123 Doc EDUCATION

CORE SURGERY JOURNAL

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Contents

CORE SURGERY JOURNAL

Volume 1, Issue 5

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CORE SURGERY JOURNAL

Volume 1, Issue 5

Dear Prospective Authors

Thank you for considering the submission of an article to 'Core Surgery'. This is a new journal aiming to educate and inform surgical trainees about relevant 'core' subject topics. Each issue will cover a topic from selected subspecialty fields: General Surgery, Trauma and Orthopaedic Surgery, Plastic and Reconstructive Surgery, Otorhinolaryngology and Neck Surgery, Neurosurgery, Urology, Paediatric Surgery, Cardiothoracic Surgery and Critical Care. 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. Authors are encouraged to submit articles on relevant topics to core surgical training.

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

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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; details can be found at **http://www.imperial.ac.uk/Library/pdf/Vancouver_referencing.pdf.** 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.**

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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

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CORE SURGERY JOURNAL

Volume 1, Issue 5

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.

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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.

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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 surgical trainees. We wish you every success with your submission. Please contact the editorial team with any questions.

TOURNIQUETS

J Turner

Tourniquets.
Back to Basics.

Tourniquet use in limb surgery is commonplace today. This article discusses the background, basics and problems associated with tourniquet use. The following two cases demonstrate two patients who suffered complications associated with tourniquet use.

Case 1

A 72 year old male was admitted for an elective left total knee replacement for severe osteoarthritis on a background of peripheral vascular disease (PVD). The patient reported a claudication distance of 20 yards and smoked 20/day for the past 50 years. On examination he had pale, hairless, cold legs with no evidence of ulceration, impalpable peripheral pulses with a biphasic flow on Doppler and an ABPI of 0.6. With these findings in pre-op assessment clinic it was decided by the orthopaedic consultant that a tourniquet would not be used for his operation in light of his severe PVD.

Unfortunately, perioperatively the patient had a significant amount of blood loss and hypotensive episodes requiring fluid challenges and ionotropes with postoperative monitoring in the surgical step-down unit. The patients Hb dropped from 13 g/dl preoperatively to 8 g/dl postoperatively and subsequently had 3 units transfused. Three days later, whilst still in step-down unit, the patient started spiking temperatures of 38-39^{o}C with a swollen, sore and erythematous left knee. He was cultured and eventually taken back to theatre to have his knee washed out. The infection did not resolve and day 5 post-op he was taken back to theatre for the third time to have his TKR revised. After careful consideration, it was decided to use a tourniquet on the revision regardless of the PVD and infected tissue, in light of the patient's previous perioperative blood loss.

Case 2

A 25 year old female was referred to the orthopaedic outpatient clinic from her GP complaining of severe episodic left knee pain made worse by weight bearing, associated with intermittent mechanical locking symptoms since her skiing accident 2 months previously. She had no significant comorbidities and on examination Apley grind test was positive on the left with negative drawer and collateral ligament stress tests. The orthopaedic registrar suspected a medial meniscal tear injury and the patient was subsequently listed for a routine knee arthroscopy with the plan for tourniquet use perioperatively.

The knee arthroscopy was successful with the trimming of a small radial meniscal tear undertaken. The tourniquet time was ~18 minutes with a limb occlusion pressure of 350mm Hg used. Postoperatively the patient complained of prolonged dysesthesia and limb weakness. It soon became apparent that sensory, motor and propioceptive functions in the lower leg were diminished. A nerve conduction study demonstrated a significantly diminished conduction time and 12 months on, the patient has still not regained any neurological function and has significant muscle wasting of the lower leg.

It should be noted that pressure related neurological injury is exceedingly rare in short term tourniquet use and traditionally a tourniquet is used for up to 2 hours. However, the case does highlight the potential for nerve injury and therefore the importance of this consideration when consenting for and using tourniquets.

Tourniquet history

Tourniquet comes from the French verb 'tourner' which means to turn, as the first tourniquets required screwing up and were first used to create a bloodless surgical field in 1864 by Joseph Lister (see figure 1). It wasn't until 1904 that Harvey Cushing developed the first pneumatic tourniquet.[1] Pneumatic tourniquet use has become routine in most operations involving the upper and lower extremities in orthopaedics and regional IV anaesthesia in anaesthetics.

Modern tourniquet structure and design

Modern pneumatic tourniquets have three basic components, a cuff secured to the limb proximal to the operative field, a pressure source that is under microprocessor control and an instrument capable of measuring, displaying and controlling the pressure exerted on the limb.

There are numerous different cuffs available today raging from cylindrical standard cuffs, tapered conical cuffs (contoured cuffs) and wide or narrow cuffs. It has been demonstrated that wider contoured cuffs (see figure 1) provide an equivalent limb vessel occlusion to cylindrical cuffs at lower inflation pressures due to a better fit of cuff and more efficient transmission of pressure, thus reducing the chance of pressure related tourniquet injuries.[1,2,3,4]

TOURNIQUETS

J Turner

Figure 1: Modern day contoured cuffs

Limb occlusion pressure (LOP) is defined as "the minimum pressure required, at a specific time by a specific tourniquet cuff applied to a specific patient's limb at a specific location, to stop the flow of arterial blood into the limb distal to the cuff."[1] Most modern pneumatic tourniquets calculate this LOP at the beginning of the operation with an automated plethysmographic system. The pressure applied is that of the LOP plus a standard pressure safety margin (as defined by AORN: the use of pneumatic tourniquets[2]) accounting for physiological variances and changes anticipated during the procedure.[2] The use of LOP as a guide to cuff pressure has been shown to significantly lower the pressure applied during an operation as compared to tourniquet systems using standard preset values, thus lowering the risk of pressure related injuries.[1]

Tourniquet associated complications

Surgical tourniquets, although essential in providing a bloodless surgical field, have numerous associated complications[1,2,3,5,6]. A recent Norwegian study has reported the incidence of tourniquet associated complications at 0.028%, however this study excluded compartment syndrome from its analysis and failed to report post tourniquet compartment syndrome[12]:

1) Nerve injury was reported at 0.024% in the above study.[12] Pressure related dennervation can range from a mild transient loss in specific nerve functions to permanent irreversible damage. The nerve damage occurs mostly secondary to mechanical stress across the nerve, usually at the edges of the cuff, and less frequently due to ischaemia of the nerve.

2) Post tourniquet syndrome is the commonest tourniquet associated complication and is defined as the postoperative swelling, weakness and numbness of limbs after tourniquet release initially due to post-ischaemic reactive hyperaemia and later due to oedema and haematoma formation. Risk factors for developing post tourniquet syndrome include increased duration of application and incorrect cuff pressure occluding venous outflow but not arterial inflow (usually due to calcification of arterial vessels in diabetes and patients on long-term steroids).

3) Despite application of a tourniquet, intraoperative bleeding can be a significant problem with a lack of venous return due to a number of factors; incorrect application of cuff or selection of appropriate cuff, slow insufflations of the cuff, incomplete exsanguination of the distal limb prior to cuff application and intramedullary nutrient vessel bleeding.

4) Compartment pressure syndrome can occur secondary to tourniquet use in any limb compartment that a tourniquet has been applied to. It occurs due to a combination of prolonged external compression resulting in increased capillary permeability secondary to tissue ischaemia, and an increase in intra-compartmental contents due to the surgery itself.

5) Whilst the tourniquet is inflated, metabolic waste products in the ischaemic limb accumulate, including lactic acid, potassium, carbon dioxide and myoglobulin due to rhabdomylosis. On deflation of the cuff these metabolic waste products are released into the systemic circulation resulting in a fall in mean arterial pressure, tachycardia and a transient renal impairment. These effects in fit young patients will be insignificant, but frail elderly patients with significant comorbidities may not compensate so well.

6) Toxic reactions to local anaesthetics can occur in IV regional anaesthetics (Biers block) due to the accidental release of a bolus of LA into the systemic circulation resulting in drowsiness, respiratory depression, tinnitus, bradycardia, hypotension and eventually death. The major causes of such reactions are usually due to technical errors or at the end of an operation when the tourniquet is deflated resulting in release of metabolic waste products and LA.

TOURNIQUETS

J Turner

Tourniquets.
Back to Basics.

There are numerous other tourniquet associated complications reported, some of which include the following:

- Digital necrosis secondary to prolonged limb occlusion.
- Pressure sores due to a combination of shearing forces and prolonged pressure.
- Chemical burns due to the close application of chemical prep solution to a patient's skin under the tourniquet.
- Increased risk of developing deep venous thrombosis
- Tourniquet pain during and post application due to a combination of cuff pressure and post-ischaemia reactionary hyperaemia.
- The heat from surgical lights and instruments cannot be dissipated along the full surface area of the limb when a tourniquet is applied resulting in thermal damage.

Most of these tourniquet associated complications can be prevented by applying the correct, wide contoured cuff to the patient, ensuring complete exsanguinations of the distal limb, using the correct LOP for that patient, avoiding shearing and twisting of the cuff perioperatively and minimising the duration of occlusion with 15 minute pauses in cuff insufflation every 60 minutes[2,3,6].

Tourniquet contraindications

The relative contraindications for tourniquet use in surgery are discussed in table 1:

Relative contraindication	Pathophysiological justification
Extremity infection	Embolisation of septic material can occur during exsanguination of the limb.[3]
Open Fracture and crush injuries	Increased risk of developing compartment syndrome without tourniquet.
Tumour distal to the tourniquet	Embolisation of tumour material can occur during exsanguination of the limb.[3]
Increased intracranial pressure	Release of carbon dioxide on cuff deflation results in a 50% increase in cerebral perfusion.[7]
Peripheral vascular disease	Tourniquet pressure can rupture atherosclerotic vessel plaques resulting in vessel occlusion.[8]
Previous revascularisation of the extremity	More susceptible to arterial occlusion when pressure applied to extremity.[3]
Extremities with dialysis access (AV fistula)	Prolonged venous occlusion increases the risk of developing a fistula thrombosis.
Sickle cell anaemia	Stasis, hypoxia and acidosis in the distal limb can precipitate a sickle cell crisis.[9]
High risk of venous thromboembolism	Studies have demonstrated an increased risk of postoperative DVT following tourniquet use.[10]
Acidosis	Release of lactic acid on cuff deflation can worsen any pre-existing acidosis.
Left ventricular failure and severe hypertension	Bilateral limb exsanguination and tourniquet insufflation increases circulating volume by 15%.[11]
Diabetes and long-term steroid use	Require higher LOP than normal due to calcified vessels.[3]
Skin grafts	Increased risk of skin trauma with the use of tourniquets, may irreversibly damage grafts.

Table 1: Relative contraindications to tourniquet use and their pathophysiological justifications

Discussion

Case 1 describes a patient with severe peripheral arterial disease who required a total knee replacement. The case demonstrates the relative contraindications of tourniquets, including peripheral arterial disease and extremity infection (see table 1). It also demonstrates the risks of not using tourniquets and how the pros and cons for using a tourniquet have to be weighed up on a case by case approach to ensure that the best decision is made for each patient.

Case 2 describes a patient who underwent a routine 20 minute knee arthroscopy with a tourniquet inflated to an average 350mm Hg. It demonstrates that regardless of all the guidelines and the current understanding of tourniquet associated complications, post procedure injuries such as tourniquet associated dennervation can still occur. This case demonstrates the unpredictability and potentially significant morbidity associated with tourniquet complications and how regardless of the age, health and routine nature of the procedure being undertaken, the dangers of tourniquet use should never be underestimated.

Conclusion

Tourniquets have been used in surgery since the mid 1800s and are very effective at creating a bloodless surgical field for safer and faster operations. However, there are significant complications associated with the use of tourniquets requiring vigilant observation, practice and preoperative assessment of every patient with a case by case approach.

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TOURNIQUETS

J Turner

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MCQs

Regarding the use of tourniquets, state whether the following are true or false:

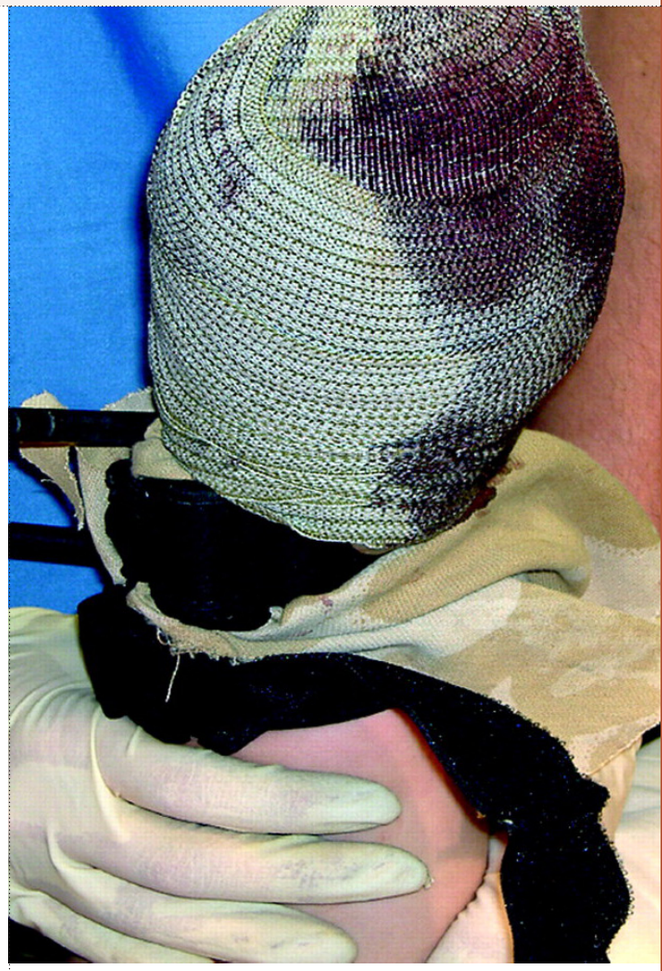
1) Nerve damage occurs most commonly due to ischaemia of the nerve.

2) Relative contra-indications include extremity infection.

3) Increased intra-cranial pressure is an absolute contra-indication.

4) Risk factors for developing post tourniquet syndrome include increased duration of application and incorrect cuff pressure occluding venous outflow but not arterial inflow.

5) Skin grafts are an absolute contra-indication for the use of tourniquets.



Answers:

1. F

2. T

3. F

4. T

5. F

Author

Jake Turner
F2 East Midlands Deanery
jake.turner@nhs.net

INGUINAL HERNIA REPAIR

K Mann

Inguinal Hernia Repair.
General Surgery.

Aetiology & Pathology

The inguinal region is a commonly operated upon and is a staple operation for a surgical trainee. Its anatomy is complex and the layers of the abdominal wall converge to form its walls. An inguinal hernia is a protrusion of viscera or tissue from the abdominal compartment into the inguinal compartment. The basic principles of hernia development are that of increasing intra compartmental pressures and weakness of the tissues between compartments. [1][2]

Inguinal hernias can occur in children and peak between infancy and childhood. 3-5% of newborn children may have an inguinal hernia. In adults there is preponderance to males of the ratio 12:1 to women. They occur most commonly in patients within their sixth decade of life. 65% are direct in nature and are four times as likely to present bilaterally than with indirect hernias. [1]

Type	Definition
Reducible	A Direct hernia protrudes directly through the posterior wall of the inguinal canal, medially to the epigastric vessels. An Indirect hernia emerges through the deep ring, lateral to the epigastric vessels and travels along the canal.
Irreducible	An incarcerated hernia that presents as a hard tender inguinal mass.
Obstructed	An irreducible hernia with obstruction of the bowel lumen with symptoms of nausea, vomiting, distension and not passing flatus.
Strangulated	An irreducible hernia that may contain bowel or omentum with compromised blood supply.

