy
B. Fowlers Syndrome
C. Cauda Equina
D. Urinary Tract Infection
E. Constipation
F. Prostate Cancer

BOO (BLADDER OUTLET OBSTRUCTION)

Rohit Chaturvedi

Match each case below with the most likely diagnosis above.

1) 42 year old female fell from ladder while decorating, has developed weakness in legs, incontinence of faeces, saddle anaesthesia, and has palpable suprapubic mass.

2) 67 year old gentleman presents in the outpatient department with worsening LUTS over a period of 3 months. DRE is suspicious however his PSA is 2.0ng/ml. He is also awaiting an orthopaedic assessment for back pain.

3) 24 year old female presents to the accident and emergency department in urinary retention with a residual volume of 1200ml. On examination you note she suffers from acne and hirsutism. She states in her family history she has known polycystic ovaries.

4. True/False

1) A diagnosis of High Pressure Chronic Urinary Retention is based on the residual volume.

2) Women may suffer symptoms of "prostatism".

3) Bladder Outlet Obstruction refers to the relationship between detrusor pressure on voiding and urinary flow and is therefore defined as high pressure, low flow.

4) The ideal investigation for complicated lower urinary tract symptoms is a uroflow.

5) Lower urinary tract symptoms correspond to the causal aetiology.

5. Which statement is correct?

1) PSA is sensitive and specific for prostate cancer.

2) A patient with urodynamically proven bladder outflow obstruction and low IPSS score is best served by a TURP.

3) The purpose of a DRE is to assess the prostate.

4) Raised serum creatinine suggests acute urinary retention.

5) Fluid volume charts are a useful tool in the assessment of frequency and nocturia.

Answers

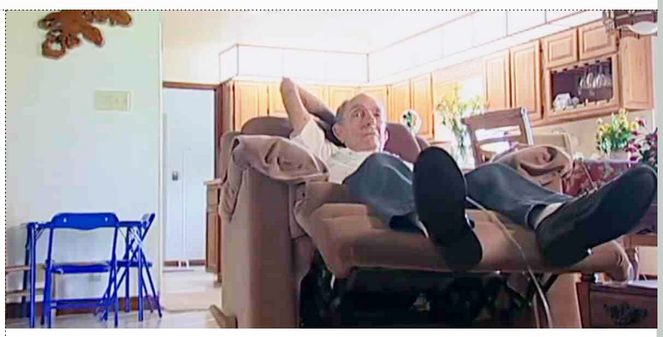
Q1) D

Q2) a=3, b=5, c and d=2, e=1

Q3) 1= cauda equina, 2= Prostate cancer (remember PSA is not specific and may not be raised if poorly differentiated i.e. high Gleason grade), 3= Fowlers Syndrome

Q4) F,T,T,F,F.

Q5) 5 (2= if a patient does not symptomatic of their BOO or feels they have an adequate quality of life a TURP may not be of use when considering risk against benefit. 3= remember that the DRE also assesses anal tone and presence of faecal loading)



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FACIAL NERVE PALSY – A SURGICAL TRAINEE'S GUIDE

Ameet Gupta

Facial nerve palsy – A surgical trainee's guide. Otorhinolaryngology & Neck Surgery.

Facial nerve palsy is a commonly encountered clinical presentation and as such all junior surgical trainees should be well versed in its pathophysiology and management. The aim of this article is to outline the neuroanatomy of the facial nerve (cranial nerve VII), aetiologies, both common and important of facial nerve palsy, and clarify the management of the most common causes.

Neuroanatomy

The facial nerve (CN VII) arises from the junction between the pons and medulla (cerebellopontine angle) just lateral to the abducens nerve. At this point, the facial nerve is split into two distinct fibres. The first is the motor root and the other is the sensory/parasympathetic root (nervus intermedius). Both fibres pass through the pontine cistern into the temporal bone via the internal acoustic meatus and fuse to from the facial nerve. The nerve runs along the facial canal (in the petrous temporal bone) and reaches the medial wall of the middle ear. In the facial canal, the nerve branches into the stapedius, chorda tympani and the greater petrosal nerve. These nerves have the following functions:

i. Stapedius nerve – innervation of the stapedius muscle.

ii. Chorda tympani – parasympathetic fibres to submandibular and sublingual salivary glands. Taste fibres from anterior two thirds of tongue.

iii. Greater petrosal nerve - preganglionic parasympathetic fibres to sphenopalatine ganglion and postganglionic fibres to nasal and lacrimal glands.

The facial nerve bends sharply back (this is the geniculate ganglion) and passes down on the posterior wall of the middle ear and exits the cranium via the stylomastoid foramen. On leaving the cranium, the nerve enters the parotid gland (parotid plexus) and gives rise to five branches which innervate the muscles of facial expression:

i. Temporal
ii. Zygomatic
iii. Buccal
iv. Mandibular
v. Cervical

Aetiology

Although there are many causes of facial nerve palsy we will split them into 3 categories.

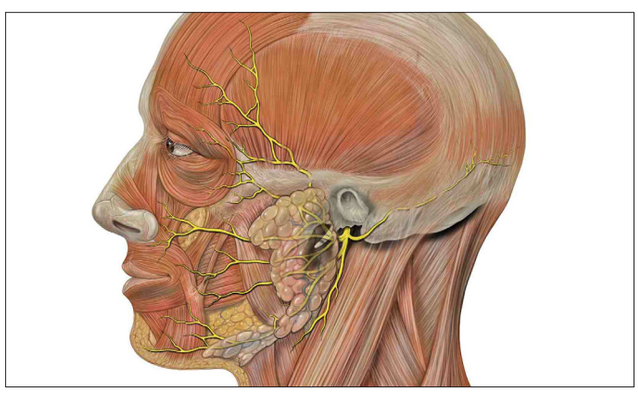
i. Upper Motor neurone
ii. Lower Motor neurone
iii. Others

Figure 1 shows the causes of facial nerve palsy opposite.

Clinical presentation

Take a history of the presenting problem, for example if there was postauricular pain (common in both idiopathic nerve palsy and Ramsay Hunt syndrome), or a painful ear with hearing loss (acute otitis media on its own or mastoiditis as a complication of cholesteatoma), a long standing hearing loss with an intermittent smelly discharge (cholesteatoma), a parotid mass (a parotid malignancy) or any other associated symptoms is essential. Past medical history is also relevant (diabetes mellitus, connective tissue or autoimmune disorders).

The first sign of facial nerve palsy is facial muscle weakness (predominantly unilateral). Patients may notice that one side of their mouth or eyebrow droops. Associated symptoms such as, decreased tear production, mild otalgia, hyperacusis (due to stapedius dysfunction) and altered taste are not uncommon. Vestibulocochlear nerve involvement (facial nerve runs close to cranial nerve VIII) can also be seen . Be aware of additional symptoms such as hearing loss, vertigo, nystagmus and tinnitus that may indicate a brainstem lesion. Once facial nerve palsy has been recognised it is important to illicit if it is and upper or lower motor neurone lesion (see clinical examination).



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Ameet Gupta

Lower Motor Neurone	Upper Motor Neurone	Other
Idiopathic (Bell's Palsy)	**Cerebrovascular accidents**	**Guillain-Barre**
Infective Herpes Virus Type 1 Herpes Zoster (Ramsay-Hunt) Otitis Media/ Malignant externa Mastoiditis Parotitis Others (TB/Polio)	Infective HIV Syphilis Meningitis Leprosy	Infective Lyme disease Encephalitis Mumps Poliomelitis
Trauma Skull base fracture Temporal bone fractures Facial injuries Parotid surgery Birth injury – forceps delivery		
Tumours 7CN tumour Cholesteatoma Parotid gland tumours Meningioma Schwannoma Skull base tumour	**Tumours** Acoustic Neuroma Posterior Fossa neoplasms	**Sarcoid** Melkerson Rosenthal syndrome (recurrent 7CN palsy, furrowed tongue, facio-orbital skin lesions) Langerhans cell histiocytosis
Vasculitis/Autoimmune diseases	**Vasculitides**	Myaesthenia gravis Multiple sclerosis
Congenital Moebius syndrome (facial diplegia & CN defects)		Diabetes mellitus Hypertension Pregnancy
Drugs – local anaesthetic		

Figure 1. Causes of facial nerve palsy.

Upper Motor Neurone Lesions

Cerebrovascular accidents – These are common and it is important to identify as this as a cause as it will help identify this as a cause and they will require prompt medical management. Look for other signs of cerebrovasular disease (contralateral limb weakness, dysphasia/dysarthria/diplopia).

Infective (HIV, Meningitis, Syphilis) – These are unusual causes. A clinical history and examination are vital to rule out the range of causes listed in figure 1. Look for opportunistic infection in HIV. Patients may be systemically unwell and present with meningism (photophobia, neck stiffness).

Multiple sclerosis (MS) – An uncommon cause.

Tumours – Progressive or recurrent facial nerve palsy should raise suspicion. Other cranial nerve symptoms, especially from nerve V and VIII, should be considered as suspicious and investigated further. Acoustic neuroma, is an intracranial tumour of the vestibulocochlear nerve (CN VIII) and it usually presents with unilateral senineuronal hearing loss, tinnitus and vertigo but it rarely presents with a 7CN palsy.

Vasculitidies – Whilst uncommon, look for other systemic signs in the eyes, skin, chest and kidneys.

Lower Motor Neurone Lesions

Bell's Palsy (idiopathic) – The most common cause of facial nerve palsy. The prognosis is worse with age, complete paralysis, late onset of recovery, loss of taste and postauricular pain. It accounts for over 80 percent of reported cases. Studies have implicated latent herpes simplex virus and its re-activation. It is more common in diabetics, respiratory tract infections and pregnancy. The clinical presentation is of a rapid unilateral facial weakness over one day after excluding other pathological causes. Diagnosis is a clinical. The incidence is between 20-32/100,000 affecting 1 in 65 people over a lifetime. HSV-1 has been implicated as a possible aetiological agent. Patients do better if they are given prednisolone and acyclovir as early in its onset as possible.

Infective

i. Herpes zoster oticus (Ramsay Hunt syndrome) – If herpes zoster infection is associated with facial nerve palsy it is known as Ramsay Hunt syndrome. Caused by reactivation of latent varicella zoster virus in the geniculate ganglion. Less common then Bell's palsy but causes more severe dysfunction and poorer prognosis, especially in patients presenting with complete paralysis, vertigo, underlying diabetes or hypertension. Maximal loss of function is seen at 1 week. Characterized by severe otalgia, unilateral vesicular eruption (dermatomal distribution) around the ear, tongue or soft palate and can involve the vetibulocochlear nerve. Diagnosis made clinically.

ii. Otitis media/externa – Otitis media can rarely result in facial nerve palsy. Urgent myringotomy is needed. Malignant otitis externa can cause facial nerve palsy. These patients need to be admitted for intravenous antibiotics and CT scanning.

iii. Parotitis - Parotid infection can cause facial nerve palsy but if parotid cause is suspected then neoplasm must be ruled out. Detailed examination of the head and neck with attention to masses and lymph nodes. Bilateral facial nerve palsies can occur in mumps infection.

Trauma – Skull base fractures and temporal bone fractures can present with facial nerve palsy.

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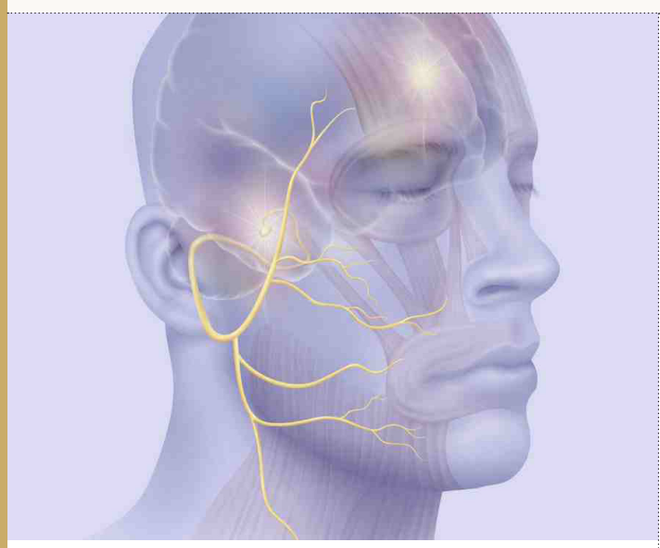

Tumours – Cholesteatoma is a destructive squamous epithelial growth of the middle ear. It can affect the facial nerve in a minority of cases, usually when there is an associated mastoiditis with a dehiscent 7CN.

Other

Lyme disease – caused by bites from ticks (Borellia), is seen in endemic areas. History of recent travel (USA) or contact with dears is important to note. Erythema migrans and tick bites imply Lymes disease.

Guillian-Barre – Causes bilateral palsy.

Examination and Investigation

A full neurological examination of both peripheral and cranial nerves is essential. Clinical examination can reveal if the lesion is upper or lower motor in nature, by asking the patient to 'raise their eyebrows'. In upper motor neurone lesion this function will be retained as there is contra-lateral upper motor neurone supply. If the lesion affects the lower 7CN the unilateral forehead movement is lost completely. If the eyelid cannot be closed fully then Bell's phenomenon maybe seen (upward deviation of the eye when closure of the eye is attempted).