Table 1; Definitions and types of inguinal hernias [1]

Anatomy learning tips

As we first assist and begin to perform hernia repairs it is difficult to truly appreciate the anatomy of the inguinal canal. The canal is a 4cm tunnel in the anterior and inferior abdominal muscles with deep and superficial rings. It is essentially a combination of abdominal muscle wall layers that merge to form the layers of the canal. Moving from lateral to medial the walls change in layer composition. There are many descriptions and diagrams to explain this unique anatomical region that provide more detail that will be offered here. The single most helpful tool to understand the anatomy is the operation itself. It is important to concentrate while assisting in order to apply learnt principles to the operator's dissection. During emergency procedures the anatomy may become distorted and requires greater understanding of the structures involved. [3]

Wall	Composition	
	Medial	*Lateral*
Anterior	Aponeurosis of external oblique	Aponeurosis of external and internal oblique
Posterior	Conjoint tendon	Conjoint tendon
Superior	Internal oblique and transversus abdominis	Internal oblique and transversus abdominis
Inferior	Inguinal ligament and lacunar ligament	Inguinal ligament and iliopubic tract

Table 2; Anatomical layers of the inguinal canal [3]

Clinical features

As we can see from the above definitions, a patient may present at various stages of inguinal hernia pathology. The initial symptoms of a newly occurring hernia are pain and swelling, precipitated by actions that cause increased abdominal pressure. The pain or swelling may persist or resolve without intervention. The hernia may protrude easily and may reduce spontaneously or require manipulation. Exquisite tenderness can occur with incarcerated, obstructed or strangulated hernias. [1]

There are key clinical features that need to be established upon clinical presentation;

Is there a hernia? Is it inguinal or femoral?

The history is key to the diagnosis and the clinical examination should confirm the diagnosis. Examination should be performed standing and lying flat. The exact position of the swelling should be elicited in relation to the pubic tubercle and the inguinal ligament and a contralateral hernia should be examined for.

Is it a complicated hernia? What is its nature?

The degree of protrusion should be assessed by examining the scrotum for inguinal scrotal development. Reduction of the hernia should be attempted and if it is irreducible then an assessment of contents should be made. Bowel sounds maybe auscultated for and an abdomen x-ray should be performed to assess for obstruction. [1]

INGUINAL HERNIA REPAIR

K Mann

Inguinal swellings
Inguinal hernia
Femoral hernia
Haematoma
Lymph node
Inguinal abscess
Hydradenitis suppurativa
Pseudoaneurysm
Tumour
Undescended testicle

Table 3; Differential diagnosis of an inguinal hernia [4]

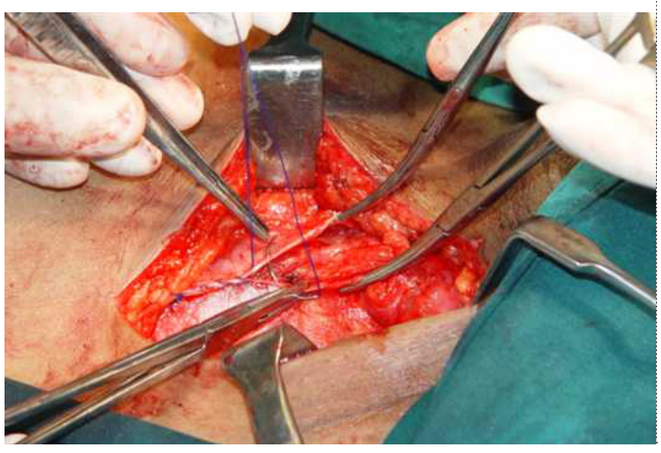
Tips

The position of the hernia is important in delineating its origin. If it lies medial and inferior then it is likely to lie inferior to the inguinal ligament and to be femoral in origin. If it lies lateral and superior then it is likely to be inguinal. It is difficult to assess clinically for a direct or indirect hernia but is not important because it has no affect on the decision to operate.

Reduction of hernia can be very difficult and may decide whether an operation is necessary. It is often advisable for the patient to reduce the hernia because they may be accustomed to doing it regularly. For difficult cases the patient should lie in the Trendelenburg position and have good analgesia before reduction. In any case it is worth attempting but often if the patient fails then it is unlikely to reduce and forceful attempts should be avoided.

A patient may present for the first time with inguinal pain and a reduced hernia. There may be tenderness on examination but a swelling may not be elicited. Initially on the development of a hernia the tissues may cause pain but this settles with time as the defect becomes established. A cough impulse is not diagnostic of a hernia as it may be elicited in normal patients.

The contents of the hernia are often omental fat or small bowel loops. If it is an incarcerated hernia then bowel sounds may be absent despite containing bowel. It is essential to ensure the patient is not obstructed because the operation may involve a small bowel resection.



Management and Operative Indications

The management of an inguinal hernia ranges from conservative management to an emergency operation. If the patient has a reducible hernia and the pain can be controlled with basic oral analgesia then it can be repaired electively. There is a case for semi-elective repair if the patient is struggling with pain or the size of the hernia and is impinging on a normal lifestyle. Indications for emergency operative management are an incarcerated or strangulated inguinal hernia. If there are diagnostic difficulties then an ultrasound may be helpful in delineating the source of the swelling.

The principles of the operation are to restore the integrity of the musculo-aponeurotic inguinal canal and preserve the cord structures. There have been many described open approaches but over the last decade a mesh repair has been proved to have the smallest recurrence rates. This can be done under a local anaesthetic for patients who are deemed unsafe for a general anaesthetic. This can be achieved with an ilioinguinal nerve block together with local wound infiltration. A laparoscopic hernia repair can be done either intraperitoneally or totally extraperitoneal prosthetic repair (TEPP). The indications for this operation are for recurrent hernias or for bilateral hernias. This is rarely done in an emergency situation and is beyond the scope of this article. [1][2]

Consent

Consent should only be gained from trainees able to perform the operation. Consent for an inguinal hernia repair involves discussion on the procedure, haemostasis, pain control, and infection risks. The patient should be made aware of the recurrence rate (0.77% [2]) and the need for further operations. Damage to the ilioinguinal nerve may result in persistent pain and numbness at the wound, which may eventually resolve. An important complication is that of an infected mesh which will require antibiotics and emergency removal. Highlighting the risk of vascular damage to the testicle, especially with recurrent hernias, may be worth discussing.

Operative Preparations

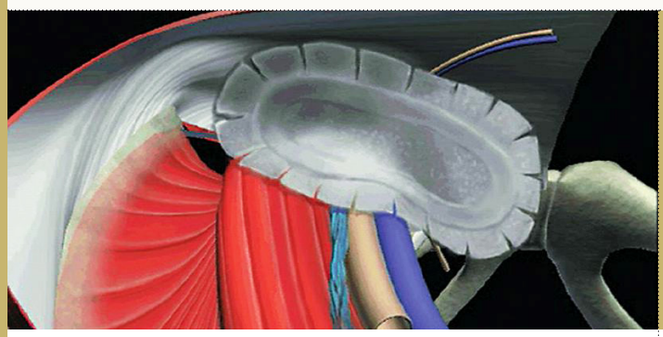
The open repair is generally a day case procedure but may require longer admission in the emergency repair. The patient is given a general or a local anaesthetic and placed supine. A basic general surgical tray is utilised for this operation and a trainee should be familiar with the instruments and their applications. [2]

Before patient preparation a patient safety check should occur between surgeons and theatre staff including the operation and patient allergies. Draping the open mesh repair involves creating a square site with the inguinal canal adequately exposed. Draping should expose the anterior superior iliac spine and the pubic tubercle and at least 5cm above and below these landmarks.

Some surgeons prefer to give antibiotics prophylactically but there is no evidence to support this.

INGUINAL HERNIA REPAIR

K Mann



Inguinal Hernia Repair.
General Surgery.

The Lichtenstein Repair

This is not a detailed account of the procedure, which can be found in many operative textbooks but basic principles and advice on general difficulties.

The incision employed for this operation begins medially at one fingerbreadth above the pubic tubercle. It runs along the skin crease towards the anterior superior iliac spines for approximately 5-7cm. During dissection it is important to recognise Scarpa's fascia and the aponeurosis of the external oblique, which directs you towards the external ring. The fibres are separated and cut, opening the anterior wall of the inguinal canal. [2]

It is at this stage that the avascular plane between wall and cord structures is found using blunt dissection, most commonly with a finger. The cord needs to be mobilised and placed in a hernia ring. The cord is displaced superiorly to reveal the posterior wall where a defect or a bulge may be present. Cord exploration involves incising the cremasteric fibres while holding the cord between thumb and index finger. The hernia sac is often a white curved structure, which may be peeled towards the abdominal cavity or dissected with sharp instruments. Once the sac is separated it can be opened and the contents reduced. The sac is twisted at its base and after ensuring the contents have reduced, transfixed and cut. If it is a direct hernia then the defect may require plicating before mesh placement. [1][2]

Before mesh placement the external oblique fibres should be separated from the internal oblique muscle. The mesh should be trimmed into the characteristic shape with a curved medial end with wide and thin tails. The curved end is anchored to the pubic tubercle and the thin tail is sutured to the inguinal ligament with a continuous suture, taking care of the major femoral vessels below. The wide tail should be sutured deep to the external oblique using interrupted sutures within the created pocket. The cord should sit between the slit of the wide and thin tails. The ends of the tails are then overlapped and sutured creating a new internal opening. There must be sufficient overlap medially, superiorly and laterally with well placed sutures for a good repair. The external oblique fibres should be closed with a continuous suture to reform the external canal. The cord should be sitting snugly in the opening and then a layered closure is performed. [2][3]

Tips

When dissecting the external oblique aponeurosis, care should be taken to avoid the ilioinguinal nerve, which lies just underneath. Once visualised it should be kept away from the rest of the dissection and preserved if possible.[2]

Locating the avascular place between cord and canal can be difficult at first but the finger is the best tool for the job. Your finger should sweep along the inguinal ligament and along the posterior wall under the cord. This is done similarly above the cord with the contralateral finger, alternating sweeps until the tips of the fingers meet under the cord, revealing the plane.

Examining the floor of the canal is important to find a direct defect. If there is nothing obvious then the cord needs careful exploring to find an indirect hernial sac. There is no excuse for missing a direct defect or an indirect sac.

When beginning to perform hernia repairs it is incredibly difficult to dissect the cord structures in order to find a sac. The vas deferens is an important landmark to feel, as it is the structure that lies most posteriorly. Our eyes become trained to visualise the white layer of peritoneum with experience. Careful dissection, avoiding sharp instruments, by gently parting cord structures may be helpful until confidence increases.

Large long-standing sacs and combined hernias can be extremely difficult to isolate and repair. The aim is to deal with each sac separately. This can lead to inadvertent damage and great care is needed. It is important not to continue if you are out of your depth and call for senior assistance. [2]

When transfixing the base of the emptied sac it is important to resect the peritoneal sac before cutting the ligature. This is to keep control of the base before it falls into the abdominal cavity to ensure the suture is secure and the peritoneal edge is not bleeding.

It is important that a pocket is created under the external oblique so that the mesh sits safely and that there is adequate overlap. As we develop our experience we will develop our preferences on how and when to shape the mesh. When suturing the mesh in place it is important to place the suture at varying depths on the ligament fibre to prevent them from splitting.

INGUINAL HERNIA REPAIR

K Mann

When performing an inguinal hernia operation in female patients, it is important to remember that the cord contains the round ligament. This has little importance and can be resected. The operation is simpler and the sac may be easier to locate.

In an emergency situation the operation can be very difficult. Great care is needed to locate the sac and explore its contents. There may be an adherent omentum, which can be ligated and resected safely without causing complications. If there is bowel contained within the sac, it must not be allowed to fall into the abdominal cavity before its viability is assessed. This can be done by inspection and manipulation to observe its colour and peristalsis. Senior help should be sought if there is any doubt or if a resection is required. However this is rare and the basic principles of hernia repair apply.

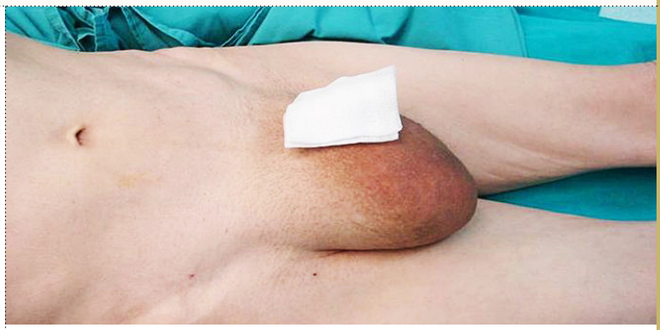
Recurrent inguinal hernias are difficult procedures to perform, especially if we are unsure of how the initial operation was performed. A recurrent inguinal hernia often occurs medial to the mesh placement. The incision should be placed along the same wound but made longer in order to begin dissecting with virginal tissue planes. This aids in dissecting through fibrous tissue from the previous procedure. With experience the inguinal canal structures and sac can be dissected out through difficult tissue planes and a formal repair carried out. An alternative approach to the hernia repair may be the best option.

Post Operative Management and Complications

The operation note should highlight the type of hernia uncovered. Unless the patient had a small bowel resection the post operative period is similar for elective and emergency operations. Once the patient has recovered from the anaesthetic they are encouraged to mobilise. If their pain is controlled with oral analgesia then they can be discharged. Generally they are not allowed to drive or perform rigorous exercise for 4-6 weeks.

Early complications include haematoma and infection. Superficial would infections can be managed conservatively with antibiotics or operatively. If there is suspected mesh infection then the mesh should be removed. A haematoma may form from inadequate haemostasis or due to the patient's medications and will require evacuation. The scrotum may enlarge and appear bruised. There may be a haematoma or a hydrocoele underlying but both should resolve spontaneously without intervention. Ischaemic orchitis or damage to the vas deferens is rare. [1]

Long-term complications involve, recurrence, chronic pain and numbness. Numbness can occur medial and inferior to the wound for a prolonged period of time without disability. However chronic pain is more of a concern and may require regular analgesia and a pain team review. A recurrent hernia will require further repair. An open operation may reveal severely adherent tissues and so a laparoscopic repair is recommended.



Laparoscopic Hernia Repair

This has become a popular first line operation for unilateral inguinal hernia repairs. The intraperitoneal approach allows direct visualisation of the hernia, blunt dissection of the sac from the abdominal wall, and mesh placement. It is easier to appreciate the anatomy than with a TEPP repair. [1][2]

The TEPP approach requires a firm grasp of the anatomy as it is viewed at an angle rarely seen. The space between the posterior rectus sheath and the peritoneum is enlarged by the insertion of a balloon through a paraumbilical incision. The key to operating in this space is to focus on the inferior epigastric vessels. Blunt dissection is performed medial and lateral to the vessels to find peritoneum, which may be part of a sac. The sac is reduced with blunt dissection and a mesh can be placed within the space. [1][2]

Both procedures require experience with laparoscopy and a sound knowledge of the anatomy. They each carry their own risks and benefits.

MCQs

Which of the following statements are true?