Otoscopy should be carried out to exclude ear pathology and 512KHz tuning fork tests (Rinne and Weber) should be done to assess any hearing deficit.

Examination of the head and neck should exclude primary tumours, especially if there is a parotid masse.

Grading of the palsy is done using the Modified House-Brackman scoring system (Figure 2).

The diagnosis of Bell's palsy and Ramsay Hunt syndrome is made on history and clinical examination and requires no other investigations in the acute setting. Other investigations are used if the diagnosis is unclear.

Blood test - White cell count and inflammatory markers may be raised in acute infections. If a vasculis is suspected the erythrocyte sedimentation rate (ESR), C Reactive protein (CRP) and cytoplasmic anti-neutrophil cytoplasmic antibody (cANCA) may be raised. Blood cultures should be sent for pyrexial patients.

Pure tone audiology (PTA) - A key investigation if any vestibulochochlear involvement is suspected. Can be done as a baseline measurement of hearing to monitor improvement of symptoms.

Electromyography (EMG) - Not used routinely in acute setting. EMG has been used to assess facial muscle degeneration in complete palsy for research purposes.

Polymerase chain reaction (PCR) - VZV can be detected in tears, middle ear fluid, saliva and blood mononuclear cells, but is not essential to make the diagnosis.

CSF - Unless meningitis or MS is suspected this should not be required. Senior medical/neurological advice should be sough before collecting a sample. Schirmer's test – Assess lacrimation (not used routinely).

CT - Should be done in the context of trauma to assess for petrous temporal fractures. If an intracranial cause is suspected (i.e. tumour/CVA, malignant otitis externa, or cholesteotoma.

MRI – This is advisable for diagnosing intracrainial (acoustic neuroma) and parotid tumours or if MS is suspected.

Grade	Function
I	Normal function
II	Slight weakness, normal symmetry and tone, complete eye closure and forehead movements with effort, mouth slightly weak
III	Some asymmetry, complete eye closure and forehead movements with effort, mouth slightly weak
IV	Obvious asymmetry with disfigurement, incomplete eye closure and no forehead movements, mouth asymmetry
V	Only barely perceptible movement, asymmetry at rest, incomplete eye closure, no forehead movement, slight mouth movement
VI	No movement

Figure 2. Modified House-Brackman grading system

Treatment

Eye protection - This must be given to all patients with facial nerve palsy with incomplete eye closure and protection. A decrease in tear production can lead to dryness and corneal damage. Give lubricating eye drops and ointment for night use. If the eye does not close fully then tape shut over night.

FACIAL NERVE PALSY – A SURGICAL TRAINEE'S GUIDE

Ameet Gupta

Corticosteriods - Corticosteroids are the mainstay of idiopathic or post traumatic palsy without bony narrowing of the canal. The most widely accepted regime is 7 days of prednisolone 40-60mg per day with a tapering dose, given within the first 72 hours of the onset of symptoms.

Antiviral therapy – Acyclovir is used in the treatment of Ramsay Hunt syndrome but is also used in Bell's palsy as facial weakness precedes vesicles in some cases, 800-1000mg per day in 4-5 divided doses for 7-10 days is an acceptable regime (care with contraindications, especially renal). The use of antivirals in conjunction with steroid treatment has been shown to significantly improve recovery.

Surgery – In otitis media with progressive pain a bulging tympanic membrane and pyrexia , an urgent myringotomy is needed along with antibiotic, steroid and antiviral therapy. Surgical decompression of the middle ear in Bell's palsy is not carried out as the risks of further nerve damage out weighs any potential benefits. A surgical opinion must be sought for tumours.

Physical therapy – There is no evidence that physical therapy in the acute setting has any benefit. Facial massage and facial retraining can be advised for patients who make a poor recovery, but there is little evidence to support that this alters the natural history.

Special considerations

Treatment in Pregnancy - Very little evidence exists to show that antivirals are harmful in pregnancy but its use is restricted to severe cases. Acyclovir is excreted in breast milk and breastfeeding should be discontinued if given. Prednisolone is relatively safe in pregnancy and short courses of up to 40mg/day are safe during breastfeeding.

Children - Facial nerve palsy is much less coming in children but Bell's palsy still remains the top cause. Children have a better prognosis. If children present with a complete palsy they also have a poor prognosis as do adults. There is no compelling evidence that antivirals or steroids improves outcome in children.

Complications

Facial myokymia – uncontrolled fine facial movements

Hemifacial spasm – intermittent contraction of facial muscles

Crocodile tears – Lacrimation while eating

Synkinesis – contraction of groups of muscles that do not usually contract together (i.e. eyes and corner of the mouth)

Blepharospasm – spasmodic eye closure

Prognosis and follow up

Eighty percent of patients with a complete Bell's palsy have a full recovery with no treatment. The vast majority of patients with a partial idiopathic palsy make a complete recovery. The rate of recovery is less for patients with Ramsay Hunt syndrome. Patient diagnosed with a facial nerve palsy of unknown aetiology should receive oral steroids antivirals, as early in its onset as possible and pending results. The treatments to be aware of are:

i. Botulinum A toxin injection for blephrospasm and synkinesis

ii. Gold weight implants for eye closure

iii. Facial reanimation surgery to improve function and cosmetic appearance

Top Tips

i. Thorough history (otological, neurological and systemic symptoms).
ii. Full neurological, head and neck examinations plus tuning fork tests and otoscopy.
iii. A pure tone audiogram is desirable.
iv. If vesicles are present think Ramsay Hunt syndrome
v. Treatment: prednisolone and acyclovir for 7-10days (as early in the onset as possible if presenting within the first week).
vi. Follow up in ENT clinic in 6 months.
vii. Patients with otitis media require URGENT surgery.
viii. Imaging only required if pathology unclear.
ix. If in doubt consult ENT team.
x. A tumour should be excluded in an ipsilateral recurrent 7CN palsy particularly if the paralysis was progressive or incomplete.

Causes of Facial Nerve Palsy Questions

Select the most appropriate diagnosis for the scenarios below. Each option maybe used once, more than once or not at all.

A. Multiple sclerosis
B. Otitis media
C. Ramsey Hunt syndrome
D. Parotid tumour
E. Acoustic neuroma
F. Bell's palsy
G. Cerebrovascular accident
H. Otitis externa
I. Cholesteotoma
J. Acute parotitis
K. Guillain-Barré syndrome

FACIAL NERVE PALSY – A SURGICAL TRAINEE'S GUIDE

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Question 1. A 61-year-old woman presents with a 24 hour history of sever left sided ear pain. She has also noticed that her face is drooping on the left side. On examination you notice several vesicles over the left ear.

Question 2. A 50-year-old man presents with a history of dizziness, tinnitus and left facial numbness. Examination reveals left sensorineural hearing loss, and left trigeminal and facial nerve palsies.

Question 3. You see a 13-year-old boy in an ENT emergency clinic. He has presented after waking up with right sided facial weakness. He also complains of right ear pain and muffled hearing on the right side. On examination he is pyrexial, has a Grade IV facial nerve palsy and otoscopy revels a red bulging tympanic membrane on the right side.

Question 4. A 39-year-old Mexican woman complains of facial asymmetry after an episode of swine flu 1 week ago. Examination revels a grade III facial nerve palsy. He is otherwise well.

Question 5. You are called to see a 91-year-old lady on the medical ward. She has been admitted from a nursing home after becoming 'off legs'. On examination, the woman is unkempt and has an obvious left sided facial weakness. Her temperature is 38.1oC. A neurological exam is difficult as she suffers with dementia but you notice that she has a tender swelling over the left parotid gland. Otoscopy is normal.

Answers

Question 1. C. Ramsey Hunt syndrome

Question 2. E. Acoustic neuroma

Question 3. B. Otitis media

Question 4.. F. Bell's palsy

Question 5. J. Acute parotitis

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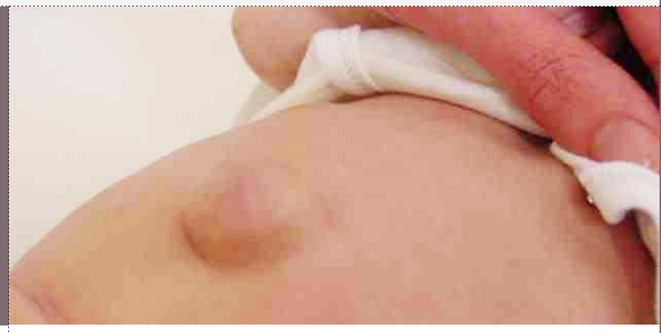
INGUINAL HERNIA/HYDROCELE

Gregory Shepherd



Paediatric Hernias & Hydrocoeles.
Paediatric Surgery.

An 18 month old boy presents to A&E with a twelve hour history of a swelling in his right hemi-scrotum. The A&E doctor reports the child had two non-bilious vomits yesterday. He is also coryzal and has had a normal bowel motion today. The doctor is concerned he has a strangulated hernia. When you arrive the patient is happily playing in the children's area and smiles as you approach him.

1. What is the differential diagnosis?

2. What important points need eliciting in a history and examination?

3. Does this child need an operation and if so, should it be as an emergency or elective procedure?

Introduction

Inguinal hernia and hydrocoele are one of the most common congenital abnormalities seen in paediatric surgical practice. Their underlying pathology is very similar, as is the surgical correction. However, the decision as to when to treat is markedly different, and so a clear understanding of these conditions and confidence in correctly differentiating between the two is vital.

Pathophysiology

During normal intra-abdominal development of the testis, a strand of mesenchymal cells develops at the lower pole of the testis. This develops into a band like structure, the gubernaculum (or round ligament in females). This extends down through the abdominal wall in the groin to the base of the scrotum or labia. At the eighth week of gestation an outpouching of peritoneum forms, following the gubernaculum through the developing abdominal wall layers to the scrotum. The testis descends down this peritoneal channel or processus vaginalis (PV) to the scrotum. The PV usually closes off around the time of delivery, or shortly afterwards. The distal part of the PV surrounding the testis remains, forming the tunica vaginalis. If however the PV remains patent, fluid can pass down this channel, collecting around the testicle or in the cord, forming a hydrocoele. (Patent processus vaginalis PPV see fig 1). If the channel is large enough to accommodate bowel, omentum or ovary then a hernia is present. In the first year of life 90% of hydrocoeles resolve spontaneously and there is still potential to resolve up to the age of 2 years.

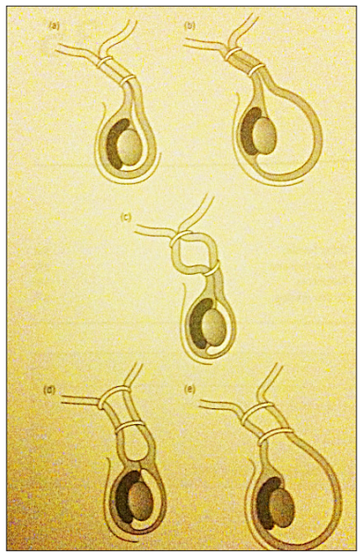


Fig 1 (fig 35.2 p302 Paediatric Surgery 2nd edition Burge, Griffiths ISBN 0-340-80910-8)

Incidence of clinically apparent Patent Processus Vaginalis
M:F 5:1
60% Right Sided
30% Left Sided
10% Bilateral
30% of premature infants
2% of all children

History & Examination

The majority of children will present with a swelling in the groin or scrotum. In an infant this may be noticed at a health check or when a parent is changing a nappy. Older children may report it themselves. A hernia will often be an intermittent swelling more noticeable when the child is straining or crying. A hydrocoele can also be intermittent although it is often present for longer periods. Important points to elucidate are if the swelling is ever painful, nonreducible, if there is any vomiting or any abdominal distension. On examination if the swelling is present it is usually possible to differentiate between a hernia and a hydrocoele.

INGUINAL HERNIA/HYDROCELE

Gregory Shepherd

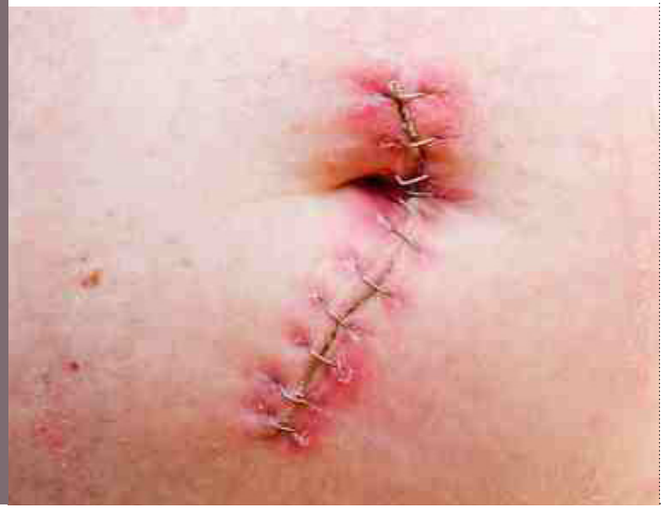
Most hydrocoeles are fluid within the tunica vaginalis around the testicle and so it is possible on examination to reach on top of or above the swelling. Hydrocoeles of the cord or infantile hydrocoeles can present a difficulty as they clinically can appear like a hernia because they extend up the cord, or they only reside in the cord, not extending into the tunica vaginalis. Herniae can be reduced, and it is often possible to hear or palpate the gurgle of bowel as it reduces. This reducibility is the only reliable way of distinguishing between a hernia or hydrocoele. It is never possible to palpate above a hernia.

Transillumination can be helpful but should not be relied upon as young children's bowel can transilluminate as the tissues are so thin. If a swelling is not evident it may still be possible to feel the thickened layers of the empty sac by palpation of the cord structures, especially as they pass over the pubic tubercle. Because they feel like layers of silk sliding over each other, this is called the 'silk sign'. Even without clinical evidence of a hernia, most surgeons would operate on the child based on a clear convincing history from the mother or patient who point to the appropriate place as the site of the swelling.

Management

Because of the potential for resolution, surgical correction for hydrocoeles is not offered until they are 2 years of age. Once seen, a patient with a reducible hernia will be put on the waiting list for a repair. The younger the patient, the more likely the hernia will incarcerate and strangulate; therefore the more urgently it needs to be performed. (12% of all children under 12y with hernia will incarcerate; 30% incarcerate in term babies) Most centres would try to accommodate a neonate with a hernia within 2 weeks, under 6 months of age within 6 weeks; older children added to the waiting list would normally have their operation within 2-3 months. Babies that are still on the neonatal intensive care and therefore have never left hospital, should have their hernias fixed before discharge.

Hernia repair is normally performed as a day case procedure. Although expremature babies up to 54-60 weeks gestational age, are usually observed overnight as they are at risk of apnoeas post anaesthetic; the exact age criteria depends on the local anaesthesiology guidelines.



If a hernia however is non-reducible, it is said to be incarcerated. If left, the neck of the hernia compresses the contents causing venous congestion, leading to oedema, eventually cutting off the arterial supply and subsequently causing ischaemia and infarction (called strangulation). This compression within the canal can affect the testicular blood supply, resulting in ischaemia or infarction of the ipsilateral testis. This can lead to subsequent atrophy or loss of the testicle, especially in the infant. Initially, fluid resuscitation is often required due to significant fluid losses. Then, to prevent strangulation, efforts are made to urgently attempt reduction of the hernia with strong analgesia combined with sedation if necessary. Reduction should be attempted or supervised by an experienced surgeon once the child is comfortable.

Techniques vary, but the general principle is one of providing slow constant pressure in the line of the canal on the lower end of the hernia; whilst simultaneously applying pressure over the ipsilateral external ring (unlike adults where the constricting ring is the deep ring), directing the hernia back into the abdomen. An emergency repair of these hernias without reduction, results in a high complication rate (~10%) due to the swelling and oedema, so once reduced repair is not attempted for another 24-48 hours to allow this to subside.

Consent	
Benefits	Prevent risks of incarceration (for hernia repair): -damage to hernial contents (bowel, omentum, ovary) -damage to cord structures (vas, vessels and testicle) -mortality
	Cosmesis
Risks	Bleeding, infection (1%)
	Damage to Vas and Vessels (1%) leading to testicular atrophy or loss of testicle (<1%) (higher in incarcerated herniae)
	Cryptorchidism (1%)
	Recurrence (1% but higher in premature infants)

Operation	
Skin prep	Chlorhexadine solution
	To include area from umbilicus to top of thigh
Drape	Superior drape - cover from just below umbilicus upwards
	Inferior drape - cover top of thigh and perineum. Scrotum to be accessible in a male.
	Lateral drape - cover lateral to ASIS
	Medial drape - cover just short of the midline so that external genitalia accessible

INGUINAL HERNIA/HYDROCELE

Gregory Shepherd

The incision is made with a 15 scalpel in the natural skin crease above the groin for improved cosmesis. The skin crease is usually superior to the inguinal canal, but in children the superficial tissues can be easily retracted down over the canal. The lateral end of the incision need not extend beyond the deep ring and the medial end should be in line with the superficial ring (Fig 2).

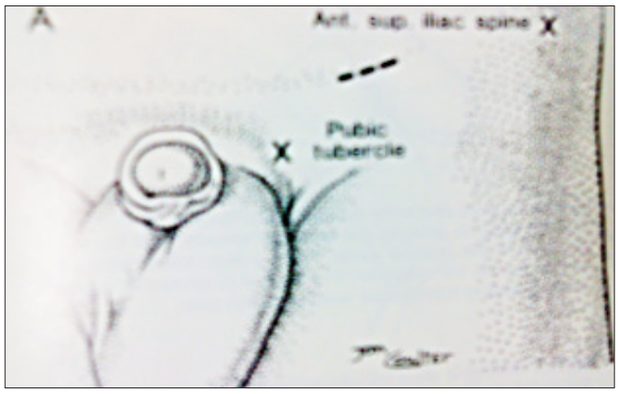


Fig 2 - ISBN 081519210x Pediatric Surgery (Welch, Randolph et al) pp781

Usually palpation of the cord, rolling it over the pubic tubercle gives a good guide of the positioning of the superficial ring. The deep ring lies at the midpoint of the inguinal ligament (extends from the ASIS to the pubic tubercle – not to be confused with the positioning of the femoral artery at the mid-inguinal point, which lies at the midpoint of a line from the ASIS to the pubic symphysis).

Dissection through tissues can be a combination of sharp and blunt dissection with scissors or with diathermy (bipolar or monopolar). This description will be for the former, but the principles are the same.

Blunt dissection with scissors is performed down to Scarpa's fascia through the superficial fat. Scarpa's fascia is held up with toothed forceps and a small window made with a snip of the scissors. The tip is placed through the window and then spread. Small retractors are placed within the window and spread to reveal the underlying tissues. A fatty layer of varying thickness is dissected through onto the external oblique. The plane between fat and external oblique is dissected out, down to the inguinal ligament and medially to the superficial ring. Care should be taken not to dissect inferior to the inguinal ligament as the femoral vessels are extremely close by. The fibres of the external oblique can be seen to form a V at the superficial ring where they pass around the cord as it exits the canal. By pressing into the wound with the retractors either side of the canal, an impression of the path of the cord can be seen to bulge upwards. An incision is made in the external oblique over the canal and scissors used to extend the window towards the superficial ring (Fig 3).

Fig 3 – ISBN 10-0412591103 Pediatric Surgery (Rob and Smith's Operative Surgery 3rd Edition) pp3

If necessary, the window can be extended to open the superficial ring. By holding the inferior leaf of the open external oblique and sweeping the deeper tissues from its internal surface, one has an impression of where the cord structures lie. The internal oblique aponeurosis is now visible. 2 pair of Debakey non tooth forceps can now be used to dissect through this layer. By pressing gently though the layer with closed forceps and allowing the natural spring of the forceps to open up, one can see the deeper tissues. The second Debakey can be passed closed into the deeper tissue while the first pair are open and then opened themselves in turn; this is repeated until the cord contents are seen. If there is difficulty in identifying where the cord structures are, a gentle tug on the testicle externally produces movement along the cord, visible in the wound. Once identified, the cord can be picked up with the Debakey. The cord is covered in a thick layer of cremaster. Using the Debakey or an artery clip the cord can be dissected free either side until an inverse V or "trouser-leg" can be seen either side (the proximal and distal parts of the cord forming the 2 legs while the first Debakey is holding the apex of the V). A curved clip or right-angle can be passed underneath through the space of the V to the other side (Fig 4).

Fig 4 ISBN 10-0412591103 Pediatric Surgery (Rob and Smith's Operative Surgery 3rd Edition) pp6

INGUINAL HERNIA/HYDROCELE

Gregory Shepherd

The clip bridges the incision holding the cord contents out of the wound. The last layer is the internal spermatic fascia, which can be opened by tearing it between Debakeys. The shiny pale sac of the hernia or PPV which lies anterior to the vas and vessels, should now be visible. Due to this position it is safe to pick up the sac with the Debakeys. Now one must sweep the tissues off of the sac with the Debakeys, carefully advancing around the sac to the posterior where the vas and vessels should be. These structures should not themselves be held by the forceps, but swept clear so as not to damage them (Fig 5).

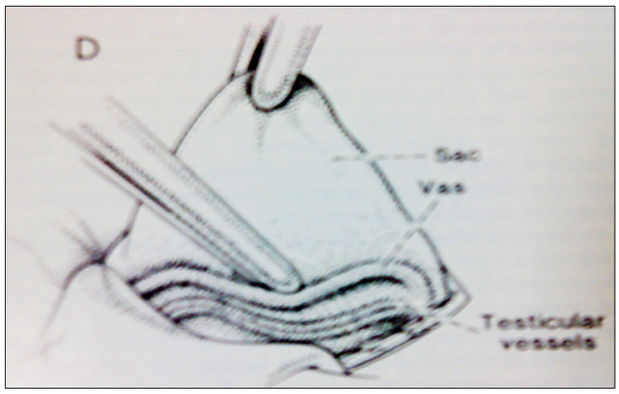


Fig 5 ISBN 081519210x Pediatric Surgery (Welch, Randolph et al pp781

The vas is a white firm tubular structure, which can be easily palpated against the underlying clip. Once dissected apart from the sac, these important structures are retracted to one side to avoid damaging them. An artery clip is applied to either side of the sac and the sac transected. Multiple clips can be applied to the edges to maintain control while looking inside the proximal sac to the deep ring to ensure the whole sac has been taken before a final clip is placed across the proximal sac. In girls traction on the inside lateral wall of the sac where the round ligament is will deliver the ovary and fallopian tube for inspection. The distal sac can be opened further to drain any residual hydrocoele. The vas/vessels are held with traction while the remaining tissues are swept away from the sac up to the deep ring, this is done by applying pressure to the tissues next to the sac with forceps or a swab in the direction of the deep ring (Fig 6).

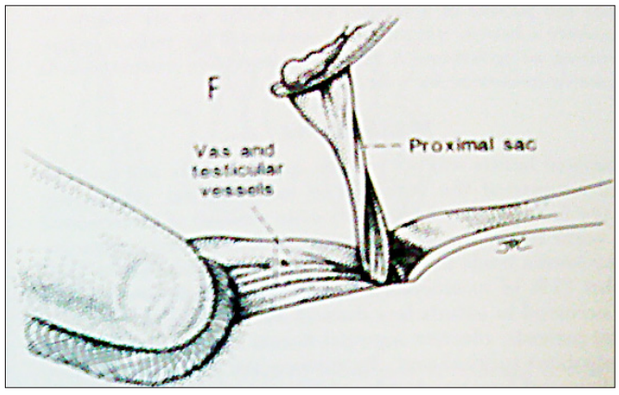
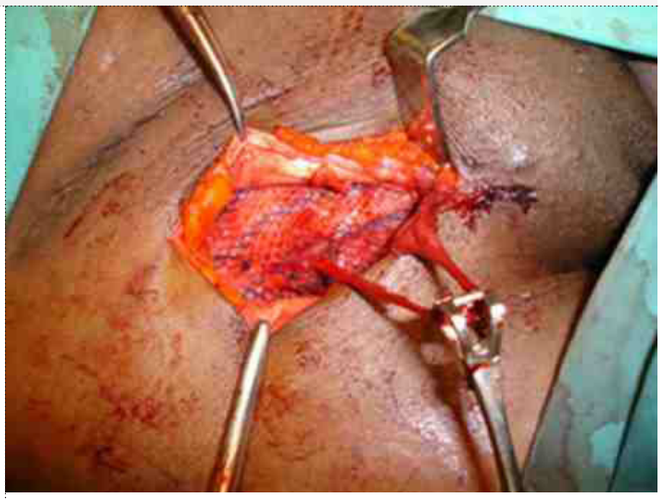


Fig 6 - ISBN 081519210x Pediatric Surgery (Welch, Randolph et al) pp782



Preperitoneal fat identifies that dissection is sufficient. A twist is made on the proximal sac, ensuring the vas and vessels are clear. The hernia sac is transfixed at the deep ring with 3/0 vicryl and then the sac excised. Once tissue and suture are cut, the transfixed stump is seen to retract back into the wound. Placing traction on the testicle reduces the cord structures, allowing closure of the external oblique and Scarpas with 3/0 vicryl. Care should be taken to ensure the deeper structures are not caught in the sutures. 5/0 vicryl or monocryl can be used as a subcuticular suture, knotted or not depending on surgeon's preference, with a waterproof dressing. It is good practice to check one more time that the testicle still resides in the scrotum.

Documentation

Everyone has their own way to document operations, normally including Incision, Procedure, Findings, Closure and Postop Plan. But it is important in this procedure to note specifically the vas and vessels being identified and preserved, the size of the testicle compared to the contralateral testis and that it lies in the scrotum after closure. In females the presence of an ovary if visualised, should be noted.

Special Concerns

One of the most common ways patients with disorders of sexual differentiation (DSD) present to a surgeon is with a hernia. Therefore phenotypic girls with herniae, especially bilateral, should have their ovaries visualised intraoperatively to confirm XX genotype. If a testis or ovotestis is seen, many surgeons advocate gonadal biopsy (although not all) and then referral to an endocrinologist is required. Likewise, phenotypic boys with Mullerian-inhibiting substance deficiency may have mullerian structures in the hernia (fallopian tubes).

INGUINAL HERNIA/HYDROCELE

Gregory Shepherd

Questions

1. You see a 1 yr old boy in clinic with a non reducible scrotal swelling that you can palpate above; The testis lies towards the posterior of the swelling and feels normal; you should:

A - Arrange an urgent USS of the testis to rule out any hidden testicular pathology and followup in clinic with the result

B - Reassure parents this is a benign condition and requires an operation; book the child for ligation of PPV as a daycase.