1. The femoral canal

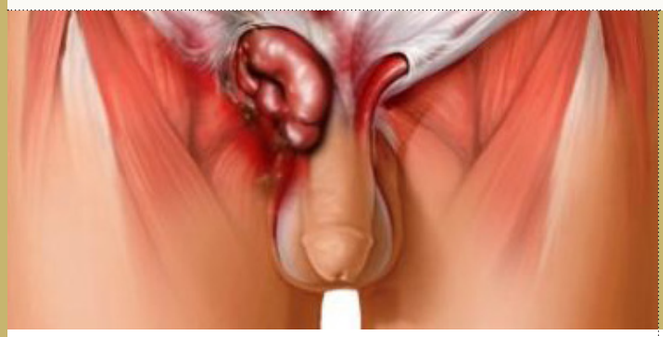
(a) Lies lateral to the femoral vein
(b) Has the inguinal ligament as its anterior border
(c) Has the lacunar ligament as its lateral border
(d) Has the pectineal ligament as its posterior border
(e) Contains the lymph node of Cloquet

2. With regards to Inguinal hernias:

(a) A direct hernia passes through the deep inguinal ring into the inguinal canal.
(b) A femoral hernia is more common than an inguinal hernia in females.
(c) An inguinal hernia can be distinguished from a femoral hernia by its relationship to the inguinal ligament.
(d) The inferior epigastric vessels lie medial to the deep inguinal ring.
(e) The floor of the inguinal canal is formed by the conjoint tendon.

INGUINAL HERNIA REPAIR

K Mann



Inguinal Hernia Repair.
General Surgery.

3. A 60-year-old male presents with an inguinal hernia of recent onset. Which of the following statements are TRUE?
(a) The hernia is more likely to be direct than indirect.
(b) Presents through the posterior wall of the inguinal canal, lateral to the deep inguinal ring.
(c) If direct is more likely to present with bilateral hernias.
(d) Is more likely than a femoral hernia to strangulate.
(e) The sac is congenital.

4. A 65-year-old female requires emergency surgery for a strangulated inguinal hernia. Which of the following is correct?
(a) The sac is formed by an unobliterated processus vaginalis.
(b) The hernia is direct rather than indirect.
(c) Such herniae never contain small intestine.
(d) Strangulation never results in bowel ischemia and gangrene requiring resection.
(e) Indirect inguinal herniae are never found in female patients.

5. The following structures may be injured during surgery to repair an inguinal hernia:
(a) The ilioinguinal
(b) The femoral nerve
(c) The superior epigastric artery
(d) The nerve to the psoas major muscle
(e) The genitofemoral nerve

Answers:

1. B, D, E
2. D, E
3. A, C
4. B
5. A, E

Viva questions

1. What are the boundaries of the inguinal canal?

2. What are the contents of the inguinal canal?

3. What are the boundaries of the femoral canal?

4. Name the different types of inguinal hernia repair?

5. What are the complications of an inguinal hernia repair?

6. Describe the Leichtenstein repair of an inguinal hernia.

7. Describe the ways you can repair a femoral hernia.

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[1] Paterson-Brown, S (2009). Core Topics in General and Emergency Surgery, Fourth Edition. Saunders, Elsevier.

[2] Lowe, D (2006). Surgical Pathology Revision, Second Edition. Cambridge University Press.

[3] Whitaker RH & Borley NR (2005). Instant Anatomy, Third Edition. WileyBlackwell.

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Author Details

Kulbir Mann
General Surgical Registrar,
Frimley Park Hospital,
Portsmouth Road,
Frimley, Surrey, GU16 7UJ.

Email: Dr.kooliebear@gmail.com

INGUINAL HERNIA REPAIR

K Mann

Operative photos

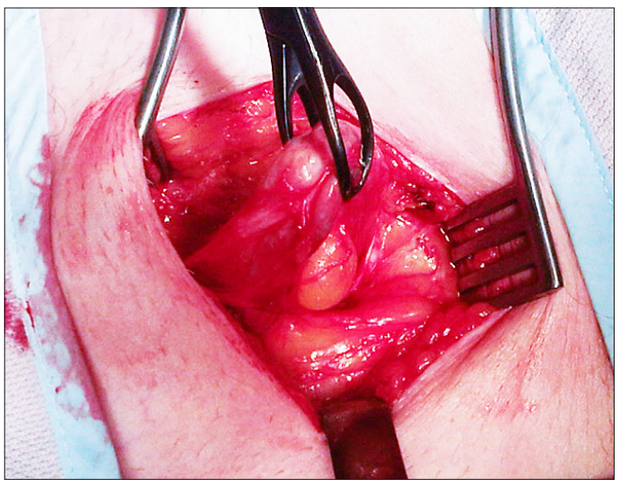


Figure 1; Dissection of the inguinal canal, cord contents held in Lanes forceps

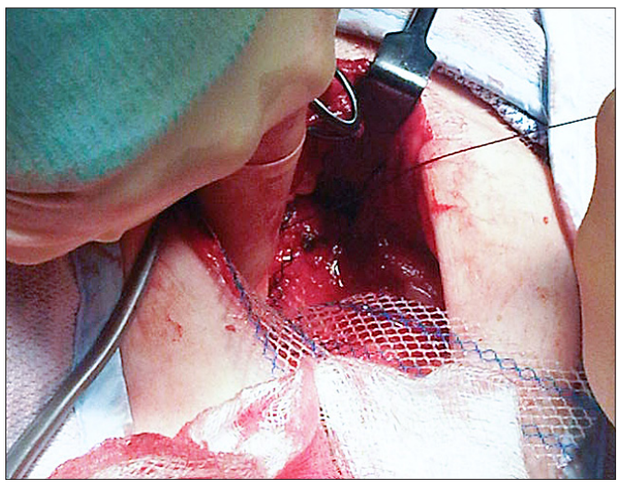


Figure 3; Placing the mesh on the posterior wall, first suture to pubic tubercle

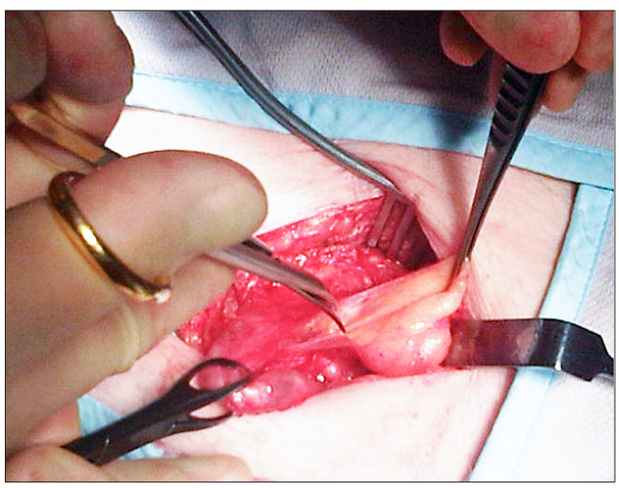


Figure 2; Cord contents retracted to left, dissection of sac with McIndoe scissors

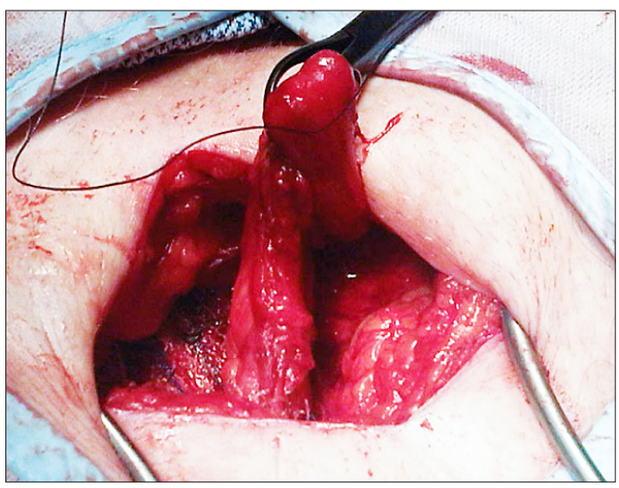
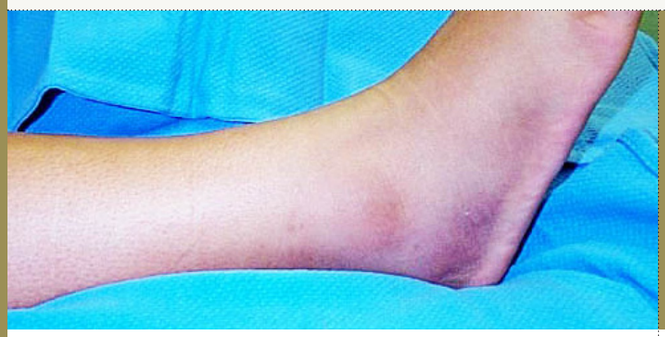


Figure 4; Mesh in situ with cord contents retracted above.

ANKLE FRACTURE: ANATOMY, CLASSIFICATION AND INJURY MECHANISM

V P Patel



Ankle Fracture:
Anatomy, Classification and injury Mechanism.
Trauma & Orthopaedic Surgery.

Case vignette

A 38 yr old female slipped on ice sustaining an isolated twisting injury to her ankle. She was taken to A+E and you are the Orthopaedic doctor on call. On examination she is unable to weight bear and has obvious bruising, swelling and tenderness, both on the lateral and medial aspect of her ankle. She has no distal neurovascular deficit. Initial radiographs of her ankle are shown below (figure 1).

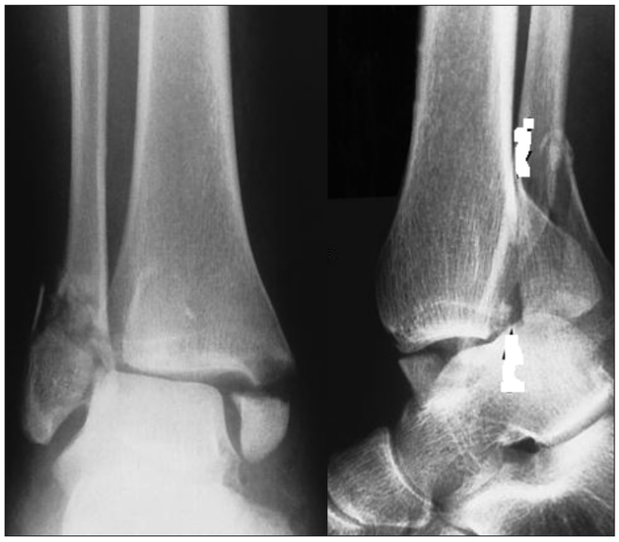


Figure 1: Mortise and Lateral Radiographs demonstrating a trimalleolar ankle facture with talar shift.

What do you suspect was the mechanism of injury?

Functional anatomy:

Ankle stability is conferred by bony architecture, ligamentous and capsular structures. To understand injuries that occur around the ankle joint a good knowledge of the fundamental soft-tissue structures that surround it is essential.

The deepest layer is the capsule containing the ligaments of the ankle; the middle layer includes the tendons, which traverse the joint to reach the foot; and the most superficial layer is made up of the fibrous bands (retinaculi), which hold the tendons in place as they act on the foot.

There are three distinct groups of ligaments supporting the ankle joint: (a) the syndesmotic ligaments; (b) the lateral collateral ligaments; and (c) the medial collateral ligament (Deltoid) (figure 2).

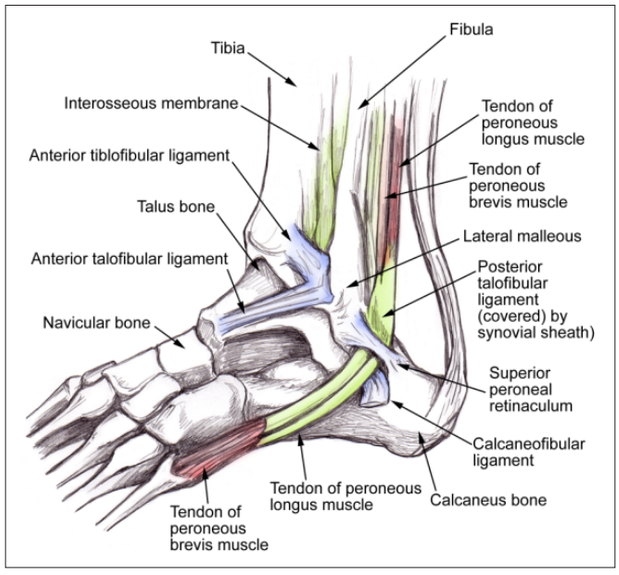


Figure 2: Diagram of ankle structures

Image reprinted with permission from Medscape.com, 2011. Available at: **http://emedicine.medscape.com/article/91344-overview**

a) Syndesmotic ligaments:

The syndesmosis is composed of the interosseous ligament that connects the tibia and the fibula throughout their entire length. This ligament is strengthened inferiorly by two thickened fibrous bands: the anterior inferior tibiofibular ligament and the posterior inferior tibiofibular ligament.

ANKLE FRACTURE: ANATOMY, CLASSIFICATION AND INJURY MECHANISM

V P Patel

The distal lateral border of the tibia is concave, with anterior and posterior tubercles. The anterior tubercle is the site of origin of the anterior tibiofibular ligament, and the posterior tubercle is the site of attachment of the deep component of the posterior tibiofibular ligament. The anterior tubercle overlaps the fibula. This relationship is the basis for the radiologic interpretation of tibiofibular syndesmosis alignment. [1]

The syndesmosis is primarily responsible in maintaining the integrity of the ankle mortise and failure can cause the ankle mortise to widen resulting in abnormal ankle joint loading.

b) Lateral collateral ligaments:

The lateral ligaments are composed of three components; anterior talofibular ligament (the most commonly injured ligament in the ankle), posterior talofibular ligament and the calcaneofibular ligament (Figure 2). The anterior talofibular ligament resists anterior subluxation of the talus when the ankle is plantar flexed, and it is susceptible to injury in inversion ankle sprains. The calcaneofibular ligament resists inversion with the ankle in dorsiflexion and stabilizes both the ankle and subtalar joint. The posterior talo-fibular ligament is the strongest of the lateral ligaments and prevents posterior and rotatory subluxation of the talus.

c) Medial ligament (Deltoid Ligament)

The deltoid ligament is composed of four bands intermingled with each other and extending from the medial malleolus to the navicular, talus, and calcaneus (Anterior tibiotalar, Posterior tibiotalar, Tibiocalcaneal ligament) forming a superficial and deep layer. The deltoid ligament provides medial ligamentous support of the ankle primarily through the deep layer and the superficial layer of which the tibiocalcaneal ligament being the strongest component is responsible for resisting eversion of the calcaneus. [2]

Ankle injuries

Ankle fractures are divided broadly into those due to rotational forces which result in fractures of the distal fibula and tibia, as well as ligamentous injuries and those secondary to axial loading forces. Axial loading forces result in fractures of the tibial plafond *(Pilon fractures)*. This article will concentrate on the management of ankle fractures due to rotational forces.

Determination of Ankle Stability

Determining stability requires a review of the plain radiographs as well as a thorough physical examination.

Examination

The examination of any limb obviously involves assessment of the neurovascular status, inspection for a communicating open wound and examination of the joint above and below the area of injury. With ankle injuries inspection and documentation of the degree of ankle swelling and presence of fracture blisters are important for timing of surgery.

The ankle is palpated in particular for tenderness specifically over the medial malleolus and the presence of tenderness, swelling, or ecchymosis in this area suggests the possibility medial malleolus fracture or deltoid ligament rupture. The entire length of the fibula is palpated, searching for evidence of a more proximal fibula fracture consistent with a Maisoneuve injury.

The foot and knee are examined for evidence of associated injuries. It is important to recognise and reduce a fracture dislocation. In such cases the foot will be obviously deformed and leaving the talus displaced can compromise vascularity to the foot and reduction prevents further damage to the articular surfaces and soft tissues. [3]

Radiographic evaluation

In the acute trauma setting, the standard x-ray views of the ankle include mortise (obtained with the leg internally rotated 15 to 20 deg so that x-ray beam is nearly perpendicular to the intermalleolar line), anteroposterior (AP), and lateral non weight-bearing views. Additionally if the patient is tender over the proximal fibula additional views to include the full length fibula may be required.