C - Reassure the parents this is a benign condition that does not require treatment and discharge them from followup

D - Reassure the parents that this is a benign condition that usually resolves by itself but give them the option of: follow-up in 1 year; or for the GP to refer back if it is still present in a year

E - Admit for an emergency operation for an incarcerated inguinal hernia

2. An ex 27 week baby, now term corrected gestation, is referred with a reducible inguinoscrotal swelling. One cannot palpate above on examination and it is separate to the testis. The baby is on the low dependency unit of the neonatal unit and due to go home this evening. You should:

A - Take the patient details, book him for a daycase inguinal herniotomy in 4-6 weeks time and let the patient go home in the meantime to put on weight in preparation for his surgery.

B - Book the patient for an inpatient inguinal herniotomy as a semi-elective procedure in the next couple of weeks. The patient should stay in hospital until then, delaying his discharge.

C - Reassure the parents that this should resolve by the age of 2 years, but if it is still present the GP can refer him back.

D - Book him for an inguinal herniotomy on the emergency list so that he can go home that evening as planned

E - Take the patient details and arrange an outpatient appointment for 6 weeks time.

3. A mother attends clinic with her 3 month old baby girl. She reports seeing a "golf-ball" sized lump in her daughter's groin on 3 or 4 occasions. This appears whenever the child cries and spontaneously disappears when the child relaxes. On examination there is no abnormality, even when the child cries. Mum points to the superficial inguinal ring as the site of the previous swelling. You should:

A - list the child for a daycase inguinal herniotomy

B - Reassure mum that there is no abnormality and discharge her back to the GP

C - Explain to mum this was probably a hernia, but it has likely resolved. Arrange for a further clinic appointment in a year to reassure her.

D - Admit the child to the ward and discuss this case of Munchausen-by-proxy with the child protection team.

E - Reassure mum this is a benign condition that should resolve by the age of 2 years. If it is still present then, the GP can refer her back.

4. A 3 year old boy is admitted as an emergency via the GP with an irreducible left inguinoscrotal swelling. The child has been coryzal for a few days, has a cough and vomited twice. His abdomen is full but nontender. His right testicle and hemiscrotum examine normally. The left inguinoscrotal swelling is irreducible, tender, one cannot get above it, but it transilluminates. The left testicle is not palpable. He is dehydrated and requires fluid resuscitation. Parents report the swelling has been there for almost 18 hours. The most appropriate course of action is:

A – Reassure the parents this is a hydrocoele, probably exacerbated by the viral illness. Send him home with a date to come in as a daycase for a PPV ligation.

B – Explain to parents this is likely a torsion of an undescended testis. As it has been 18 hours the testis is dead and an orchidectomy can be scheduled for the following day.

C – Explain to parents this is an inguinal hernia that has become stuck. Give the patient some paracetamol and ibuprofen and attempt reduction after 20 minutes. If this fails proceed to emergency theatre for operative reduction.

D – Explain to parents this is an inguinal hernia that has become stuck. Book the child on the emergency list for emergency herniotomy as a CEPOD 1a.

E – Explain to parents this is an inguinal hernia that has become stuck. Attempt a reduction with opiate analgesia and ketamine sedation with the supervision of the consultant. If this fails proceed to emergency theatre for operative reduction

5. Whilst operating on an inguinal hernia in a 9 year-old girl, a testis is identified in the sac; as the operating surgeon you should:

A - Excise the testis, counsel the parents on your findings and refer to an endocrinologist

B - Biopsy the testis, inform the parents their child is in fact a boy and refer to an endocrinologist

C - Leave the testis, counsel the parents on your findings and refer to an endocrinologist

D - Leave the testis, immediately perform a PV examination to assess the female anatomy, counsel the parents on your findings and refer to an endocrinologist

E - Excise the testis, inform the parents their child was a boy, but he is now a girl as you have removed the testis

INGUINAL HERNIA/HYDROCELE

Gregory Shepherd

Answers

1. – D

If one can palpate above the swelling, it is a hydrocoele. At this age this is due to a patent processus vaginalis, not testicular pathology. Operation as a daycase is offered at 2 years of age as the likelihood of spontaneous closure after this age is minimal.

2 - B

Babies with hernias on the neonatal unit should have them repaired before discharge as they have the highest rate of incarceration. Ex prems up to 56-60 weeks gestational age should be admitted overnight after their surgery as they are at risk of apnoeas postoperatively and so daycase repair is not appropriate.

3 - A

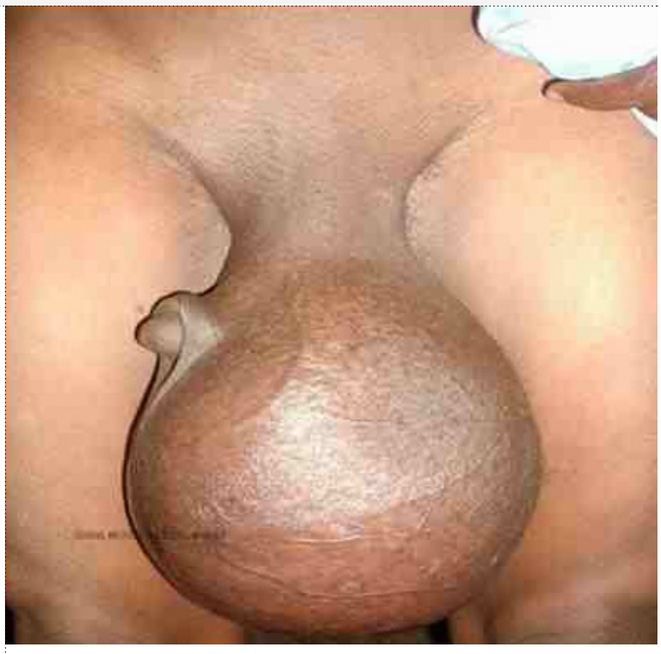
A clear history from mum is sufficient evidence of a hernia. The risk of incarceration precludes waiting a year for it to reappear.

4 – E

This is an irreducible hernia. As the child is dehydrated and vomiting this could well be strangulated and should not be assumed to be part of their viral illness. An attempt at reduction should be made with adequate analgesia or sedation. Only if this fails should operative reduction be performed.

5. - C

Both leaving the testis and biopsy of the testis are appropriate courses of action, however a surgeon should avoid assigning a sex to the child until all investigations are completed and counseling with an Endocrinologist and Paediatric Urologist has taken place. A pelvic USS is the most appropriate examination for assessing the internal anatomy. Any EUA should only be performed once consent has been gained and parents and child have been counseled regarding the potential significance of possible findings.



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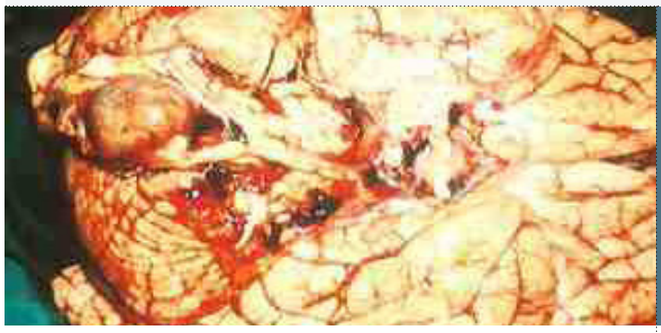
SUBARACHNOID HAEMORRHAGE

Rohitashwa Sinha and Anshuman Sengupta



Subarachnoid haemorrhage (SAH) describes a bleed, usually from a ruptured intracranial aneurysm, into the subarachnoid space.
Neurosurgery.

Abstract

Subarachnoid haemorrhage (SAH) describes a bleed, usually from a ruptured intracranial aneurysm, into the subarachnoid space. Many die of the immediate consequences of the initial bleed; 21% die within 24 hours. In those that survive to medical attention, subsequent irritation of brain parenchyma, impaired cerebral perfusion and interruption of CSF flow result in additional complications which, if untreated, contribute to morbidity and mortality. It is therefore imperative to stabilise patients and achieve a diagnosis as a matter of urgency so that appropriately selected patients can be treated on a neurosciences unit. If a culprit lesion is found, definitive treatment by endovascular coiling or surgical clipping is a priority to prevent re-bleeding, which has a mortality rate of up to 80%. This is followed by intensive support and early recognition and management of complications such as vasospasm, hydrocephalus and hyponatraemia. Some management strategies for SAH are widely adopted on the basis of high quality evidence, but the complexity and persisting morbidity and mortality of the condition has meant that a great deal of research is being carried out in order to find novel treatment strategies. This article reviews the pathophysiology of SAH and summarises the salient features in assessment and management of sufferers. It also provides a brief overview of recent research and controversies in the area.

Case Vignette

Mrs Berry, a 54 year old lifelong smoker, complains of an abrupt severe headache whilst at work. She is found by her husband later in the day, having collapsed at home. An ambulance crew assess her to be obeying commands and eye opening to voice with confused speech. She is noted to have a BP of 190/110; all other observations are within normal limits. En route to the emergency department (ED) she has two episodes of vomiting. A computed tomography (CT) scan on admission shows hyperdense blood within the basal cisterns extending towards the left Sylvian fissure.

Subarachnoid haemorrhage: A bleed into the subarachnoid space which directly irritates brain parenchyma and causes local pressure effects via interference with cerebral perfusion and local cerebrospinal fluid (CSF) flow.

Pathophysiology

The brain is perfused by cerebral arteries and their branches, which together form the Circle of Willis. This is a common site for aneurysms, the sudden rupture of which is responsible for the majority of SAH[1]; this article will therefore focus specifically on these lesions. See Box 1 for a more comprehensive list of causative lesions. Traditional vascular risk factors such as age, hypertension and smoking predispose to SAH in patients with aneurysms; other associations include polycystic kidney disease[2]. Theories regarding aneurysm formation include degenerative weakening in the arterial wall from atherosclerosis, and the stress of pulsations on the arterial wall at turning or branching points such as those within the Circle of Willis[3]. Furthermore, intracranial arteries have poorly developed external elastic lamina and adventitial layers and a thin media; factors that further predispose to aneurysm development under haemodynamic strain[3].

Most clinical features relate to the direct irritation of brain parenchyma and meninges, caused by blood leaking into the subarachnoid space. See Figure 1 for a CT head scan showing a typical SAH. The hydrocephalus that can complicate SAH can also be explained by disturbance of the normal physiology of cerebrospinal fluid production and flow. Figure 2 summarises this; essentially, the presence of free blood in the subarachnoid space impairs the last step, i.e. reabsorption of CSF within the arachnoid granulations of the meninges. This CSF accumulation can lead to a communicating hydrocephalus, though non-communicating hydrocephalus can rarely occur if a blood clot focally obstructs CSF flow within the ventricular system.

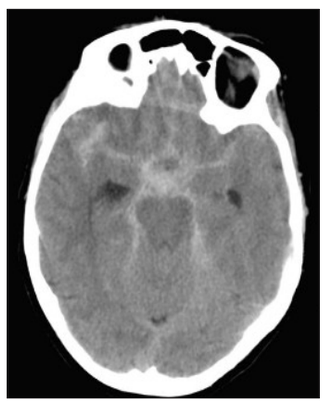


Figure 1: Axial computed tomography image of a typical subarachnoid haemorrhage.

SUBARACHNOID HAEMORRHAGE

Rohitashwa Sinha and Anshuman Sengupta

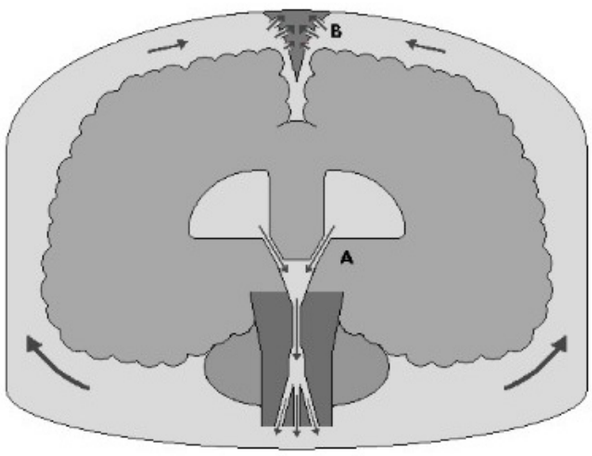
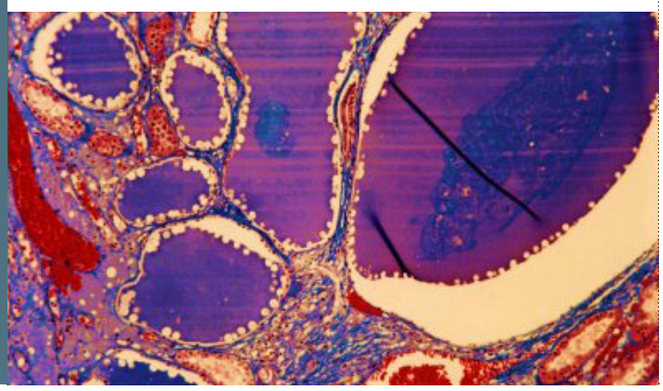


Figure 2: Schematic diagram of normal cerebrospinal fluid (CSF) flow
CSF is produced in the ventricular system (A) and passed along lateral ventricles via the foramen of Munro to the third ventricle then to the fourth ventricle by the cerebral aqueduct of Sylvius. It exits via the foramina of Lushka and Magendie to envelop the central nervous system. It is mostly reabsorbed via the arachnoid granulations into the superior sagittal sinus (B). Subarachnoid blood can impair reabsorption at (B) causing communicating hydrocephalus.

Ruptured cerebral artery aneurysm (85%)
Non-aneursymal perimesencephalic haemorrhage (~10%)

The remaining ~5% consist of SAH from:
- Arteriovenous malformations (AVMs)
- Cavernous angiomas
- Bleeds from brain tumours
- Mycotic aneurysms
- Secondary to cocaine/amphetamine abuse

Box 1. Aetiology of subarachnoid haemorrhage
NB. It is worth noting that trauma is the commonest cause of subarachnoid haemorrhage per se, however the distribution of the blood is very different and often co-exists with intraparenchymal, subdural and/or extradural bleeds.



Incidence and Impact

Subarachnoid haemorrhage has an incidence varying between 2 and 23 per 100,000 patients, depending on the country[4]. The overall incidence rate across all age groups in the UK has been quoted at 9.7 per 100,000 person-years[5]. It affects a variety of age groups, with half of patients in one study under the age of 55[6]. The incidence increases with age; significantly more so for females than males between 65 - 74 (30.9 vs 18 per 100,000 person-years)[5]. Its impact is considerable in terms of both mortality and morbidity: the case fatality rate is 21% on the first day, 37% at one week and 44% at 30 days[5], and 20% are rendered incapable of full self-care after discharge[7]. Clearly, this condition necessitates urgent recognition, investigation and management, which will form the focus of the remainder of this article.

Clinical Features

Sudden onset severe headache is the hallmark of SAH. Box 2 summarises typical features in the history.

Site - Generally diffuse but can be occipital
Onset - Within seconds as reported by 75% of patients[8]
Character - 'Thunderclap'/'hit with a bat'
Radiation - Retro-orbital 'pressure' sensation.

Associated features
Nausea & vomiting (reported in 77%)[8]
Neck stiffness (35%)
Seizures (7%) – strong indicator for aneurysmal rupture[9]
Depressed GCS – prognostic implications
Ocular features (14%) – flame-shaped haemorrhages near the optic disc or in the vitreous body;
Terson's syndrome[10]
Focal neurological deficits – from aneurysm (e.g. CN III palsy from posterior communicating artery
aneurysm) or due to cerebral ischaemia

Timing – Initial 'thunderclap' headache becomes constant from blood continually irritating the meninges, worse in the mornings if hydrocephalus develops.

Exacerbating factors - Worsened by movement and bright light.

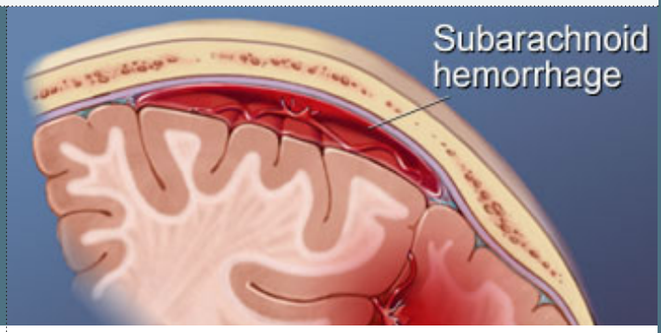
Severity - Another hallmark feature: '10/10'/ 'worst headache ever'

Box 2: Typical Clinical Features in SAH

SUBARACHNOID HAEMORRHAGE

Rohitashwa Sinha and Anshuman Sengupta

Subarachnoid haemorrhage (SAH) describes a bleed, usually from a ruptured intracranial aneurysm, into the subarachnoid space.
Neurosurgery.



Assessment and Investigation

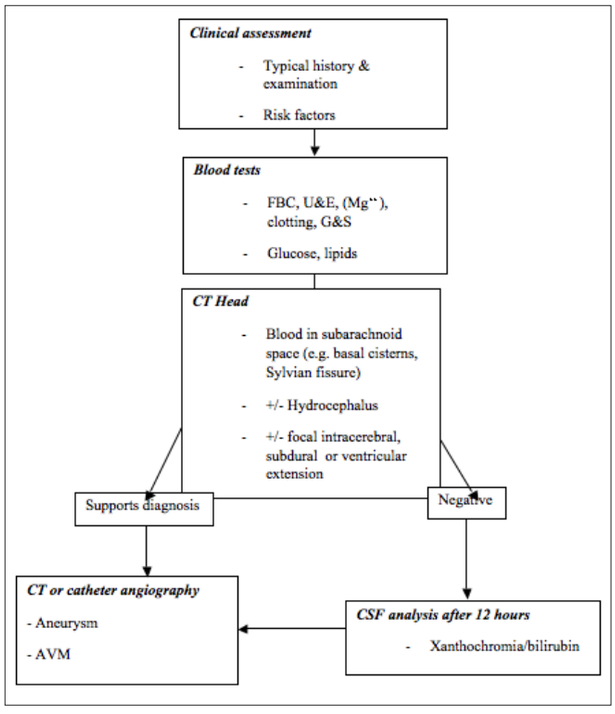


Figure 3: Assessment and investigation of suspected SAH
FBC = full blood count; U&E = urea and electrolytes; G&S = Group and screen; ECG = electrocardiograph; CT = computed tomography; CSF = cerebrospinal fluid; AVM = arteriovenous malformation

Severity scoring systems

The extent of SAH from aneurysmal rupture can vary considerably between patients. This results in a wide clinical spectrum of presentation and natural history. As mentioned previously, severe SAH is associated with significant mortality and morbidity. However, it is equally important to diagnose less severe cases in order to prevent complications such as re-bleed. To help stratify this spectrum into useful grades of severity, three scoring systems are in common use. The World Federation of Neurosurgeons (WFNS) and Hunt and Hess systems categorise patients by their clinical presentation and both correlate well with outcome[11].

The Fisher scale classifies SAH based on CT appearance and quantification of subarachnoid blood. It is a useful predictor for the development of symptomatic vasospasm, a serious complication of SAH[11]. See tables 1-3 for a summary of these scoring systems.

The World Federation of Neurological Surgeons (WFNS) grading scale depends on clinical presentation parameters		
Grade	**GCS**	**Motor deficit**
I	15	-
II	13-14	-
III	13-14	+
IV	7-12	+/-
V	3-6	+/-

Table 1: World Federation of Neurological Surgeons grading system

Hunt & Hess grading system based upon clinical features at presentation	
1	Asymptomatic, mild headache, slight nuchal rigidity.
2	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy.
3	Drowsiness / confusion, mild focal neurological deficit.
4	Stupor, moderate-severe hemiparesis.
5	Coma, decerebrate posturing.

Table 2: Hunt & Hess grading system

The Fisher Grade classifies the appearance of subarachnoid haemorrhage on CT scan:	
1	No haemorrhage evident.
2	Subarachnoid haemorrhage less than 1mm thick in diffuse distribution
3	Subarachnoid haemorrhage more than 1mm thick with localised clot
4	Subarachnoid haemorrhage of any thickness with intra-ventricular hemorrhage (IVH) or intraparenchymal extension.

Table 3: Fisher grading system Management

SUBARACHNOID HAEMORRHAGE

Rohitashwa Sinha and Anshuman Sengupta

Management

The management of SAH patients commences with an 'ABCDE' assessment at presentation. Particular attention should be paid to potential airway compromise in patients with depressed GCS, with consideration of early anaesthetic support. After stabilisation, investigations as outlined in Figure 3 should be undertaken. A positive CT or LP mandates discussion with the nearest neurosurgical (or occasionally neurological) unit. Accurate clinical and radiological evaluation of patients is critical to tailoring further management appropriate to severity. Poor-grade SAH patients may need intensive care or high dependency support. For patients with the most severe presentation amid significant co-morbidities and extensive SAH on CT, it may even be appropriate to consider conservative management. Some patients with reduced GCS may require immediate neurosurgery to lower raised intracranial pressure either by an external ventricular drain (EVD) for hydrocephalus (see Figure 4) or evacuation of haematoma.

Patients who, after appropriate evaluation, are transferred to a neurosciences unit will undergo securing of their culprit lesion, management of the potential complications and rehabilitation. Depending upon the severity grade, this can take a few days on a general neurosurgery ward or weeks with a period of critical care management. The following recommendations are applied in several centres, though there is marked inter-unit variation:

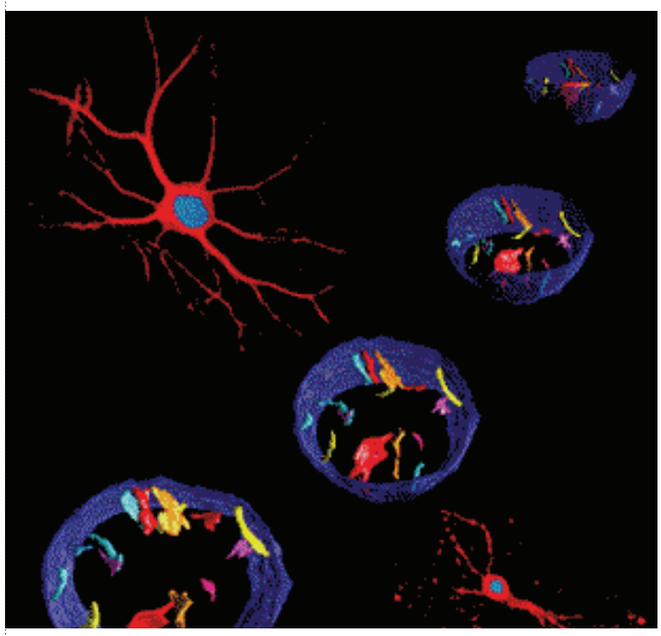
- Frequent observation for neurological deterioration which may herald a re-bleed, hydrocephalus or vasospasm.
- Bed rest to minimise intracranial haemodynamic stresses and DVT prophylaxis (TED stockings, anticoagulation used when aneurysm has been secured)
- 3 litres of 0.9% saline every 24 hours. A careful balance must be struck between the risk of rebleed and hypoperfusion in guiding BP management. Being overcautious of rebleed can lead to hypoperfusion and increase the risk of cerebral vasospasm.
- Consideration of arterial line insertion for invasive BP monitoring and access for bloods.
- Consideration of nasogastric tube if doubt about cough and swallowing reflexes.
- Oral nimodipine 60mg four-hourly for 21 days to reduce vasospasm via calcium channel antagonism; NG is preferable to IV administration to avoid systemic hypotension.
- Urinary catheterisation as a guide to end-organ perfusion.
- Analgesia (using morphine preparations at the discretion of the responsible consultant).
- Anti-emetics and laxatives to minimise intracranial haemodynamic stresses.
- As-required antiepileptic medication.
- Liaison with neuro-radiologists for CT and/or catheter angiography to identify and treat culprit lesions and prevent re-bleeding.

Endovascular coiling, usually by a neuroradiologist, uses platinum 'Guglielmi' coils to embolise aneurysms intraluminally under angiographic guidance. See Figure 5 for digital subtraction angiography images of an aneurysm before and after coiling. Surgical treatment involves craniotomy for microsurgical dissection to the culprit lesion and securing the neck of the aneurysm with a clip.

Even with the aneurysm secured, continued monitoring is warranted. The focus is on regular documentation of GCS and examination for focal neurological deficits, with prompt CT scanning in the event of deterioration. SAH patients are at risk of the following complications:

- **Re-bleeding:** a poor prognostic indicator. The risk of its development is minimised by definitive management of the culprit lesion. As the subsequent rate of mortality or disability is 80%[12], urgent securing of aneurysms is a primary objective.

- **Delayed cerebral ischaemia.** The pathophysiology of this phenomenon is not well understood but it can result in global hypoperfusion and infarction. Generalised arterial vasospasm has been implicated, with maximal narrowing between day 5 and 14 post-ictus[13]. Close monitoring of the patient's mean arterial pressure (MAP) is essential. The so-called "triple-H" regimen (hypertension, hypervolaemia and haemodilution) can be tailored according to objective parameters in an attempt to treat vasospasm, though the efficacy of this strategy in terms of outcome is unproven. Inotropic support may be required to maintain an adequate MAP. Transcranial Dopplers (TCD) and/or CT perfusion can help to diagnose vasospasm, though there is no universally approved screening tool[1].



- **Hydrocephalus:** typically characterised by a fall in GCS over a few hours. After radiological confirmation, management involves either a period of observation (about 50% resolve spontaneously[14]) or decompression by either lumbar puncture (LP), or insertion of a lumbar drain or EVD. Occasionally, persistent hydrocephalus will not resolve spontaneously or by temporary EVD insertion. In these cases long-term treatment with a ventriculo-peritoneal (VP) shunt is required.

SUBARACHNOID HAEMORRHAGE

Rohitashwa Sinha and Anshuman Sengupta

SAH is also associated with a number of non-neurological complications, including chest infections, pulmonary oedema and cardiac complications[15]. A rare but important feature in the first ten days, usually from anterior circulation aneurysms, is the development of hyponatraemia, putatively caused by "cerebral salt-wasting": natriuresis with consequent hypovolaemia[15]. This can increase the risk of vasospasm and thus the condition requires consideration of careful oral and/or intravenous sodium supplementation. Overly rapid correction is associated with central pontine myelinolysis, thus daily sodium measurements are essential.