The measurements that typically can be obtained are the medial clear space, the tibiofibular clear space, the tibiofibular overlap, the talar tilt, and the talocrural angle (Figure 3).

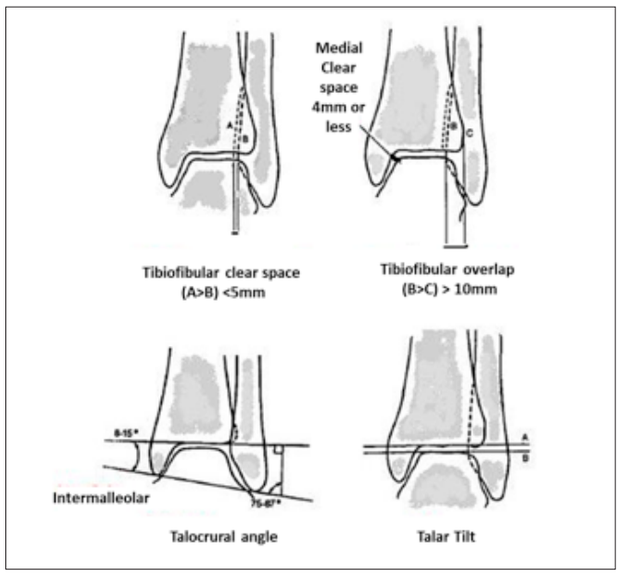
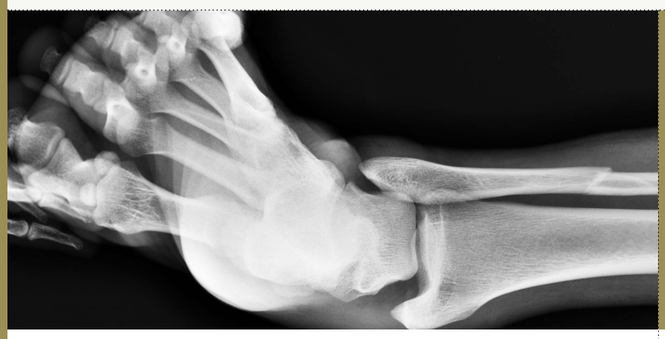


Figure 3: Diagrams demonstrating X-ray measurements

On mortise view a line formed by subchondral bone of distal tibia & medial aspect of the fibula should be continuous. The articular surface of talus should be congruous with that of distal fibula (tibiofibular line) and disruption of this line indicates: Shortening, rotation, lateral displacement fibula; syndesmotic ligaments injury. [4]

ANKLE FRACTURE: ANATOMY, CLASSIFICATION AND INJURY MECHANISM

V P Patel



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Medial clear space

The medial clear space should be equal to the superior clear space between the talus and the distal tibia and less than or equal to 4 mm on standard x-rays.

Talocrural angle

The talocrural anlge is formed by a line drawn parallel to articular surface of distal tibia and a line connecting tips of both malleoli (intermalleollar line). This angle is normally 8 - 15 degrees. Alternatively an angle formed by a perpendicular line to the tibial articular surface and the intermalleollar line can be measured. This angle is normally between 75 and 87 degrees. Using either method this angle should be within 2 - 5 deg of opposite side.

Tibiofibular clear space

The distance between the medial wall of the fibula and the incisural surface of the tibia, should be less than 6 mm.

Talar tilt

A line drawn parallel to articular surface of distal tibia and a second line drawn parallel to the talar surface should be parallel to each other.

Classification

Treatment is based on clinical examination findings, soft tissue integrity and fracture pattern. Fracture pattern is described by two most commonly used classification systems. These are the Lauge-Hansen and Weber classification systems.

Lauge-Hansen

This is a two-part system in which the first word of the classification denotes the position of the foot at the time of the injury — supination or pronation; and the second word indicates the direction of the deforming force — external rotation (eversion), abduction, or adduction.

Four major fracture types were described:, supination-external rotation (SER), supination-adduction, pronation-abduction, and pronation-external rotation (PER) fractures.

Supination-External rotation

The most common injury mechanism (figure 4). The lateral structures of the foot are stressed when the ankle is supinated and if an external rotational force is applied will results in a distal oblique distal fibula fracture. With increasing force, the posterior malleolus avulses. Finally, the medial malleolus fractures, creating a trimalleolar fracture. The structures that are damaged are, in order, the anterior tibiofibular ligament (stage I), the lateral malleolus (stage II), the posterolateral aspect of the capsule or the posterior malleolus (stage III), and the medial malleolus or the deltoid ligament (stage IV).

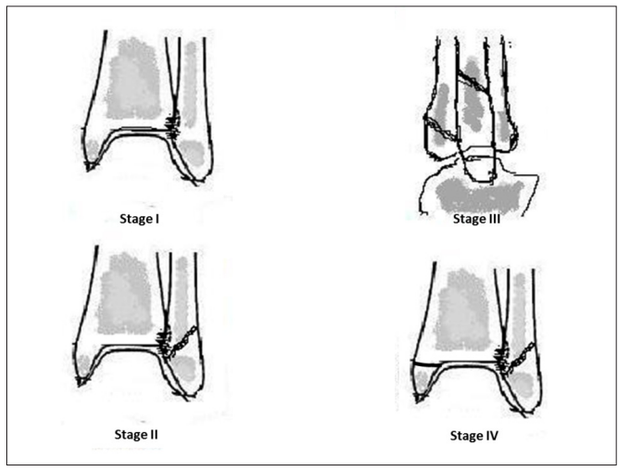

Figure 4: Serial diagrams illustrating sequential injuries sustained with a Supination - External rotation force. Stage I - Anterior inferior tibio-fibular ligament sprain, Stage II - Short oblique fracture of the distal fibula, Stage III - posterior malleolus fracture or posterior tibio-fibular ligament rupture and Stage IV - transverse medial malleolus fracture or deltoid ligament rupture.

Supination Adduction

An adduction force (Figure 5) results in a transverse fibula fracture at or below the level of the anterior inferior tibio-fibular ligament (Stage I). With increasing force the medial malleolus then sustains an oblique fracture (Stage II).

ANKLE FRACTURE: ANATOMY, CLASSIFICATION AND INJURY MECHANISM

V P Patel

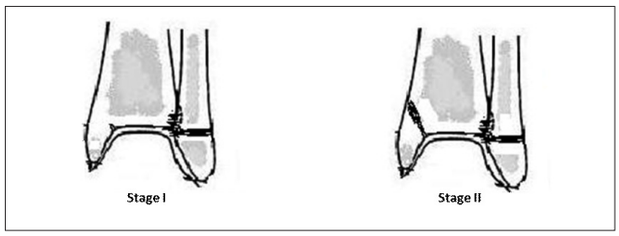

Figure 5: Serial diagrams illustrating sequential injuries sustained with a Supination - Adduction force. Stage I - Transverse fracture of distal fibular at or below the level of the anterior talo-fibular ligament or tear of the lateral ligaments only. Stage II - Vertical or oblique medial malleolus fracture.

Pronation-external rotation

With the ankle in pronation the medial structures are now under stress and when an external rotational force is applied this results in a isolated medial malleolus fracture (or deltoid ligament rupture) - stage I (figure 6). With increasing force, the anterior inferior tibiofibular ligament avulses a portion of the distal tibia and complete or partial rupture of the syndesmotic ligaments (stage II). As the force increases, a more proximal transverse fibula fracture occurs (Stage III) and finally a posterior malleolus fracture (stage IV).

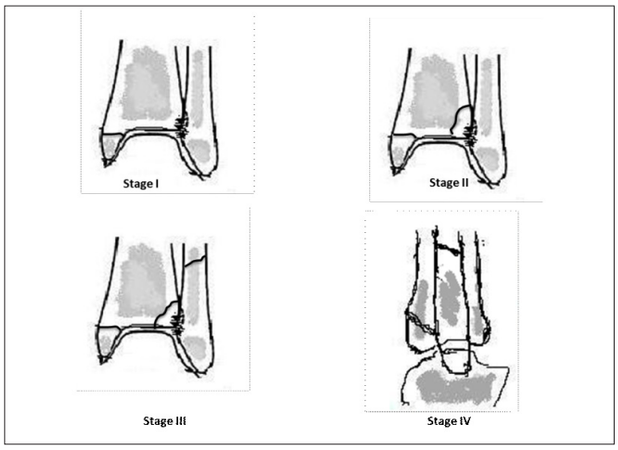

Figure 6: Serial diagrams illustrating sequential injuries sustained with a Pronation External Rotation force. Stage I - deltoid ligament rupture or transverse fracture of the medial malleolus, Stage II - rupture of the anterior inferior tibio-fibular ligament or bony avulsion, Stage III - spiral fibular fracture above the syndesmosis and Stage IV - posterior inferior tibio-fibular ligament rupture or posterior malleolus fracture.

Pronation-abduction

With a forced abduction of the pronated foot (figure 7) the medial structures are stressed resulting in a medial malleolus fracture (Stage I). With increasing force, the anterior tibiofibular ligament avulses a portion of the distal tibia (Stage II). Finally, oblique or comminuted fibula fracture occurs at the level of the syndesmosis (Stage III)[2][5].

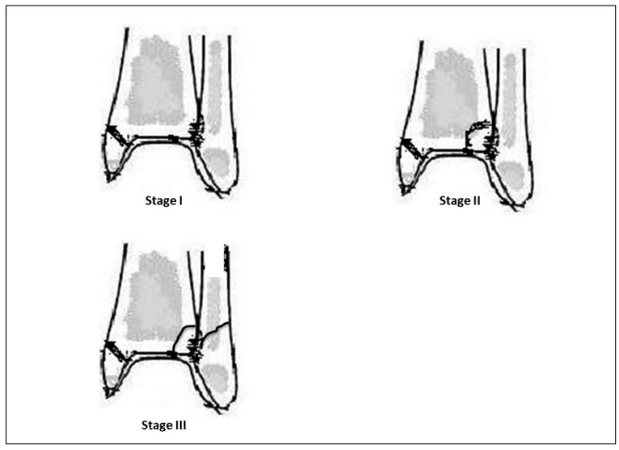

Figure 7: Serial diagrams illustrating sequential injuries sustained with a Pronation-Abduction force. Stage I - Deltoid ligament rupture or transverse fracture of the medial malleolus, Stage II - Rupture of the anterior inferior tibio-fibular ligament or bony avulsion and Stage III - Oblique fracture of the fibula at the level of the syndesmosis.

Weber Classification

Historically, the Weber classification was postulated on the philosophy that the lateral element is the primary stabiliser of the ankle. However, it is now well established the medial structures have a key role in providing ankle stability since it has been recognised that instability of the ankle results from external rotation of the talus and that the medial structures provide the primary restraint to this pattern of instability. [1]

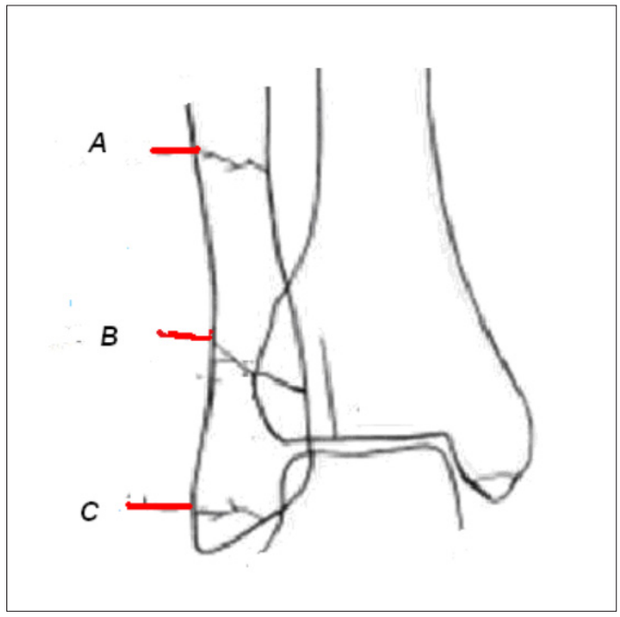

Figure 8: Weber classification.

ANKLE FRACTURE: ANATOMY, CLASSIFICATION AND INJURY MECHANISM

V P Patel

Ankle Fracture:
Anatomy, Classification and injury Mechanism.
Trauma & Orthopaedic Surgery.

The WebThe Weber classification system categorizes ankle fractures by the level of the fibula fracture. Weber A fractures are below the level of the distal tibial fibular syndesmosis. Weber B fractures are at the level of the syndesmosis, and Weber C fractures are above the syndesmosis (figure 8).

Class A fractures are considered stable, not requiring surgical repair, while class B fractures are treated by fibular stabilization, however, many advocate B fractures only require surgical repair if the medial structures are injured. Class C fractures require fibular stabilization and often syndesmotic repair.[3]

Hence, although the Weber classification is simple it ignores medial injury. In addition, the level of the fibula fracture does not always predict the need for syndesmotic repair. Type B fractures may have syndesmosis disruption, and C fractures may be stable after reduction and fixation of the fibula without syndesmosis stabilization, and in some cases may be stable enough to not require any fibular reduction and internal fixation. [2]

Case Discussion

Initial management:
Initial assessment and management of any patient presenting following trauma is according to the ATLS pathway.

This lady has an isolated injury to her ankle. Assessment of any limb injury involves the assessment of the soft tissues, namely the skin and neurovascular status i.e. capillary refill time, warmth, palpable pulses, sensation and movement.

Her skin is intact and her dorsalis pedis and posterior tibialis pulse is present. Sensation to the dorsum and sole of foot as well as movement of her toes are normal.

It is vital that any further damage to soft tissues and cartilage is prevented with immediate reduction and immobilisation of the ankle with a below knee plaster of Paris. This is particularly important in fracture dislocations where this obvious mal-alignment of the foot. After any reduction repeat radiographs and neurovascular examination is paramount.

Radiographs/ Classification:
The AP and lateral radiographs show a tri-malleolar facture (figure 1). In terms of the Lauge-Hansen Classification this fracture is a Stage IV Supination - External rotation injury. Supination and external rotation of the ankle has resulted in rupture of the anterior tibiofibular ligament (stage I), followed by a short distal oblique fracture of the lateral malleolus (stage II). With increasing force, the posterior malleolus avulses (stage III) and finally, the medial malleolus fractures (stage IV), creating a trimalleolar fracture.

By definition this would be a Weber B Ankle fracture.

The medial clear space appears to be widened and the tibio-fibular clear space distorted. There is obvious talar shift.

It is important to understand the mechanism of injury and the forces involved as this would not only give an ideas of the structures damaged but also aid in the reduction of the fracture as the opposite forces would be required to reduce the fracture.

Definitive management:
This fracture is unstable and requires open reduction and internal fixation (Figure 9). Timing of surgery is hugely dependent on the quality of the soft tissues and swelling to allow healing of the incision. Elevation of the limb is important on admission.