Patients with secure aneurysms who have been monitored and treated for emergent complications may then require rehabilitation and a follow-up screening plan for new or unsecured aneurysms. Long-term management also includes risk factor modification, screening tailored on an individual basis and psychosocial follow-up.

Case Vignette Part 2

Mrs Berry went on to have a CT angiogram that revealed an aneurysm in the left middle cerebral artery (MCA). After initial stabilisation, she was referred for endovascular coiling. Subsequently, she was managed on a neurosurgical HDU with nimodipine, triple-H therapy and other supportive measures. Despite this, she developed deficits suggestive of vasospasm on day seven. This was promptly treated with aggressive fluid resuscitation and inotropic support, resulting in improvement in her GCS and resolution of focal deficits. However, following a period of rehabilitation, a mild cognitive deficit remained, necessitating longer term neuropsychological follow-up.

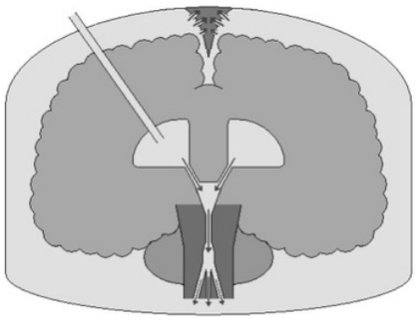


Figure 4: A schematic representation of an external ventricular drain to divert cerebrospinal fluid extracranially.

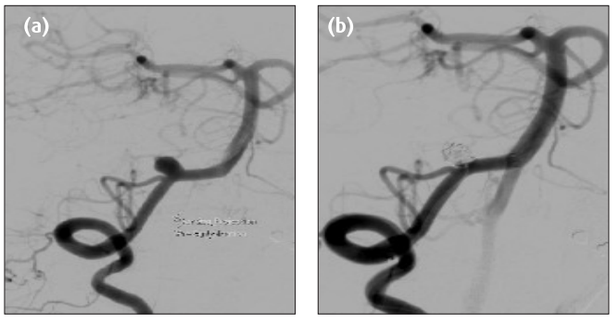


Figure 5: Digital subtraction angiography images showing a right posterior inferior cerebellar artery aneurysm before (a) and after (b) endovascular coiling.

Controversial issues in SAH

A variety of management strategies have been employed or are under investigation to reduce the burden of mortality in SAH[1]; the numerous complications result in a mortality rate of 50% in certain populations. This can result in marked inter-unit variation due to the complexity of the condition. A comprehensive review of the evidence is outwith the scope of this article; however, we supply a brief overview and refer the reader to recent American Stroke Association guidelines for more detailed discussion[16].

For example, since the International Subarachnoid Aneurysm Trial (ISAT[17]), coiling has been adopted as the first line definitive measure to prevent re-bleeding on the basis of a relative risk reduction in comparison with clipping. However, short-term follow-up from the same trial suggests reduced efficacy of aneurysmal occlusion and a higher re-bleeding rate[18]. Similar results have been replicated in a variety of studies, summarised in the ASA guidelines. Nonetheless, the evaluation at 1 year showed a significantly greater combined mortality and morbidity for patients whose aneurysms were secured surgically than those treated by coiling. It is worth noting that the patient group randomised between the two interventions had mostly low WFNS grade SAH, anterior circulation aneurysms and an underrepresented elderly population aged over 70. Therefore, the relative risk reduction for coiled patients cannot be safely generalised.

Given the profound implications of this management decision, it is critical to stress the importance of tailoring treatment specifically, taking into account the patient demographic and the nuances of the presentation. Even the timing of intervention is a source of controversy. In a study of 3521 patients, early surgery was significantly related to lower likelihood of preoperative bleeding[19], but this risk must be balanced against the benefit of optimising a patient for surgery over a few days after the initial bleed.

Throughout this discussion, we have assumed a SAH to instigate aneurysmal management. However, unruptured aneurysms can be found incidentally, giving rise to controversy in offering treatment with potential morbid complications to a well person; or follow up screening surveillance to people whose aneurysms are more likely to rupture with advancing age.

Similarly, controversy exists in the treatment of vasospasm. Whilst a Cochrane review recommended nimodipine as a preventative measure[20], the "triple-H" regimen is as yet unproven beyond the level of a "common sense" approach. Studies have suggested no difference in cerebral blood flow, vasospasm (as detected clinically and with TCDs) and functional outcomes between patients treated with normovolaemic versus hypervolaemic targets[21,22].

Other areas of interest apply to supportive measures such as magnesium replacement and glucose control. A randomised, placebo controlled trial of intravenous magnesium over two weeks showed improved outcome with reduced delayed cerebral ischaemia[23]. Statins have also been associated with reduced rates of vasospasm and death[24]. Similarly, hyperglycaemia has been linked with poor outcome, though no randomised studies have evaluated the utility of intensive glucose control[25].

SUBARACHNOID HAEMORRHAGE

Rohitashwa Sinha and Anshuman Sengupta

Subarachnoid Haemorrhage MCQs

Please select the single best answer from the 5 statements following the question stems as below:

1. In the presentation of subarachnoid haemorrhage:

a. Sudden onset headache associated with seizures makes the diagnosis of aneurysmal subarachnoid haemorrhage very unlikely.
b. Intracranial aneurysms are the commonest cause of subarachnoid haemorrhage followed by arteriovenous malformations and trauma.
c. A low threshold of clinical suspicion to investigate even mild severity sudden onset headache in patients can prevent the more severe complications such as re-bleed if low-grade subarachnoid haemorrhage is found to be the cause.
d. Subarachnoid haemorrhage has been associated with risk factors such as hypertension, smoking and polycystic ovarian syndrome.
e. A posterior communicating artery aneurysm can present with a cranial nerve VI (abducens nerve) palsy from local pressure of the aneurysm sac along the course of the nerve.

2. In the investigation of clinically suspected subarachnoid haemorrhage:

a. CT is only indicated after a negative lumbar puncture.
b. Lumbar puncture testing for red blood cells should be undertaken within 12 hours of the onset of symptoms of subarachnoid haemorrhage.
c. Lumbar puncture testing for red blood cells should be undertaken 12 hours after the onset of symptoms of subarachnoid haemorrhage.
d. CT angiography or catheter angiography are more sensitive investigations to identify culprit aneurysms or arteriovenous malformations than CT alone.
e. Lumbar puncture testing for xanthochromia is indicated within 12 hours of a negative initial CT head.

3. With regard to the severity grading systems commonly used in the management of subarachnoid haemorrhage:

a. The Hunt and Hess grade is solely based upon the patient's GCS at presentation and is a useful predictor of the development of symptomatic vasospasm.
b. The World Federation of Neurological Surgeons grade is solely based upon the patient's GCS at presentation and is a useful predictor of the development of symptomatic vasospasm.
c. The World Federation of Neurological Surgeons grade is based upon the patient's GCS at presentation and the presence or absence of motor deficit and is a useful predictor of the development of symptomatic vasospasm.
d. The Fisher grade is based upon the CT appearances of subarachnoid haemorrhage and is a useful predictor of the development of symptomatic vasospasm.
e. The Hunt and Hess grade is solely based upon the patient's GCS at presentation and correlates well with clinical outcome.

4. In the management of subarachnoid haemorrhage:

a. Fluid restriction is an important measure to reduce the risk of re-bleed and to treat hyponatraemia associated with subarachnoid haemorrhage.
b. Endovascular clipping to secure culprit aneurysms intra-luminally is undertaken with angiographic guidance.
c. Oral nimodipine 60mg orally every four hours is commonly used in patients with suspected or confirmed aneurysmal subarachnoid haemorrhage for 21 days post-ictus to reduce the risk of vasospasm.
d. Patients with secured intracranial aneurysms, either by clipping or coiling, are still at risk of re-bleed when anticoagulants are used for thromboprophylaxis and hence anticoagulants are contraindicated in aneurysmal subarachnoid haemorrhage
e. Evidence from the ISAT trial demonstrated greater efficacy of aneurysm occlusion, lower re-bleed rates and lower mortality for patients with aneurysms at various intracranial locations treated with endovascular coiling across a wide age range.

5. With regards to the complications of subrachnoid haemorrhage:

a. Re-bleed, hydrocephalus, vasospasm, hyponatraemia, seizures, pulmonary oedema, chest infections and cardiac arrhythmias are known potential complications in patients with subarachnoid haemorrhage.
b. Hyponatraemia in subarachnoid haemorrhage is caused by the 'Syndrome of Inappropriate Antidiuretic Hormone' secretion and is safely treated with fluid restriction.
c. 'Triple H' therapy of hypertension, hypervolaemia and hyperthermia is used to treat delayed cerebral ischaemia caused by vasospasm.
d. Hydrocephalus following subarachnoid haemorrhage is best managed by early insertion of a ventriculo-peritoneal shunt.
e. Patients with aneurysms secured by surgical clipping or endovascular coiling very rarely require neuro-rehabilitation.

Answers to Subarachnoid Haemorrhage MCQs:

Question 1. c
Question 2. d
Question 3. d
Question 4. c
Question 5. a

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SURGICAL TRAINING WITHIN THE CONFINES OF THE EUROPEAN WORKING TIME DIRECTIVE

John Findlay

The European Working Time Directive. Current Training Issues.

The European Working Time Directive

'[The] legislation dreamed up in Brussels is mainly concerned with the health and safety of manual workers. It was designed to protect Spanish lorry drivers or labourers working heavy machinery'.

Strongly worded rhetoric such as this, from John Black, President of the Royal College of Surgeons of England, and rebuttals from the Department of Health stating there to be 'no evidence that junior doctors' training is being compromised (Guardian, 2009) succinctly summarise the confrontation between Surgeons and Politicians. Few recent initiatives have created such partisan opinions as the European Working Time Directive (EWTD). As full implementation of this controversial legislation in the United Kingdom finally arrived in August 2009, out went the medical profession's decade of exemption, and in came public confrontation between the Surgical and political professions. Whatever the outcome of this conflict, however, it is clear that the EWTD will play a major role in shaping modern surgical training for the foreseeable future.

The directive

Although passed by the European Union in 1993 within Article 118a of the Treaty of the European Union (EC, 1993), the EWTD has its roots in the early 19th century. In 1818, the Paris Peace Conference debated, and threw out, the first legislative attempt to curb working hours in Europe. Whilst domestic laws limiting the length of manual labour shifts were gradually ratified, it was to take exactly 100 years more before the International Labour Organisation, an arm of the League of Nations, proposed a 48 hour working week, albeit unsuccessfully. 80 years later, we now witness the culmination of this process

The European Working Time Directive
Working time to not exeed 48 hours per week, averaged over 26 week period
11 consecutive hours of rest in every 24 hour period
Minimum of 24 hours rest every 7 days / 48 hours every 14 days
20 minutes break per 6 consective hours worked
4 weeks paid annual leave

Table 1. The European Working Time Directive

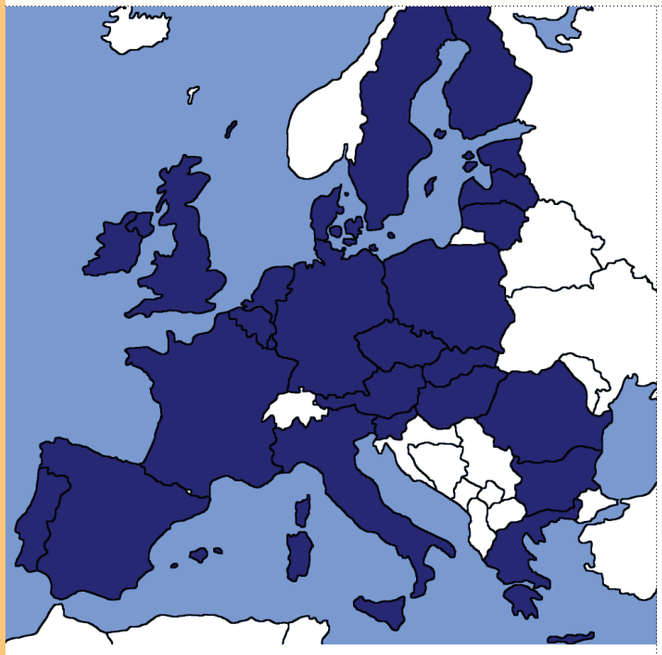
Initially, doctors were exempt from the EWTD, but this was to be revoked in 2000 with subsequent staggered caps on working weeks of 58 hours in 2004, 56 in 2007, and now 48 in 2009. This was followed by the SiMAP and Jaeger rulings, which stipulated that all on-call activity was classed as work (SiMAP, 2000). The surgical bodies in the United Kingdom have, with varying degrees of magnitude, expressed their concerns (ASGBI, 2008; ASiT, 2006; ASiT, 2009), however, it is important to consider that the EWTD represents just one facet of a changing training climate.

A changing profession

Surgical training is faced with a great deal of challenges, of which the EWTD is just one. Those to be surmounted include extrinsic legislative and policy influences, combined with rapidly evolving surgical practice. Recent years have witnessed a sea change in operating, with a rise in minimal access surgery reducing operative exposure of junior trainees. The abrupt implementation of Modernising Medical Careers, and its infamous recruitment process MTAS, provoked a media backlash, yet its legacy is now established. In response to criticism of the established Calman system of training, whereby a 'lost tribe' of SHOs were theoretically stagnating at a bottle neck prior to entering higher training, this tribe was forcibly repatriated into early selection and rigid career paths. However, whilst training became arguably more streamlined, it has been argued that many years of valuable SHO experience and training were lost in the process. The EWTD was preceded by the 'New Deal' for junior doctors in 1991, which aimed to reduce working weeks to 72 hours, and subsequently 56 hours (DOH, 1991). Furthermore, governmental pressure to reduce waiting lists and meet targets has, anecdotally, resulted in the abolition and over-booking of many dedicated training lists, further reducing operative exposure for the training surgeon.

SURGICAL TRAINING WITHIN THE CONFINES OF THE EUROPEAN WORKING TIME DIRECTIVE

John Findlay



Working weeks in other countries

The situation in other countries shows remarkable variety, even within Europe, due largely to variations in numbers of doctors per capita, and legislative attitudes. In the United States of America, where interns and residents continue to work hours far in excess of their European counterparts, the Institute of Medicine recently ruled that the minimum working week (for ensuring training and patient safety) should be 80 hours (Inglehart 2008). By contrast, in Scandinavia, where ratios of doctors to patients are significantly higher, working weeks have been curbed to 40 hours per week in Denmark and 48 hours prior to the introduction of EWTD in Sweden. However, countries with fewer staff such as Finland have struggled to maintain rotas at this level (ASGBI 2008). Germany has recently passed legislation exempting doctors from the EWTD to secure a 65 hour week, whilst many other European countries have thus far chosen to ignore the directive altogether.

Pros

The case for the EWTD is made largely on the grounds of health and safety (for patients and staff), and on lifestyle quality. There is a good evidence base that sleep-deprived junior doctors are more prone to psychological disturbance (anxiety, depression and agitation) (Owens 2001, Weinger 2002, Lewis 2003) and impairments in psychomotor performance such as laparoscopy (Taffinder 1998), and academic achievement (Stone 2000). However, the duration and structure of the optimum working week is unknown. Although working fewer hours clearly provides a luxury of 'free' time, not previously afforded, arguably these fewer hours (due to their shift distribution) may actually be more disruptive to home and family life (Thomas, 2003); indeed, roughly one half of surgeons have found this to be the case (Lowry and Cripps 2005a and b). However, being free from rota commitments may provide increased opportunites for theoretical and academic work.

Cons

Implications for service

For the NHS to become EWTD compliant, it has (had) to jettison 476,000 junior doctor hours per year, the equivalent of 9,900 doctors (British Medical Association 2004). This reduction in service provision by junior doctors is thought to necessitate an expansion of 7,500 consultants (Phillips 2004) and their workload (Lowry and Cripps, 2005a). For trusts to provide service, they must either expand the number of junior doctors (either training or non-training grades) with inevitable consequences for clinical exposure, or reduce the number of doctors covering out of hours (ASBGI 2008). These reductions in out-of-hours commitments, whilst ultimately relying on fewer doctors working harder and cross-covering more specialties (and thus arguably even less rested), might be offset to some extent by expansion of non-medical support staff roles (such as clinical support workers and nurse practioners) and programmes such as Hospital-At-Night. However, the direct surgical training opportunites of such programmes have already been criticised, as has the quality of care (Gossage 2006), and the underlying evidence is disparate (Herbertson et al, 2007; Findlay et al, 2009). Further to this, is the evolving separation of elective and emergency surgical services, combined with centralisation of emergency services (ASGBI, 2008).

Implications for surgical training

The central tenet of surgical opposition to the EWTD rests upon its status as a craft specialty, whereby fewer working hours equates to less training exposure. In the 1980s, when surgical trainees worked traditional 120 hour weeks, they accrued 32,000 hours of training; with a 48 hour week this is set to reduce to merely 18,000 (Chesher 2002)). This reduction in volume has been demonstrated (within a 56 hour week) to result in a reduction of operating theatre exposure of 25%, with a consequent reduction in logged cases of 21%; greater reductions were seen for traditional training operations such as varicose vein surgery (Morris-Stiff et al, 2004; ASGBI 2008). This reduction leaves emergency surgical competencies particularly vulnerable (ASGBI 2008), in addition to non-operative exposure such as outpatient clinics. As yesterday's junior trainees become more senior, this situation may be exacerbated as they augment their training with operations traditionally performed by junior trainees. Yet more compounding factors, include the expansion of Independent Treatment Centres, operating free of training responsibilities. Furthermore, shift patterns threaten to avulse trainees from their trainers, reducing their contact time and impeding the training relationships that a traditional firm structure fostered. A recent survey by the Association of Surgeons in Training found the vast majority of surgical trainees to be opposed to the EWTD, expressing concern that an under-trained generaton of consultant surgeons would inevitably ensue (ASiT 2006).

SURGICAL TRAINING WITHIN THE CONFINES OF THE EUROPEAN WORKING TIME DIRECTIVE

John Findlay

Implications for patient care

Whilst gains in patient care have been suggested, due to having more rested doctors under the EWTD, concern has been expressed that the converse is true. The negative health consequences of shift work, with its disruption of the circadian rhythm have been well described, with increased prevalence of cardiovascular, gastrointestinal, endocrinological and psychiatric morbidity (Boggild, 1999; Knuttson 2003; Costa 1981; Knuttson 1999; Knuttson 2000; Schernhammer, 2003; Morikawa 2005). Furthermore, night shift workers enjoy less quantity and quality of sleep during the day (Horricks 2004) than they might have whilst spending a night on-call, and are more prone to inury at both home and work (Suzuki, 2005; Barger 2005). Less healthy trainees are arguably dangerous to patient care. Other short term problems include the reductions in continuity of care with commensurate increased instances of handover, both with the potential to compromise patient care via medical error. In the long term, the case that less experienced and trained surgeons (however well rested) will be less able to provide quality patient care is strong.

Potential solutions

Whilst the EWTD and service structure continues to be debated by professional bodies, there is much that the individual trainee can do to protect his or her training. Firstly, is the individual opt-out from the EWTD allowing trainees to voluntarily work up to 56 hours per week (although still bound by the New Deal). In theory, whilst rotas should be 48 hour compliant, the opt-out allows trainees to undertake additional sessions such as locums (often internal to cover rota gaps) to gain experience. It also may prevent enforced absences when clinical need has required a trainee to work beyond their hours. Contractually, they must often recoup this time, inevitably from their elective and training sessions. However, it is crucial that the opt-out remain voluntary, and trainees must be well acquainted with the wording of their contracts, and their indemnity insurance. Opting-out should be undertaken on an individual basis with a trainee's employer; sample letters are available from the Department of Health, Association of Surgeons of Great Britain and Ireland, and the British Medical Association.

With reduced quantity of training, quality must be improved. Whilst the Royal Colleges strive to facilitate this with curricula such as the Intercollegiate Surgical Curriculum Project, there is again much the trainee can undertake individually. This involves opportunistic, and often innovative, exposure. For example, a small study has shown that by routinely performing hand-sewn anastamoses on freshly resected bowel a trainee can quadruple his experience of this valuable training procedure (Mcllhenny 2009). Other measures include maximising study leave allowances for training purposes, taking advantage of the growing availability of surgical simulators and lengthening overall training periods with fellowships. However, it is important not to neglect commensurate non-operative experience in the quest to fill the logbook. Clearly, surgery mandates a surgeon, not a technician, and ward rounds, outpatient clinics, postoperative management and critical care environments represent excellent opportunities to improve surgical decision making.

The EWTD, in addition to a number of other changes in process or on the horizon, presents a great number of challenges to trainee and trainer alike. However, it brings with it opportunities for change and innovative training. It is crucial that as trainees we are proactive to ensure that our training is ensured. Whoever may lose out from the conflict between clinicians and policy makers, we must ensure that it is not the patient. Ultimately, the EWTD, albeit not in isolation, seeks to impose arbitrary constraint upon the working practice of doctors. To shackle those, of whom the majority by their nature will work beyond the call of duty for their patients, is a risk. It may be that the greatest threat faced by the surgical profession is the indoctrination of a generation of trainee surgeons in a shift work mentality, to 'clock-off' and 'down-tools' at 5 pm.

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SURGICAL TRAINING WITHIN THE CONFINES OF THE EUROPEAN WORKING TIME DIRECTIVE

John Findlay

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CHANGING LIVES IN THE DEVELOPING WORLD

Stephen Bennett

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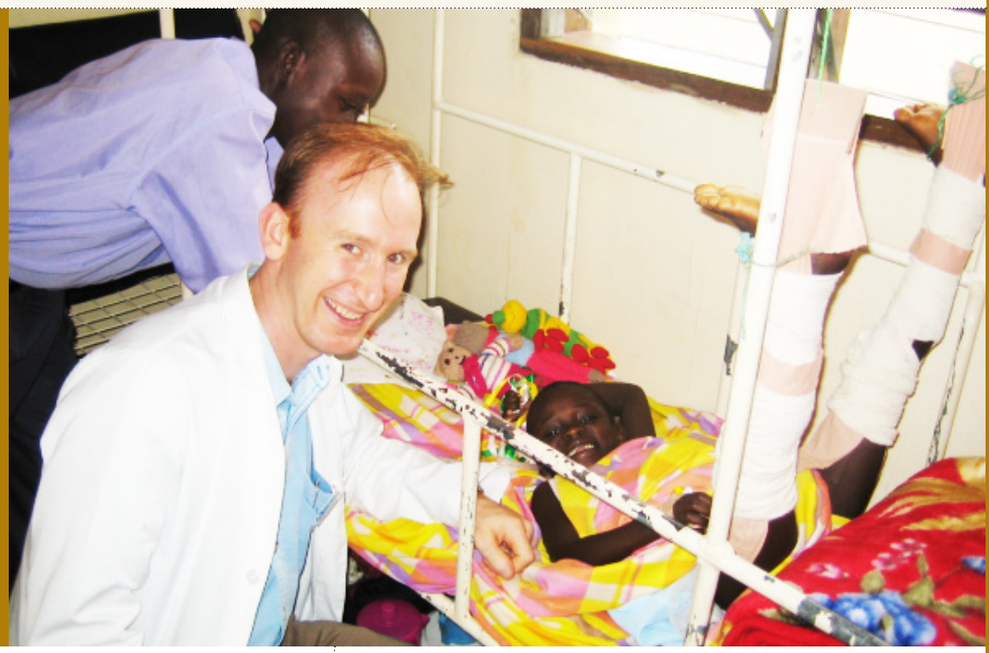

Introduction

How often do you personally make a real difference in someone's life? Sometimes in the wonderful institution that is our UK National Health Service, it can seem especially as a trainee surgeon that you're just a small cog in the machine, one small part of an increasingly large and ill-defined team jointly looking after a variety of patients, with the knowledge that most of what you do could easily be done by someone else. You may be the first doctor that the nurses call about a particular patient one morning, but often it'll be your senior who makes the decisions, and a different doctor who'll be there the next day.

I heard recently about Waswa, a baby I treated a couple of years ago. He had been six months old then, and had been very sick when he arrived at the hospital early one evening with ongoing bloody diarrhoea for the more than a week. After a quick history and examination, the diagnosis of intussusception was confirmed by a "target" appearance on handheld ultrasound I performed myself. Theatre staff were mobilised and emergency surgery performed, with a subtotal colectomy required to excise a completely necrosed colon, and an ileostomy formed to finish the operatation as quickly as possible and give this child a chance of survival. But survive he did, and a week later Waswa was discharged home with plans to return for a bowel rejoin six weeks later. I had been the only doctor in the hospital that evening. This was possibly the scariest surgical situation I had ever been in – a six month old baby who looked at death's door; an operation I could perform on an adult under supervision, but never alone or on a child let alone a small six-month baby; and no other doctor around to help. Waswa's mother was poor and couldn't afford to feed herself while she stayed to look after her baby while he recovered.

The family certainly couldn't afford to bring him back for the second procedure. I found myself giving some money for food and promising to pay for Waswa to return for his reversal. Two years later, Waswa is a healthy toddler, running around with only a abdominal scar and some oddly coloured skin at the site of his previous stoma to show that he is any different from the other children in hi s village.

Kato was a sixty year old man I treated last month on a return visit to my hospital. He actually came specifically to see me for treatment of dysphagia. He had got to the stage where he could manage liquids only by taking things slowly. Fibre-optic endoscopy confirmed a mid oesophageal tumour through which the scope couldn't pass. He was too poor to afford the bus fare to get to the national hospital, let alone the cost of a CT scan to stage the disease or subsequent operative or chemo-radiation treatment. Our hospital had no ventilation facilities so even an un-staged oesophageal operation was impossible.

However, I was able to dilate his tumour and place an oesophageal stent to at least allow him to be able to swallow over the next few months before his tumour progresses. I had set up an endoscopy unit two years earlier using donated equipment and supplies, and it was in regular diagnostic use by a Ugandan surgeon I had trained – I had happened to bring a dilator and stent with me on this visit, and they had been put to good use.