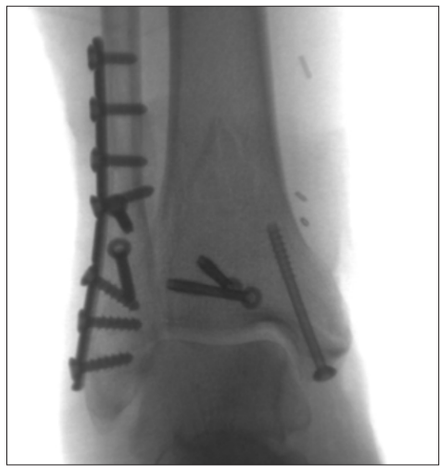

Figure 9: Post-op X-rays following open reduction and internal fixation

ANKLE FRACTURE: ANATOMY, CLASSIFICATION AND INJURY MECHANISM

V P Patel

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MCQs

State which of the following are true or false:

1) With regards to radiographic evaluation of an ankle injury:
a) The talocrual anlge is formed by a line drawn parallel to articular surface of distal tibia and a line connecting tips of both malleoli (intermalleollar line). This angle is normally 8 - 15 degrees
b) A tibiofibular clear space of less than five millimeters and widening of the medial clear space of more than four millimeters are strong indications of a syndesmotic injury.
c) The distance between the medial wall of the fibula and the incisural surface of the tibia, should be less than 6 mm.
d) A mortice ankle view is not helpful in the assessment a potentially ankle injury.
e) A line drawn parallel to articular surface of distal tibia and a second line drawn parallel to the talar surface should not be parallel to each other.

2) With regards to the classification of ankle injuries:
a) The Weber classification takes into account medial injury.
b) Weber C fractures always results in unstable injury of the syndesmosis
c) A stage 4 supination external rotation injury for Lauge-Hansen injury results in a tri-malleolar.
d) In a pronation external rotation ankle injury the anterior tibiofibular ligament avulses first.
e) A distal oblique fracture is an result of a supination external rotation injury .

3) With regards to the clinical assessment of an ankle fractures:
a) In the ottwa ankle rules age is not a factor
b) Inability to weight-bear with tenderness and bruising on the medial malleolus is always as a result from a fracture.
c) Palpation of the proximal end of the fibula is not useful in diagnosing a potential unstable injury following a rotational ankle injury.
d) Gross ankle swelling is a contraindication for surgical fixation if indicated.
e) MRI scans are helpful in assessing for injury to the syndesmotic ligaments.

4) Biomechanical studies have shown that with ankle fractures:
a) The medial structures (i.e. medial malleolus and deltoid ligament) determine ankle stability.
b) Isolated diplaced lateral malleolus fractures cause changes in load charactertics of the talus in the mortice.
c) The deltoid ligament injury does not change the load characteristics of the talus in the mortice.
d) syndesmotic injuries with medial injury (medial malleolus and deltoid ligament) are biomechanically unstable compared with those where there is no medial injury.
e) Biomechanical studies have demonstrated that the posterior malleolus and the posterior tibial fibular ligament have a role in stability of the ankle.

5) With regards to the management management of anke fractures:
a) Posterior malleolus fractures of
greater than 50% only require surgical fixation
b) Deep deltoid ligament injuries require repair
c) With isolated lateral malleolus fractures upto
3mm of displacement is acceptable
d) All Weber B anke fractures require trans-syndesmotic fixation
e) Upto 3mm of talar shift can be accepted in
a isolated lateral malleolus fracture

Answers

1. a) T	b) T	c) T	d) F	e) F
2. a) F	b) F	c) T	d) F	e) T
3. a) F	b) F	c) F	d) T	e) F
4. a) T	b) F	c) F	d) T	e) T
5. a) F	b) F	c) T	d) F	e) F

Authors

Vishal P Patel
ST3 Registrar Trauma and Orthopaedics
Vishalpatel@doctors.org.uk

DUPUYTREN'S CONTRACTURE OF THE HAND – WHAT A CORE TRAINEE SHOULD KNOW

Ching RC, Kotwal A

Dupuytren's Contracture Of The Hand – What A Core Trainee Should Know.
Plastic & Reconstructive Surgery.

Abstract

Dupuytren's disease affects more than 2 million people in UK (male > female) and is due to pathologically thickened longitudinal bands in the palmar or digital fascia developing across a joint, and progressively contracting, preventing it from fully straightening.

The cause is unclear, although a genetic link is considered; associated conditions include diabetes, smoking and alcohol use.

Patients present with a painless lump or contracture over the volar aspect of the hand or finger, commonly the MCPJs and PIPJs of the ring and little fingers, although any joint or digit may be affected; it may or may not impair their hand function.

Treatment is currently predominantly surgical (fasciectomy), with previous medical treatments showing limited success; intralesional collagenase injection has shown positive early results however, and could provide a viable alternative to surgery.

Prognosis is poor as treatment is non-curative; up to 60% of patients experience recurrence of disease. Although those with mild disease may never develop functional impairment or require operative intervention.

Dupuytren's contracture of the hand – what a core trainee should know

Dupuytren's disease or contracture is one of the most common conditions seen in hand surgery, affecting more than 2 million people in the UK.[1] It is seen in both orthopaedic and plastic surgery, and it is therefore likely that, as a core surgical trainee, you will manage it at some point. It is important to understand how the disease arises, what patients present with and what can be done about it.

Definition

Dupuytren's disease is a fibroproliferative disorder whereby pathologically thickened longitudinal bands in the palmar or digital fascia develop across a joint, and contract, preventing it from fully straightening.[2] (Figure 1)

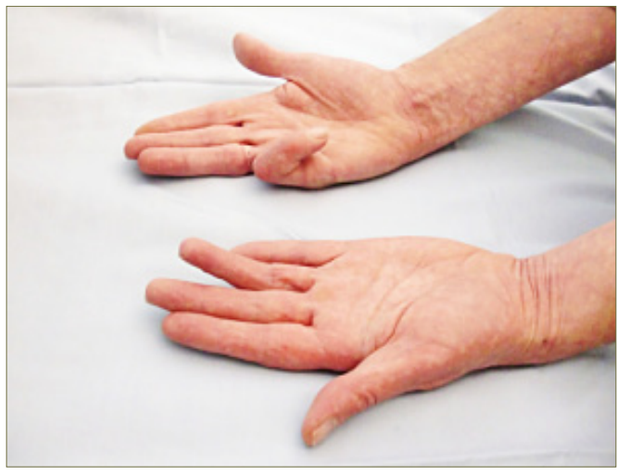

Figure 1: Dupuytren's contracture affecting right little finger PIPJ and left ring finger MCPJ

Epidemiology[1,3]

It is predominantly seen in those of Northern European or Scandinavian descent, males more than females and increases in incidence with age – men typically present at 55 years of age, and women 10 years later.

Aetiology

The exact aetiology of the disease is unclear, but some associations are apparent: diabetes, smoking, hand trauma and alcohol use.[3] There is also thought to be a genetic aspect, and although the precise mechanism is yet to be understood, an autosomal dominant inheritance with variable penetrance has been proposed.[4]

DUPUYTREN'S CONTRACTURE OF THE HAND – WHAT A CORE TRAINEE SHOULD KNOW

Ching RC, Kotwal A

Anatomy[2,5]

In order to understand the pathology behind the disease, it is necessary to know the normal anatomy of the palmar fascia.

The palmar fascia is continuous with the forearm fascia, and the fascia of the dorsum of the hand. It is thin over the thenar and hypothenar eminences; the thick central part, triangular in shape and overlying the long flexor tendons, forms the palmar aponeurosis (PA). The PA consists of longitudinal, transverse and vertical fibres; with the longitudinal ones representing a distal continuation of the palmaris longus tendon (if present), as this merges with the palmar fascia. Distally these longitudinal fibres form four distinct bands running to the bases of the proximal phalanges of each corresponding finger, parallel to the deeper flexor tendons (hence the name pretendinous bands), where they divide into three layers. The most superficial layer inserts into the skin of the distal palm and into the proximal aspect of the flexor sheath. The intermediate layer splits and passes on each side of the flexor sheath, continuing distally as the spiral band, beneath the neurovascular bundle and, inserting into the superficial digital fascia (lateral digital sheet) on each side of the finger (Figure 2). The third layer passes on each side of the flexor sheath to near the metacarpophalyngeal (MCP) joint. In the palm, just deep to the longitudinal fibres, run transverse ones from the ulnar aspect of the little finger to the radial aspect of the index finger, over the MCP joints and the junction of the middle and distal thirds of the palm. The vertical fibres are located deep to the transverse ones, forming the sides of eight canals (four contain the underlying finger flexor tendons, and four containing the lumbrical muscles and the neurovascular bundles).

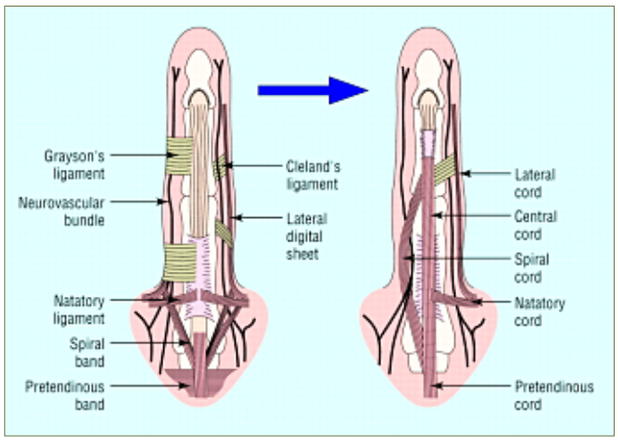

Figure 2: Anatomy of the digital ligaments and bands (left) and the pathological cords of Dupuytren's contracture (right).
Reproduced from BMJ, Townley WA, Baker R, Sheppard N, Grobbelaar AO, 332(7538):397-400, 2006 with permission from BMJ Publishing Group Ltd.

Pathophysiology[1-2,4]

In early Dupuytren's disease, fibroblasts within the fibrous bundles of the fascia proliferate and form what is clinically seen as a nodule. As the disease progresses, the fibroblasts are thought to differentiate and myofibroblasts, with their contractile elements, become the predominant cell. These cause an increase in type III collagen production (the same as seen in scar tissue; normal palmar fascia collagen is predominantly type I), which, orientated longitudinally, form a cord that results in the contracture.

Clinically, the first manifestation of the disease is the appearance of a nodule. They often appear in the pretendinous bands and, with progression, form a cord, which results in a flexion contracture of the MCP joints (when diseased, the term 'band' is replaced with the term 'cord', e.g.: pretendinous cord). Of all the fibres forming the palmar aponeurosis, it is only the longitudinal ones which are involved. The continuation of these fibres into the finger means that the disease also develops here – primarily involving the spiral band, superficial fibrofatty fascia and the lateral digital sheet – forming the spiral cord, the central cord and the lateral cord, respectively (Figure 2). As the fibres of the spiral band pass deep to the neurovascular bundle when initially travelling to the lateral aspect of the finger, and then pass superficial to it when attaching to the middle phalanx, the neurovascular bundle is often displaced medially, proximally and superficially, by the spiral cord as disease progresses and proximal interphalangeal (PIP) joint contracture develops. This displacement puts the neurovascular bundle at great risk during surgery.

Clinical assessment

Patients will initially present complaining of either a painless lump in their palm or finger, or more commonly later, once a contracture has developed. It is most often seen affecting the MCP joints or PIP joints of the ring and little fingers, 45% bilaterally,[3] with the distal interphalangeal (DIP) joint being spared.[6] It can affect any of the fingers or thumb, however. They will have noted pitting or thickening of the palmar skin for some time prior to the development of a nodule. After months or years the nodule or cord will start to contract and the stage at which patients present will often depend on the degree of functional impairment. When taking a history it is important to identify if the contracture interferes with occupation, hobbies or activities of daily living (e.g.: washing or dressing).[1,3] The patient may also be prompted to seek medical input due to a fear of the lump being cancerous.

The same process that causes Dupuytren's to develop in the palms, can cause ectopic fibrosis to form in the soles of the feet (Ledderhose disease), the penis (Peyronie's disease), or over the dorsum of the PIP joints (Garrod's knuckle pads). [1,3] It is imperative that these manifestations are identified, as patients with severe bilateral disease, a strong family history and such ectopic disease are said to have Dupuytren's diathesis, and subsequently a poorer prognosis and require more aggressive treatment and follow-up.

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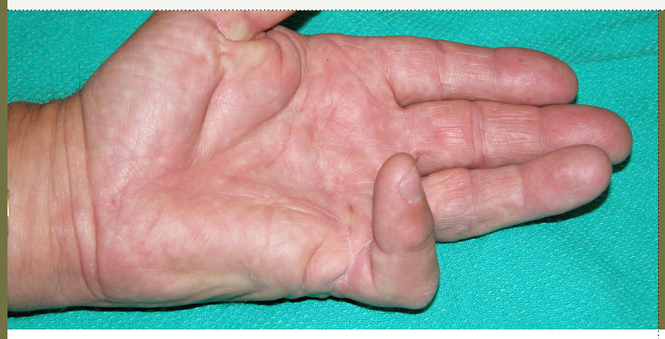
Ching RC, Kotwal A



Dupuytren's Contracture Of The Hand – What A Core Trainee Should Know. Plastic & Reconstructive Surgery.

When examining the hands, other differential diagnoses of a palmar lump or being unable to fully extend a finger must be ruled out; these include rheumatoid changes, a ganglion, post-traumatic joint contraction or a locked trigger finger. All fingers are inspected, with all joints and tendons assessed (Table 1). In addition to identifying the affected digits, the sensation of each is checked, as nerve damage is a possible complication of operative management and the preoperative status should be documented. In order to assess patients with Dupuytren's disease adequately, examination is not limited to the hands; patients also have their general health, and subsequently their surgical risk, investigated.

Inspect	Palpate	Test
Scars	Tenderness	Flexion (FDS/ FDP/ FPL)
Deformity	Nodules	Extension (EDC/ EI/ EDM)
Contracture	Cords	Abduction (APL/ dorsal interossei)
Skin changes	Sensation (RDN/ UDN/ median/radial/ulnar nerves)	Adduction (palmar interossei)
		Active + passive range of movement (wrist/ MCPJ/ PIPJ/DIPJ)

Table 1: Hand examination summary

(RDN: radial digital nerve; UDN: ulnar digital nerve; FDS: flexor digitorum superficialis; FDP: flexor digitorum profundis; FPL: flexor pollicis longus; EDC: extensor digitorum communis; EI: extensor indicis; EDM: extensor digitorum minimi; APL: abductor pollicis longus).

As core surgical trainees, we are involved in seeing these patients in outpatient clinics, and as the diagnosis is solely clinical, it is imperative to develop these assessment skills. Only after taking a full patient history, including hand dominance and occupation; identifying any associations, ectopic disease or family history the patient has; and completing a thorough examination, are you in a position to consider which treatment options would be most appropriate for the patient.

Management

As with most surgical conditions, the treatment options can be divided into non-operative and operative (table 2).

Non-operative		Operative
Monitoring of disease as outpatient	Radiotherapy	Fasciotomy
Serial splinting	Collagenase injections	Fasciectomy

Table 2: Summary of management options

Patients with minor disease (no contractures or those not interfering with function) can be observed initially, as they may never develop disease severe enough to warrant intervention.[1] Patients who do have disease that affects their daily activities, but who are deemed a high surgical risk due to medical comorbidities may also be managed conservatively in the first instance – with the aim to either avoid surgery altogether or to optimise the patient medically before an operation is planned. Serial splinting has previously been considered as an option for these patients, but is not practical as in itself limits hand function, and lacks any valid evidence.[7]

Medical treatments have so far not been widely recognised. Future options include radiotherapy and intralesional injections of substances to disrupt the cord, such as collagenase. Radiotherapy is thought to improve symptoms by affecting the development and growth rate of fibroblasts in the palmar fascia. NICE guidance reports studies describing regression of disease post-radiotherapy in up to 56%[8] but the potential side effects of such a harmful modality may rule it out as a truly viable option.[7] Collagenase (derived from Clostridium histolyticum) injections are thought to lyse the collagen and subsequently allow rupture of the cords through manipulation, resulting in the release of contractures for those with advanced disease.

A recent large prospective multi-centre clinical trial using intralesional collagenase on both MCP and PIP joints showed 64% of joints achieved within 5° of full extension 30 days following injection up to a maximum 3 times, and was well-tolerated with few adverse events reported.[9] These findings were corroborated by a second smaller trial,[10] and an 8 year review of patients who received collagenase reported a recurrence in 6 out of the 8 patients enrolled, but the recurrence was felt to be less severe than the primary disease, and patient satisfaction with the initial procedure was high.[11] These findings support the hope for collagenase to become a primary treatment option and for some patients to possibly avoid surgery with all its inherent risks.

DUPUYTREN'S CONTRACTURE OF THE HAND – WHAT A CORE TRAINEE SHOULD KNOW

Ching RC, Kotwal A

Surgery currently, however, does remain the treatment option of choice in those fit enough, with fasciectomy being the most popular. Surgery is required in those with contractures of the MCP joint >30-40° or >0-20° for a PIP joint.[4,12] Intervention is undergone for PIP joint contracture at a lower threshold due to the laxity of its collateral ligaments when in the flexed position; these subsequently shorten once even a mild contracture develops resulting in rapid deformity. Individual consideration needs to be applied to each patient regarding the impact of the disease on their hand function however. Fasciotomy, an outpatient procedure, involves incising the diseased fascia with a blade or bevel of a needle rather than excising it, and, although providing decent short-term results, it is not definitive on its own and is linked with high recurrence rates. It is therefore deemed more appropriate in those unsuitable for major surgery.[13]

Fasciectomy (Figure 3) is the removal of disease through an open incision under regional or general anaesthesia, and is the most common procedure performed for Dupuytren's disease. It may simply remove all macroscopically visible disease (limited fasciectomy) or involve complete removal of the palmar fascia (radical fasciectomy).[1] The procedure can be performed through several different incisions used by hand surgeons, including a zigzag incision or a longitudinal incision closed with a Z-plasty. These methods are used to avoid any straight longitudinal scars on the volar surface of the palm or fingers, as these may subsequently shorten and form contractures of their own.

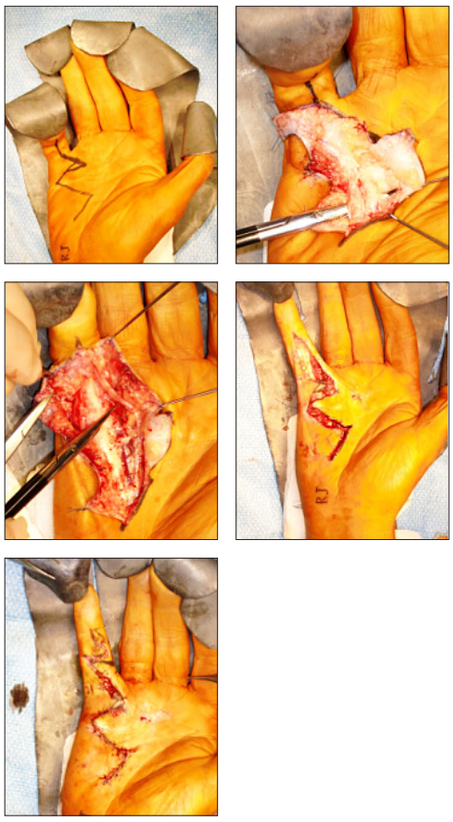


Figure 3: Fasciectomy with Z-plasty over little finger PIPJ

When performing the operation it is imperative to identify and protect each digital artery and nerve prior to any excision, as the nature of the disease causes displacement of the neurovascular bundles into abnormal positions, as previously described. This is often done by starting the procedure in relatively normal tissue (often the proximal palm) where vital structures are more easily identifiable; these are then closely followed and protected as the diseased tissue is excised. Closure of the wound may also be done in a variety of ways. Direct primary closure of zig zag incisions may be possible, but, after excision and fully extending affected joints, there may be areas of apparent skin loss that require attention.

The Z-plasty as previously mentioned, is a way of closing a longitudinal cut to avoid a longitudinal scar (Figure 4). Other options include applying a full-thickness skin graft (a split-skin graft is more likely to contract) or leaving the wound open to heal by secondary intention – the McCash technique.[2,6] Postoperatively, a splint may be applied with flexion of the MCP joints 45-70° and extension of the IP joints. Complications of surgery include digital nerve or vessel damage, infection, haematoma, skin flap necrosis, skin graft failure or chronic regional pain syndrome.

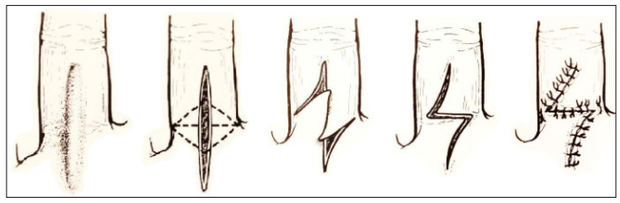


Figure 4: Z-plasty

This figure was published in Fundamental Techniques of Plastic Surgery, 10th edition, AD McGregor, IA McGregor, Hand surgery, Page No 194. Copyright Elsevier (2000).

Prognosis

Many patients with an indolent disease course will not develop contractures and therefore not require any surgical management. For those patients who do require intervention, an improvement in hand function following fasciectomy is noted,[14] with them often being able to return to work or other pre-morbid activities, although regaining their full pre-morbid state is unlikely. However, as treatment is not curative and only palliates the effect Dupuytren's disease has on hand function, recurrence is high (up to 60%[1]), with the time frame dependent on the aggressiveness of the disease. Those with a strong diathesis (family history and ectopic disease possibly present) may have to undergo several procedures.

Acknowledgement

Thanks to Mr Russ Jeffers (Consultant Hand Surgeon, Bradford Royal Infirmary) for assistance in obtaining fasciectomy photographs.

DUPUYTREN'S CONTRACTURE OF THE HAND – WHAT A CORE TRAINEE SHOULD KNOW

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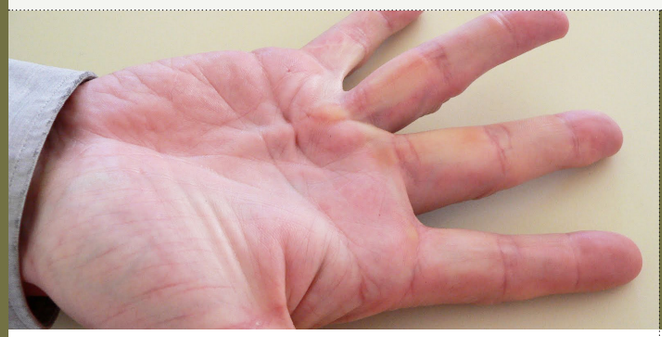

Dupuytren's Contracture Of The Hand – What A Core Trainee Should Know. Plastic & Reconstructive Surgery.

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Clinical vignette

Mr F is a 68 year-old right-hand dominant retired male shop-keeper who presented to hand clinic complaining of difficulty using his right hand, specifically when doing woodwork which is one of his hobbies. He reports his ring and little fingers on the right "get in the way". Mr F denies any pain in his hand, or problems elsewhere on the body. Notable past medical history includes hypertension and well-controlled non-insulin-dependent diabetes. On examination, Mr F was found to have a palpable nodular cord over the volar aspect of the right ring finger with a flexion contracture of the MCPJ (40°) and PIPJ (25°). He was also noted to have a small nodule with no contracture over the left ring finger MCPJ. Mr F subsequently underwent preassessment and was listed for fasciectomy to right ring finger as a day-case procedure under general anaesthesia. Intra-operatively, the affected tissue was excised and full extension of all affected joints was achieved; closure was accomplished with a Z-plasty. He was subsequently placed in a splint to await outpatient hand physiotherapy. Three months postoperatively, his range of movement was well-maintained and Mr F was happy he could continue with his woodwork.

Questions

1. Which of these is not an associated condition of Dupuytren's disease?

a. Lederhose disease
b. Peyronie's disease
c. Keinbock's disease
d. Garrod's disease

2. If present, where does the Palmaris longus tendon lie in relation to the palmar aponeurosis?

a. Inserts superficial to it
b. Inserts deep to it
c. Continues lateral to it
d. It merges with it

DUPUYTREN'S CONTRACTURE OF THE HAND – WHAT A CORE TRAINEE SHOULD KNOW

Ching RC, Kotwal A

3. Why does contracture occur?

a. Due to an increased presence of type I collagen
b. Due to flexor tendon involvement
c. Infection
d. Myofibroblast proliferation

4. Why is the digital neurovascular bundle at high risk during Dupuytren's fasciectomy?

a. During the proliferative stage, it enlarges
b. It is displaced into an abnormal position by the cord
c. It becomes adherent to the skin
d. The common incisions used lie directly over the path of the bundle

5. Which is the least likely way to close a fasciectomy wound?

a. Primary closure
b. Split-thickness skin graft
c. Full-thickness skin graft
d. Z-plasty

Answers

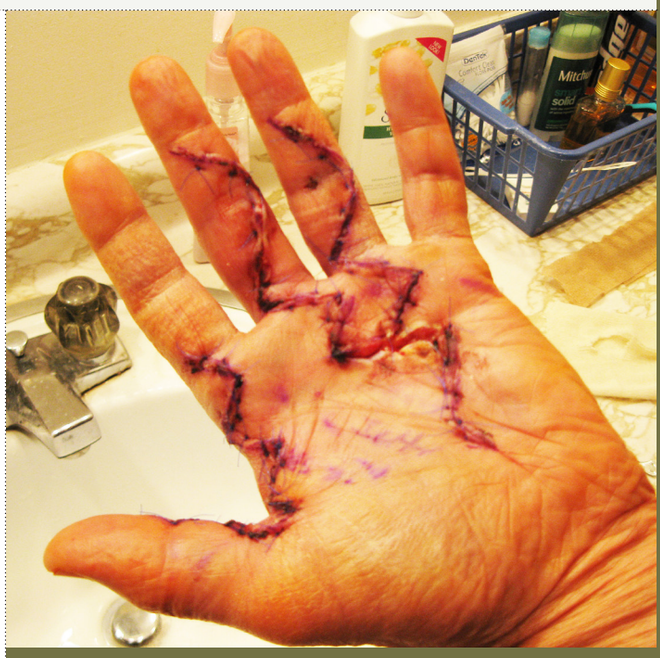
1. c. Keinbock's disease. This condition involves idiopathic avascular necrosis of the lunate bone.

2. d

3. d. There is then deposition of type III collagen, rather than the more common type I.

4. b

5. b. This is likely to contract.



Dupuytren's Contracture Of The Hand – What A Core Trainee Should Know.
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Corresponding author

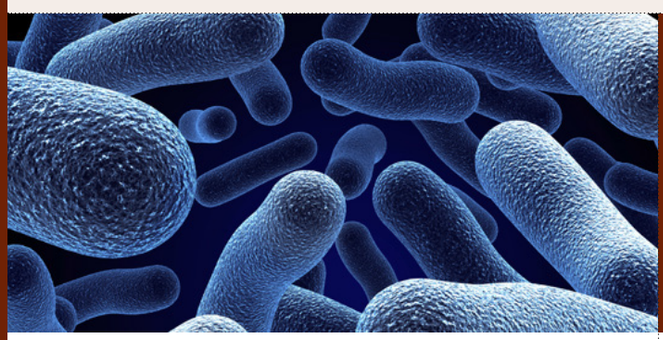
Rosanna Ching
Core Trainee Year 2 in Plastic Surgery
18 Hesketh Road
Leeds LS5 3ET
rcching@doctors.org.uk

2nd author

Ashutosh Kotwal
Specialist Registrar in Plastic Surgery

COMMENSALS: WHEN IS NORMAL ABNORMAL?

E Yiannakis



Commensals: when is normal abnormal?
Surgical Microbiology.

Abstract

Intra-abdominal infection is a cause of significant morbidity and mortality in the surgical patient. The microbiological organisms responsible for these infections are usually derived from the patient's own gastrointestinal (GI) flora. Knowledge of this flora is thus essential in allowing the clinician to effectively manage cases of intra-abdominal infection. An understanding of the normal microbiota of the GI tract also facilitates the identification of infections that may have originated from the gut and guides for the selection of appropriate empirical and targeted antibiotic therapy. This article aims to outline the commensal organisms of the GI tract, explain their importance in cases of secondary peritonitis and identify fundamental principles in the selection of empirical antimicrobial regimens for these infections.

Introduction

Intra-abdominal infection is an important and common surgical problem. Often the organism isolated from microbiological culture of intra-abdominal specimens gives an indication as to the source of infection and may also be the first warning of post-operative complications. The ability to interpret these results relies on an understanding of the microbes that represent the 'normal flora' throughout the gastrointestinal (GI) tract. In addition, an understanding of this flora allows the clinician to predict the possible pathogens responsible for infection in cases of secondary peritonitis and is hence crucial in directing empirical antibiotic therapy prior to obtaining culture results. The following two case studies aim to provide a focus from which a discussion on the normal GI flora and its impact on intra-abdominal infection can be initiated.

Clinical Vignettes

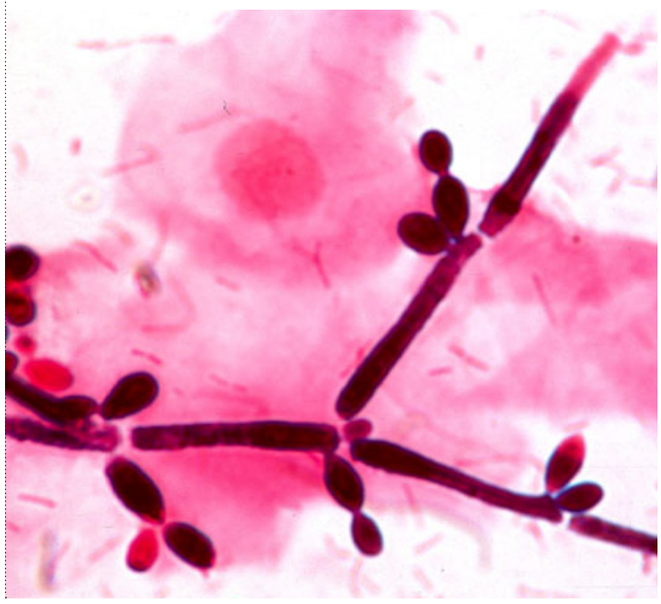
Case history 1:

Mr RL, an 81 year old gentleman, presented to the General Surgical Take with a two week history of worsening right flank pain, accompanied by swinging fevers and new onset confusion. On assessment, Mr RL was pale, tachycardic and febrile with a temperature of 38.5°C. Examination of his abdomen revealed him to have marked right loin tenderness, although there was no evidence of guarding or peritonism. Six months prior to this admission, he had undergone the formation of a transverse loop colostomy following an episode of large bowel obstruction attributed to diverticular disease. He had no other past medical history of note and was not taking any regular medications.

An initial diagnosis of pyelonephritis was made and Mr RL was commenced on intravenous co-amoxiclav. Despite this treatment, over the next 48 hours his overall condition deteriorated. He complained of worsening pain and continued to spike temperatures above 39°C. A CT scan of the abdomen was arranged and, after discussion with microbiology, the co-amoxiclav was replaced with meropenem.

Unexpectedly, the CT scan exposed a number of abnormalities, the most striking of which was a large right psoas abscess measuring about 6 x 3.5 x 8.6 cm. Note was also made of right renal hydronephrosis, with a dilated ureter extending down to the vesicoureteric junction. The dilated renal pelvis and ureter were in very close proximity to the psoas abscess and there were small perinephric collections that appeared to be in continuity with the abscess.