CHANGING LIVES IN THE DEVELOPING WORLD

Stephen Bennett

The hospital I was in was Kiwoko Hospital in Uganda, a 250 bed rural mission hospital where I worked as one of two surgeons in 2007 while taking a year out from the middle of my Specialist Registrar training in Edinburgh. The hospital is situated 80km north of the capital, Kampala, seving a rural population of approximately 500,000, in the region known as the Luwero Triangle made infamous during the 1981-86 civil war when approximately 1/3 of the population was killed. The healthcare at Kiwoko began in 1988 when Dr Ian Clarke, an Irish GP, came to work there. From clinics held under a tree, and then in the local church vestry, a health centre and then a hospital were built. The hospital now has 7 wards – male and female (medical and surgical), paediatric, nutrition, TB, maternity and Neonatal Intensive Care – along with a twin theatre suite that is well equipped by Ugandan standards. It also has a nationally recognised Nurse and Laboratory Training School.

The hospital functions on a budget of only £600,000 per year. It is a Christian hospital that serves the needs of the local and extended community and although patients have to pay for their treatment, no one in need is turned away – a "Good Samaritan Fund" pays for them. However in reality, only 30% of the funding comes from patient fees, with 10% from the government, while 60% comes from outside agencies. Medical staff are in short supply in Uganda, with most doctors either leaving for South Africa or the West following their training, or preferring to stay in the capital where the money is. In 2007 Kiwoko was fortunate to have four Ugandan doctors and there were two other European doctors working alongside me. I was supported by UK-based charity Skillshare International both financially and practically throughout my year.

Why go to the Developing World?

There is a huge and increasing healthcare burden in the developing world. HIV/AIDS, TB, Malaria and other tropical diseases cause widespread health problems, but the more western diseases such as Diabetes, heart disease and cancer are becoming more and more common. Most doctors in these countries prefer to work in the cities, if not abroad, but the great majority of the population are rural and too poor to travel to these cities. Healthcare is often only available from nurses or clinical officers with limited training, so doctors who are prepared to work in these communities are of great benefit to the local people. For many doctors in the West, one of the initial reasons for wanting to become a doctor (albeit a fairly idealistic one formed at a young age!) was simply to help people, and an individual doctor can help a great number of people in the Third World, arguably many more than is possible back home.

However, a Western doctor in the Developing World is able to do far more than just treat people. It is possible to make a lasting effect on healthcare through the development of services and the training of local people. As the saying goes, buy a man a fish and you feed him for a day. Buy a man a fishing rod and teach him to use it and you feed a family for life! Local healthcare workers are usually extremely interested in being taught new skills and knowledge. Westerners also have a huge range of contacts and the ability to use tools like the internet to find donated equipment or other things that may just not be available in a Developing World country. I was able to train Ugandan doctors in Trauma management and critical care skills and develop their skills in Gastrointestinal surgery. I was able to get donations of fibreoptic endoscopy equipment and set up a complete endoscopy unit, I got a number of new surgical instruments brought out from the UK, and several companies donated many items of equipment including a number of automatic patient monitors and suture boxes. For the first time patients have been able to get diagnostic endoscopy without having to travel to the capital and the Ugandans doctors have enthusiastically taken to their new skills!

Ultimately, the person who possibly benefits most from a period working in the developing world is not the local people or the local doctors, but the doctor who decides to work there. The work is hard and the hours long, but the rewards are immediate and myriad. The variety of the workload is stimulating and exciting. Working with limited resources and a handful of textbooks provides the opportunity to develop operative skills and allow the confidence to cope with any circumstance. Most valuable of all is the genuine thanks and appreciation shown daily by patients and relatives who recognise that although things may not always go well, they see you doing the best you can with what you have available. It is also very humbling to see people not only surviving but thriving in situations of extreme poverty, and has a profound effect on your attitude towards possessions even if you would not describe yourself as materialistic.

CHANGING LIVES IN THE DEVELOPING WORLD

Stephen Bennett

When to go?

The traditional time for young doctors to travel and work abroad has usually been after housejobs, or more recently after foundation year two. This may be ideal for working as a junior in somewhere like Australia or New Zealand, but may not be the best time to go to the Developing World. The lack of other doctors means that you will need more knowledge and confidence than can usually be accumulated in the early years of training. A doctor going to Africa must be a help and not a burden on those around them.

A Surgeon, in particular, must have suitable operative skills to be able to cope with very unusual situations. However it is difficult to take time out of training before reaching Consultant level, and by that stage, many surgeons will feel themselves too specialised to be of use, or have other commitments such as family to consider. I was fortunate to have a Training Committee who allowed me to take a year out after SpR3, at which point I was comfortable operating independently in some areas and had operative skills which allowed me to take on operations I had never done before, and I would say it was an ideal stage to go and be useful.

The other consideration is how long to go for. Healthcare in the developing world is so different from Western hospitals that it can take over a month to get to the stage where you settle in and start becoming useful. As with every job, you become more useful with time. To get the most out of it, you should look to spend three to six months as a minimum. My twelve months allowed me time to assess how best I could develop services, make some changes and acquire equipment, and I would have accomplished far less had I gone for a shorter visit. One benefit of an initial long-term visit has been that I have been able to return for 10-14 day periods over the last couple of years and have settled straight back into the work requiring no re-acclimatization.

Where to go?

The Developing World is in such need of doctors that in theory you can go anywhere you want to! The main types of organisation would be Mission Hospitals, Government Hospitals, Private Hospitals and Non-Governmental Organisations (NGOs). Mission hospitals are found throughout the developing world and are often well run despite little resources. Many will take doctors of any faith or none, but you will have to be sympathetic to the hospital's status. These hospitals will often take short term volunteers but will usually be unable to provide recompense other than living accommodation.

Government hospitals are probably the hardest place to find a placement as local bureaucracy may often be unreceptive, and salaries may be non-existant. Private hospitals may look for junior doctors, but these are usually found in the main cities catering for wealthy individuals with western-type diseases, which may defeat the whole purpose of the visit. NGOs are often a good source of job, but usually require a significant amount of prior experience and may not be flexible in where you get sent as they often only work in major relief areas.

Who to go with?

Volunteering with smaller hospitals may be fairly easy, but many people will want some sort of funding and/or support from an experienced organisation, and I certainly benefitted from my association with Skillshare International. Other similar organisations would probably include VSO, MSF and Merlin. These organisations are used to the logistics of preparing for service overseas and can provide much needed in-country support.

Skillshare International were superb at preparation work including health checks, visa support, a training weekend, and financially paying for vaccinations, antimalarials, travel, insurance, equipment and a small monthly allowance. Missionary Organisations based in the UK will probably want a commitment to two or three years and may not therefore be as useful for the shorter-term volunteer.

CHANGING LIVES IN THE DEVELOPING WORLD

Stephen Bennett

Practical Considerations

There are many things to consider before going overseas, and even after returning. Health insurance, vaccinations and antimalarials are a must. Travel and possessions insurance may also be essential. Other arrangements such as pension contributions and National Insurance in the UK need to be sorted out in advance. Developing world experience is unlikely to be counted towards training time in the UK, but it may be possible to get recognition for some placements, and this should be determined months ahead of time. Even such simple things as arranging flights with an airline with a large luggage allowance can be very important.

Living in a Developing Country requires sensitivity to local customs and culture. Women may have to wear long skirts and cover their shoulders. Mission Hospitals may have a no-alcohol rule as drinking may be culturally unacceptable. Living in the midst of great poverty may require sensitivity to avoid being seen to spend large amounts of money.

Coming back home afterwards can also be hard. It is possible to make very deep friendships in a short space of time, and leaving these relationships is difficult. Settling back into Western Medicine takes time and hard work when you have become used to working with very limited resources and to looking after patients with very different diseases. I have found Uganda calling me to return with increasing insistence and I am sure I will go back to work there again in the future.

Conclusions

For the junior surgeon who has a heart for people, a desire to see healthcare in action in a very different context, and the desire to personally make a huge difference in peoples lives, a period of time spent working in the Developing World may be of great benefit. A year out of training may help formulate career aspirations and develop skills to a much greater stage than may occur in a similar period at home. Although many possibilities for this sort of work exist, going with a volunteer organisation to a well organised rural hospital may provide the best opportunity for surgery and development. Planning ahead is essential and volunteer organisations will help with many practicalities. However, working closely with your training committee will help make the time fit into your training sensibly. From my own experiences, I can strongly recommend working in the Developing World during Specialist Training.

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More information about Kiwoko Hospital and its work can be found in "The Man With The Key Has Gone" by Dr Ian Clarke, "There's A Snake In My Cupboard" by Dr Nick Wooding, and from the Friends of Kiwoko Hospital Website, **www.fokh.org.uk.** For more on the realities of day-to-day life of a surgeon in Africa, see my blog, **www.aboutlife.com/steveinafrica.** Skillshare International's website **www.skilshare.org** may be helpful to see what volunteer organisations can offer. The BMA also have a useful guide to working overseas including specific information on the developing world **(http://www.bma.org.uk/international/working_abroad/broadeningyourhorizons.jsp).**

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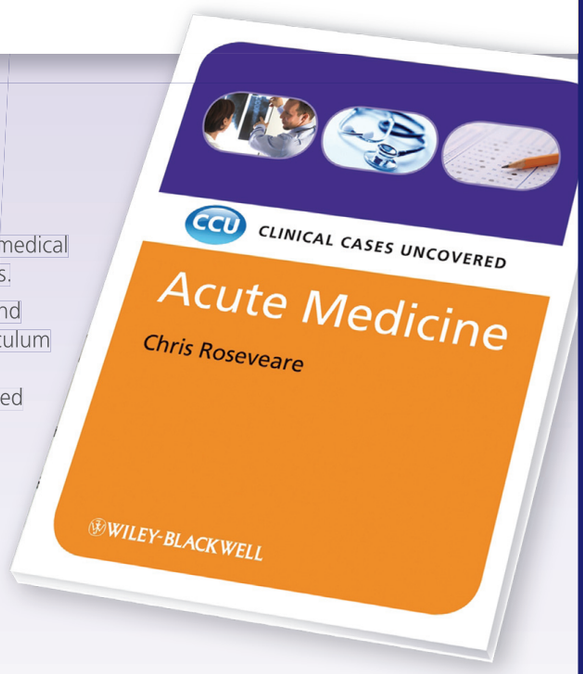

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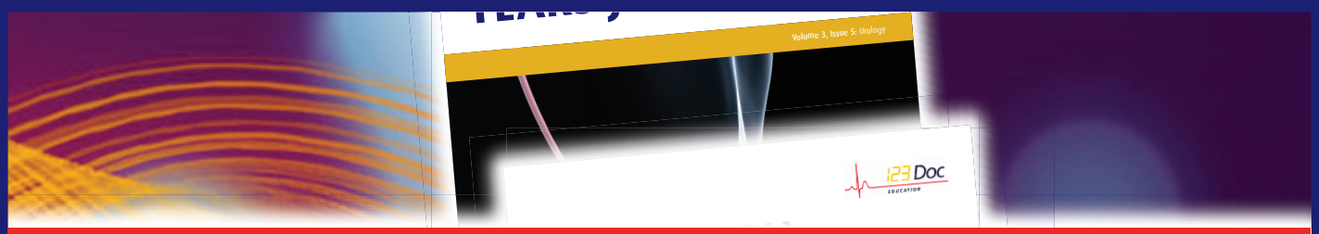

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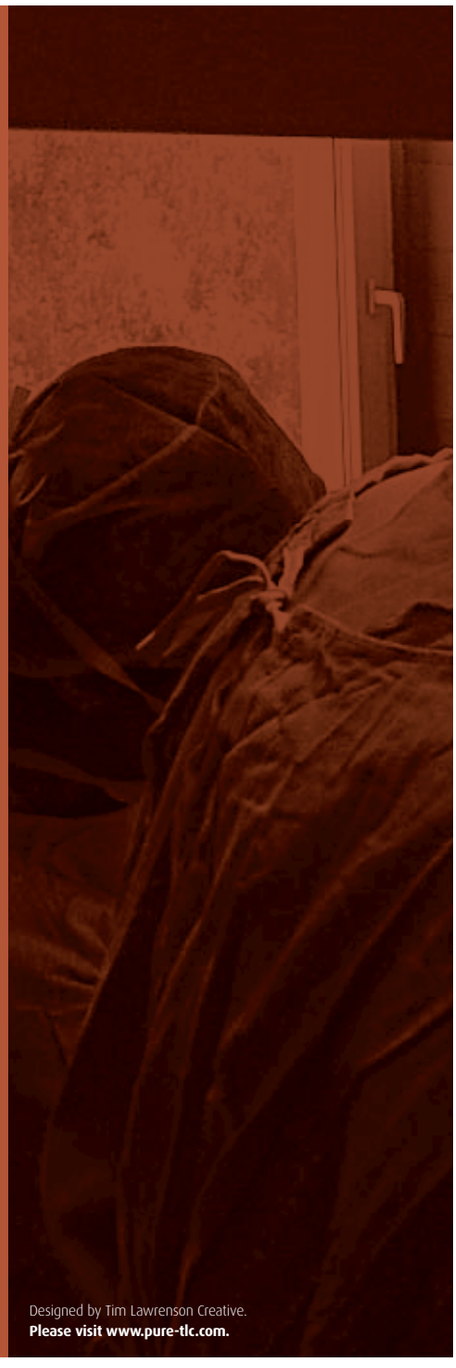

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