In light of these findings, an urgent radiologically-guided drain was sited with immediate drainage of a large volume of offensive pus. On culture, this pus grew a heavy, pure growth of *Candida albicans,* an organism that was also isolated from the admission blood cultures. The patient was subsequently started on treatment with the echinocandin antifungal agent Caspofungin. At this point, the question was posed as to why Mr RL had developed a deep-seated candidal infection in a sterile site.



COMMENSALS: WHEN IS NORMAL ABNORMAL?

E Yiannakis

Further imaging and ensuing investigation of the gastrointestinal tract answered this question. On further analysis of the CT scan it was noted that the right ureter was in close proximity to the rectal staple line. This area of bowel had significant soft tissue thickening, suspicious of tumour. A hypodense area in the right lobe of liver, suggestive of a metastasis, was also observed. Histology of a tissue biopsy of this liver lesion confirmed adenocarcinoma. In the context of the patient's history, this was felt to be secondary to a primary colonic malignancy, the likely cause of his previous admission with bowel obstruction. Erosion of the tumor into the right ureter provided the portal via which the *Candida albicans* gained entry to the renal tract and subsequently into the psoas muscle.

Case history 2:

Mr IL, a 68 year-old-gentleman with a recent diagnosis of adenocarcinoma of the oesophagus, was electively admitted for an Ivor-Lewis oesophagectomy. Apart from his newly diagnosed malignancy, Mr IL was exceptionally fit and had no background of respiratory or cardiovascular disease. His operation was uneventful and he was transferred to the Surgical High Dependency Unit for routine post-operative care. He received intra-operative prophylaxis with cefuroxime according to local antimicrobial guidelines.

Over the next forty-eight hours, he started spiking high temperatures and became progressively more breathless. A chest Xray showed new patchy consolidation in the right lower lobe as well as small bilateral pleural effusions. A diagnosis of hospital acquired pneumonia (HAP) was made and he was commenced on Piperacillin/Tazobactam (Tazocin®). His condition continued to deteriorate and he required intubation, ventilation and admission to the Intensive Care Unit. Despite antibiotics, he remained difficult to ventilate and his temperature did not normalise. Sputum samples sent to microbiology grew a mixture of respiratory tract commensals only and were unhelpful in confirming a diagnosis. In view of his worsening condition, he had a repeat chest Xray which showed an increase in the size of the right pleural effusion necessitating the insertion of a chest drain. Surprisingly, culture of a sample of pleural fluid revealed growth of the gram-positive bacillus *lactobacillus,* an organism that is a member of the normal flora of the oropharynx and gastrointestinal tract. This finding went against the provisional diagnosis of HAP and raised the concern of a connection between the pleural space and the oesophagus. Subsequent imaging with a CT scan confirmed an anastomtic leak.

Discussion

These two cases illustrate examples of circumstances where the discovery of GI tract commensals in sterile sites indicated a significant underlying surgical problem. In both cases, knowledge of the likely origin of each of the microorganisms cultured guided further investigation and contributed to the establishment of a diagnosis. In short, the organism pointed to the site of pathology. Conversely, an appreciation of the indigenous inhabitants of the gut also allows the clinician to predict the agents likely to cause infection in cases of secondary peritonitis.

Secondary intra-abdominal infections usually occur as a result of contamination of the peritoneal cavity by bowel contents following loss of integrity of the normal mucosal barrier[1]. This breach of the GI tract can occur as a result of a multitude of pathologies, for example intestinal infarction, obstruction or trauma[2] and can involve any of the intra-abdominal viscera. The spectrum of causes of secondary peritonitis includes both community-acquired infections, such as appendicitis, as well as more complex nosocomial causes such as anastamotic leaks[3]. In all cases, the type, quantity and combination of organisms that can spill into the peritoneum depends on the normal microflora at the anatomical site of the lesion.

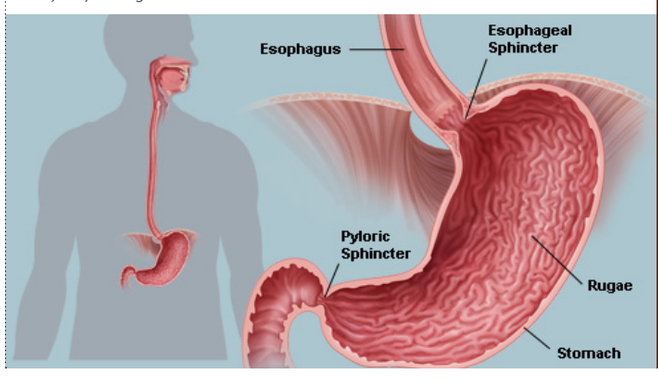
Normal flora of the gastrointestinal tract

The microorganisms that compose the indigenous flora of the GI tract are similar for all humans and remain relatively stable over the lifetime of the individual[4]. For the purposes of outlining the characteristic commensal organisms, the GI tract can be divided into three components: the oesophagus and stomach, the small intestine and the large intestine. Each of these areas has a typical ecology of micoflora that varies in density, type and variety of microbial species[1]. Factors that can influence the members of the GI normal flora include specific host features, such as immunocompromise, and drug exposure, particularly previous antibiotic treatment. Although antibiotics are administered with the aim of targeting a specific pathogen, many are broad-spectrum with the destruction of members of the normal commensal bacteria as a consequence of their use[5]. This also results in reduction in the diversity of the microbial flora[5]. Another undesirable outcome of antibiotic use is the selection of antibiotic-resistant organisms in the intestine, which can then act as a reservoir for resistance genes in the gut microenvironment[6]. The clinical importance of this alteration in the normal flora is that it places the patient at risk of secondary peritonitis with resistant organisms. Other drugs, such as proton pump inhibitors, can also impact on the commensal organisms by altering the microenvironment of the flora via changes in pH[7]. It is important to consider all these factors when selecting empirical antibiotics.

The following summary is representative of members of the normal GI flora in the healthy individual.

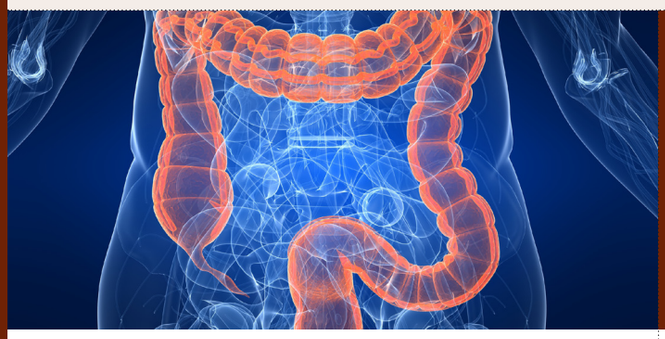
Oesophagus and Stomach

Factors that influence the exact composition and number of organisms include diet and gastric pH. Generally however gram-positive bacteria including *lactobacilli* and *streptococci* and the yeast *Candida* account for the majority of organisms[1,4].



COMMENSALS: WHEN IS NORMAL ABNORMAL?

E Yiannakis



Commensals: when is normal abnormal?
Surgical Microbiology.

Small Intestine

The members of the microbiota of the small intestine are very similar to those of the oesophagus and stomach[1,4]. The density and diversity of flora increases as the large bowel is approached.

Large Intestine

The microbiological community in the large intestine has the highest density of organisms, estimated at 10^2 bacteria/gram, of any part of the human body[8,9]. In the large bowel, the normal flora is composed primarily of anaerobes, which outnumber facultative organisms by 10^3:1 to 10^4:1, 1,[10]. The majority of these anaerobes belong to the divisions of *Bacteroides* and *Firmicutes*[10]. Aerobic members of the normal colonic flora include gram-negative organisms such as *E.coli, Enterobacter, Klebsiella, Proteus and Pseudomonas;* gram-positive organisms such as *Enterococci* and *Streptococcus viridans* and fungi, mainly *Candida spp*[3].

It is worth noting that although any number of this diverse group of organisms can be grown from stool culture, the organisms that are isolated from culture of peritoneal fluid in cases of secondary peritonitis tend to be more select[11,12]. This relates to virulence factors distinct to the specific microbe, for example gram-negative organisms such as *E.coli* may express a number of virulence factors and consequently may be the bacterium within the mix of flora that is responsible for sepsis. Alternatively, anaerobes play an important role in abscess formation[13] and in studies using techniques that allowed the isolation of anaerobes from samples of pus from intra-abdominal abscesses, anaerobes were isolated in 60% to 70% of cases[14,15].

Empirical Antibiotic Choice

The choice of antibiotics to treat secondary intra-abdominal infections prior to culture results is influenced by the predicted likely pathogens, guided by knowledge of the gut microbiota. Other important factors include previous antibiotic use, hospitalisation and local resistance patterns[16]. In general however, because of the polymicrobial nature of these infections, broad-spectrum cover is required and any antibiotic regimen needs to include agents that have activity against gram-negative organisms, gram-positive organisms and anaerobes. Examples of single agents that would cover this spectrum of bacteria include co-amoxiclav, piperacillin/tazobactam and meropenem.

Combination regimens include the conventional combination of a cephalosporin such as cefuroxime (which has activity against some gram-negative and some gram-positive organisms) with metronidazole, an antibiotic active against anaerobes. In cases of upper-GI pathology, such as the instance of oesophageal anastamotic leak described in case 2, it is prudent to include an antifungal agent as part of empirical therapy due to the presence of candida spp in the commensal flora[1,4].

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COMMENSALS: WHEN IS NORMAL ABNORMAL?

E Yiannakis

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Multiple Choice Questions

Single Best Answer (SBA)

1. When treating Mr AE, an 81-year-old gentleman with a history of faecal peritonitis secondary to a perforated diverticulum, which of the following antibiotics would not provide adequate cover against anaerobic organisms if used as monotherapy?

A. Co-amoxiclav (Augmentin®)
B. Meropenem
C. Piperacillin/Tazobactam (Tazocin®)
D. Cefuroxime
E. Clindamycin

2. When treating Mr AE, an 81-year-old gentleman with a history of faecal peritonitis secondary to a perforated diverticulum, which of the following antibiotics would not provide adequate cover against gram-negative organisms if used as monotherapy?

A. Co-amoxiclav (Augmentin®)
B. Meropenem
C. Piperacillin/Tazobactam (Tazocin®)
D. Cefuroxime
E. Clindamycin

3. A sample of peritoneal fluid from Mr AE is sent to microbiology. Which of the following organisms is unlikely to be cultured from this sample as it is not usually a commensal of the gastrointestinal tract?

A. Candida albicans
B. Haemophilus influenzae
C. Enterococcus faecalis
D. Escherichia coli
E. Bacteroides fragilis

4. The microbiologist contacts the surgical team as blood cultures sent from Mr AE on admission are positive with growth of a gram-negative bacillus, identified as an Escherichia coli. This isolate has tested positive for the production of Extended-spectrum ß-lactamases (ESBLs). In view of this result, which of the following antibiotics should Mr AE be treated with?

A. Cefuroxime
B. Gentamicin
C. Meropenem
D. Piperacillin/Tazobactam (Tazocin®)
E. Co-amoxiclav (Augmentin®)

Extended Matching Questions (EMQs)

Four cases of musculoskeletal / skin and soft tissue infection are outlined below. For each case, please choose from the following list of bacteria the organism that is most likely to be responsible for the infection. Each option may be used once, more than once or not at all.

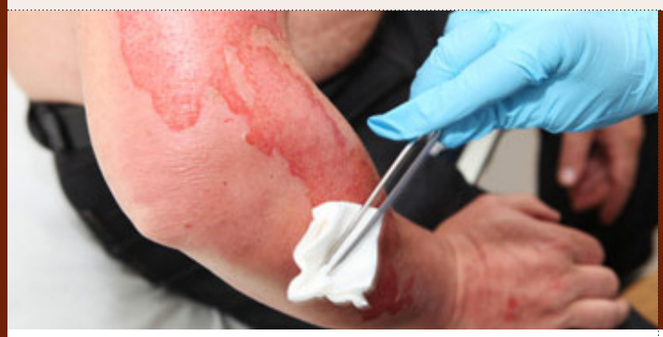
a. Salmonella enteritidis
b. Staphylococcus epidermidis
c. Staphylococcus aureus
d. Neisseria gonorrhoeae
e. Streptococcus pyogenes
f. Enterococcus faecalis
g. Pseudomonas aeruginosa
h. Clostridium perfringens

5. A 64-year-old patient with a 20 year history of rheumatoid arthritis presents with a tender, hot, swollen right wrist. She is pyrexial and looks unwell. A joint aspirate shows gram-positive cocci on microscopy.

6. A 74-year-old gentleman presents with gradually worsening pain in his leg 6 months following a right Total Hip Replacement. He is otherwise well. He is taken to theatre for exploration and samples of tissue from the hip show gram-positive cocci on microscopy.

COMMENSALS: WHEN IS NORMAL ABNORMAL?

E Yiannakis



Commensals: when is normal abnormal?
Surgical Microbiology.

7. An 18 year-old patient is admitted with 17% burns following a BBQ-related accident. The patient has been treated with co-amoxiclav, but is becoming progressively more unwell with high temperatures and worsening inflammatory markers. On examination, the burns are covered with a sloughy, greenish discharge. Swabs from the burns grow a gram-negative organism.

8. A 24 year-old female is admitted with a compound fracture of her left tibia and fibula following a fall from a horse. The wound was heavily contaminated with soil and debris. Two days following surgery, she complains of excruciating pain in her leg, which appears discoloured on examination. She is taken back to theatre urgently and samples of tissue show gram-positive bacilli on microscopy.

Answers

1. D. Cefuroxime
Co-amoxiclav, piperacillin/tazobactam and meropenem are all broad spectrum antibiotics with activity against gram-positive, gram-negative and anaerobic organisms. Cefuroxime however does not have effective action against anaerobic bacteria and as such needs to be administered with an antibiotic active against anaerobes, usually metronidazole.

2. E. Clindamycin
Clindamycin has no activity against gram-negative organisms and as such is not commonly used to treat intra-abdominal sepsis.

3. B. *Haemophilus influenzae*
Most cases of secondary peritonitis occur as a result of contamination of the peritoneum with commensals of the gastrointestinal tract. These commensals include gram-negative organisms (for example *E.Coli, Enterobacter spp, Proteus spp),* gram-positive organisms (for example *enterococci*), anaerobes (for example *B. fragilis and C.Perfringens*) and fungi, mainly *Candida albicans.* If a mixture of any of these organisms is grown from a blood culture, this strongly suggests an intra-abdominal focus of infection. *Haemophilus influenzae* however, is a commensal of the upper respiratory tract and is a common cause of community-acquired pneumonia.

4. C. Meropenem
ESBLs are ß-lactamases that are capable of conferring bacterial resistance to the penicillins, cephalosporins and aztreonam. Bacteria that are ESBL positive may also be resistant to other classes of antibiotics, such as the aminoglycosides (for example gentamicin) and the quinolones (for example ciprofloxacin). The carbapenems, such as meropenem, are currently the first-line choice of antibiotic for sepsis secondary to these organisms.

5. c. *Staphylococcus aureus*
S. aureus is the most common cause of septic arthritis, particularly in patients with intercurrent chronic joint disease. S. aureus appears as gram-positive cocci in clusters on microscopy.

6. b. *Staphylococcus epidermidis*
Coagulase-negative staphylococci (including *Staphylococcus epidermidis*) are a common cause of prosthetic joint infections. These organisms are less virulent than *S. aureus* and the indolent nature of this patient's presentation accompanied by the fact that he is clinically stable suggests a less pathogenic causative organism. Coagulase-negative staphylococci appear as gram-positive cocci in clusters on microscopy.

7. g. *Pseudomonas aeruginosa*
The moist, warm environment provided by burns supports the growth of a number of organisms; including gram negative bacteria such as *P. aeruginosa.* This patient has been on co-amoxiclav which, although broad-spectrum, does not have activity against *Pseudomonas* spp. and hence is likely to have allowed for selection of this organism. Colonies of *P. aeruginosa* typically have a greenish colour and appear as slender gram negative bacilli on microscopy.

8. h. *Clostridium perfringens*
This patient's history is suggestive of gas gangrene secondary to contamination of her wound by soil containing clostridial spores. Although antibiotics are essential, the main component of management is urgent surgical debridement of the wound. *C. perfringens* appear as gram-positive bacilli on microscopy.

Author

Dr. Eftihia Yiannakis BMedSci, BMBS, MRCP
Specialist Registrar in Medical Microbiology
Email: tiayiannakis@googlemail.com

THE NEUROPATHIC BLADDER

J Moore

The Neuropathic Bladder.
Urology.

Introduction

The lower urinary tract functions to store and void urine in a controlled manner which is coordinated by the peripheral and central nervous systems. The neuropathic bladder, more accurately known as neurogenic lower urinary tract dysfunction (NLUTD) describes a broad spectrum of conditions which cause the bladder, the sphincter or a combination of both to function abnormally due to lesions affecting their innervation. Anatomically, these lesions can be categorised as arising in four distinct areas:

1. Cerebral
2. Pontine
3. Suprasacral spinal cord
4. Sacral spinal cord and subsacral/peripheral

In order to have an understanding of these abnormalities, it is necessary to first understand normal bladder and sphincter innervation and physiology.

Anatomy and Physiology

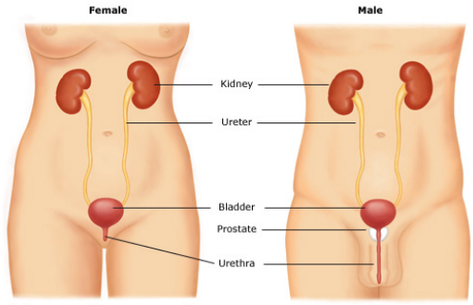
The urinary bladder wall is composed of smooth muscle (the detrusor). It has a normal capacity of 400-500mL and distension up to this volume has negligible increase in intravesical pressure.

There are 2 sphincters which enable continence (See Fig. 1)

• The bladder neck - a continuation of the bladder wall smooth muscle and therefore involuntary.
• A second sphincter, which in the male extends from the prostate to the membranous urethra and in females is mid urethral. This is a voluntary sphincter and is composed of striated muscle.

Figure 1 – Sphincter mechanism

Continence is also maintained by the levator muscles of the pelvic floor.



Innervation of the lower urinary tract consists of both autonomic and somatic nervous supply:

• Parasympathetic supply from S2-S4 causing contraction of bladder smooth muscle. The parasympathetic system also inhibits sympathetic stimulation on the internal urethral sphincter allowing it to relax. Therefore, the overall parasympathetic response is to allow micturition to occur.
• Sympathetic supply, via the presacral lumbar sympathetic ganglia and arising from T10-L2, inhibits bladder smooth muscle contraction and stimulates the internal urethral sphincter to remain closed. Therefore, the overall sympathetic response is to facilitate storage.
• Somatic motor supply from S2-S3 (pudendal nerve). The pudendal nerve originates in the Nucleus of Onuf (distinct area of the ventral anterior horn of the sacral region) and allows voluntary action on the external urethral sphincter and the levator ani.

Additionally there are corresponding afferent fibres which travel back to the cord and onwards to the pontine micturition centre and the cerebral cortex.

As the volume of urine in the bladder increases beyond 350mL, stretch receptors within the detrusor transmit the sensation of fullness to the sacral cord which contains the sacral reflex centre and a motor reflex is initiated. This causes bladder contractions and is responsible for voiding in young children. When the pontine micturition centre matures following toilet training voluntary continence is achieved at around age 3-4 years.

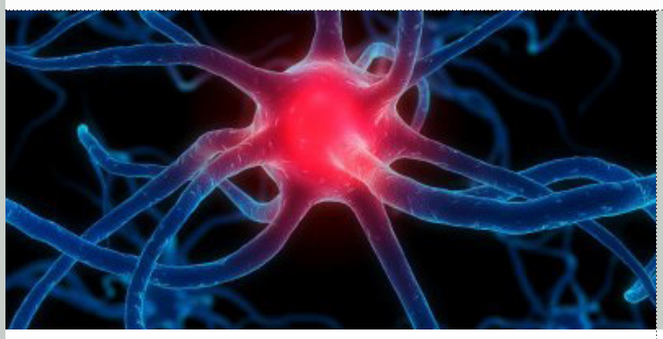
For voluntary micturition to occur there must be co-ordinated signals which result in simultaneous detrusor contraction and sphincter relaxation. This occurs at the level of the pons in the pontine micturition centre which ensures the bladder and sphincters work in synergy.

Similarly, co-ordination is required to allow storage of urine, achieved by relaxation of the detrusor and contraction of the sphincter. The micturition control centre of the frontal lobes voluntarily suppresses the spinal reflex until such a time that micturition is socially appropriate.

In summary, therefore, normal function of the lower urinary tract requires synergistic function between the detrusor muscle and the sphincter and consequently the neuropathic bladder and its associated problems occur when synergy is lost.

THE NEUROPATHIC BLADDER

J Moore



The Neuropathic Bladder.
Urology.

Spectrum of Disorders

Many neurological disorders are associated with bladder dysfunction. The pattern of lower urinary tract dysfunction can be predicted from the anatomical site of the lesion although significant variability can exist: See below (Fig. 2)

Site of Lesion	Neurological Disorder	Pattern of Dysfunction	Symptoms
Cerebral	Cerebro-vascular accident (CVA)*, brain tumours, head injury, dementia, cerebral palsy	Reduced cortical inhibition results in detrusor hyperreflexia/overactivity as the sacral reflex remains intact. These are 'safe' low-pressure bladders	Frequency, urgency, urge incontinence. Urine storage difficulties
Pontine	Parkinson's disease, multiple system atrophy (MSA), multiple sclerosis (MS)	Detrusor overactivity, detrusor underactivity, detrusor syphincter dyssynergia and external sphincter relaxation	Paradoxical retention
Suprasacral	Spinal cord injury*, MS	Lesions above T12-spastic bladder Lesions below T12-areflexic bladder Can result in 'unsafe' high-pressure bladders with risk of upper tract damage.	Reflex bladder (spontaneous voiding) Overflow incontinence
Sacral and peripheral lesions	Spina bifida, MS, diabetes mellitus, AIDS, iatrogenic (radical pelvic surgery)**	Detrusor areflexia and sphincter weakness These are 'safe' low-pressure bladders	Painless retention and overflow incontinence. Stress incontinence *(Bladder symptoms unlikely to occur in isolation)*

Figure 2 – Conditions associated with neurogenic bladder

**Initially in the acute phase following a CVA/spinal cord injury, there is cerebral shock and this is associated with detrusor areflexia due to loss of reflexes below the level of the lesion. Therefore, the patient may develop acute urinary retention and require an indwelling catheter in the early days after the insult*

***Post pelvic surgery for example, radical hysterectomy, abdominoperineal resection, radical prostatectomy, bladder dysfunction is common. However, with careful attention to continence management, up to 80% of patients will regain function with time.*

Clearly the breadth of these conditions makes the neuropathic bladder a challenge to the urologist to both diagnose and manage.

Presentation

In the overactive bladder, the intravesical pressure increases during filling and voiding. At the opposite end of the neuropathic bladder spectrum, the underactive bladder generates only very low pressures during filling and voiding.

Firstly consider the overactive bladder. When bladder pressure exceeds sphincter pressure then incontinence ensues. However, if sphincter pressure exceeds bladder pressure then retention will occur with the associated sequelae of urinary tract infections, hydronephrosis and renal impairment. This is known as detrusor sphincter dyssynergia or DSD.

The underactive bladder is unable to generate sufficient pressure to empty. These patients may present with retention and consequences as detailed above, however, if this is low pressure chronic retention, there will be no hydronephrosis or subsequent renal failure. Post micturition there is likely to be a high residual volume. A rise in intra-abdominal pressure such as coughing, sneezing may cause incontinence.

Problems associated with storage of urine manifest as frequency, urgency, nocturia and incontinence. Problems with voiding include, poor flow and intermittent stream, hesitancy, straining, and terminal dribbling. There may of course commonly be overlap between these with patients presenting with both types of symptom.

THE NEUROPATHIC BLADDER

J Moore

Investigations

Investigation of the neuropathic bladder begins with full history and examination. History of specific relevance would include: See figure 3

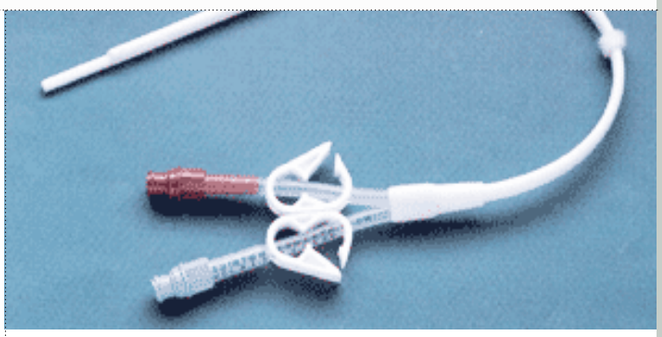
- **Details of known congenital abnormalities and/or known neurological problems and general past medical history**
- **Symptoms of storage or voiding**
- **Bladder sensation**
- **Previous related urinary tract problems, investigations and management**
- **Bowel history including associated faecal incontinence and anorectal sensation**
- **Sexual history/erectile dysfunction**
- **Medication known to affect the urinary tract**
- **Lifestyle factors-diet, oral fluid intake, alcohol**
- **Effect of the urinary symptoms on quality of life.**

Figure 3: Specific points for history taking

Physical examination will include full abdominal and neurological examinations. Urine microscopy, culture and sensitivity and cytology may help to identify infections or carcinoma in situ which can cause irritative symptoms. Baseline renal function should be documented and a renal tract ultrasound may also be valuable. The patient should complete a voiding diary as this can be insightful.

The simplest measure of lower urinary tract function is a urinary flow rate expressed in mL/sec, often followed by a post micturition bladder scan to determine if the bladder has been fully emptied. This is mainly used to demonstrate bladder outflow obstruction, however, a reduced flow (<15 ml/s) may also be secondary to a hypotonic bladder. The two cannot be distinguished on a simple flow rate, however, as a urinary flow rate is a simple non-invasive test and should be performed before more complicated invasive studies.

Urinary flow rate is one component part of urodynamic studies. (Note that, the term ''urodynamics'' encompasses any investigation of urinary tract function, although it is often used incorrectly as a synonym for cystometry.) In neuropathic patients the gold standard investigation is pressure/flow cystometry. The main aim is to reproduce the patient's symptoms to examine which part of the lower urinary tract is causative. The activity of the bladder during both filling and voiding can be assessed.



A dual lumen intra-vesical catheter is sited and the initial residual volume recorded. The bladder is filled at the required rate and a continual recording of the pressure is measured (P_{ves}). The patient is subsequently instructed to suppress the desire to void and to indicate any sensation of fullness and urgency. First sensation of bladder filling, first desire to void, strong desire to void and maximum cystometric capacity should be recorded. The voiding phase of the study begins when the permission to void instruction has been given or when spontaneous voiding begins. A catheter placed in the rectum measures the intra-abdominal pressure (P_{abd})and by subtraction the detrusor pressure (P_{det})is isolated from the intravesical pressure.

In patients with spinal cord injury above T6, there is a risk of autonomic dysreflexia. This condition can be life-threatening and manifests as bradycardia, headache, flushing and sweating above the lesion. It can be precipitated by spontaneous bladder overdistension in these patients by, for example, a blocked catheter, bladder calculus or whilst performing cystometry. During cystometry, it can be avoided by slow filling of the bladder however, an emergency protocol should be in place in centres performing this investigation on spinal injury patients.

Simple bladder pressure studies are enhanced with contrast media and radiological screening and this is known as videocystometography (VCMG). This yields additional anatomical information on the bladder and urethra and may identify vesico-ureteric reflux, the level of any obstruction, urethral mobility and stability of bladder base.

Electromyography, another test that may be considered, measures the electrical activity of the striated muscle of the pelvic floors but is often impaired by the high incidence of artefact and therefore only very rarely used due to the difficulty of interpretation. It is performed at the same time as cystometry.

It is important to remember that the neuropathic patient may also develop common lower urinary tract disorders that afflict the normal population such as bladder outflow obstruction secondary to benign prostatic hypertrophy or stress incontinence in the post menopausal woman. These may complicate investigation findings and subsequent management but should not be overlooked. It is also worth noting that with disease/symptom progression that urodynamic studies may need repeating at appropriate intervals and management adjusted accordingly.

THE NEUROPATHIC BLADDER

J Moore

Management of the Neuropathic Bladder

Following thorough investigation, exclusion of reversible causes and assessing the impact of the symptoms and expected benefit of intervention, treatment modalities should be considered. The primary goals of treatment should be: See Figure 4:

- **To prevent damage to the upper renal tract**
- **To reduce incontinence**
- **To improve quality of life.**

Figure 4: Aims of Management in patients with NLUTD

Management strategies should always be planned using the following hierarchy:

1. Conservative measures
2. Medical
3. Surgical

Conservative measures involve pelvic floor exercises, bladder retraining, attention to fluid intake and dietary modifications.

Catheterisation (indwelling urethral catheter, suprapubic catheter or clean intermittent self-catheterisation depending on the individual patient) or condom sheath is commonly involved in the management of the neuropathic bladder. Catheters may be a short or long term measure and their associated benefits and risks should be taken into careful consideration. Indwelling urethral catheters have obvious and immediate effect for both ends of the neuropathic spectrum that is, the incontinent patient and the patient in retention. However, they are not without complications which include, urinary tract infections, bladder spasms and bypassing, haematuria, bladder stones and long term inflammation possibly leading to malignancy. Suprapubic catheters have similar risks to urethral catheters but may cause less bladder spasm as the balloon does not lie on the trigone. They may also be easier to manage by the patient due to their more superior lying position and may be responsible for fewer UTI's as they are not in contact with the perineum. Preferable to both types of indwelling catheter is clean intermittent self catheterisation (CISC).

The Neuropathic Bladder. Urology.

Its use mimics normal bladder function whether it is carried out by timed or volume drainage. It reduces the intravesical pressure and prevents damage to the upper tract which can occur at intravesical pressures as low as 40cm H2O. The patient requires good dexterity and cognitive ability to perform this technique but when able, it is associated with fewer UTIs, freedom from catheter bags, autonomy and does not impede sexual relationships. The complications of CISC include urethral trauma and strictures.

Medical management may involve

- anticholinergics
- antispasmodics
- oestrogen derivatives
- tricyclic antidepressants

Anticholinergics are competitive muscarinic receptor antagonists. They exert their effect by reducing spontaneous detrusor activity during filling and reducing Pdet. Examples include, solifenacin, tolterodine and trospium. Side effects include, dry mouth, palpitations, blurred vision, flushing and constipation. They are contra-indicated in narrow-angle glaucoma and myasthenia gravis.

Antispasmodics relax bladder smooth muscle. This effect increases the bladder capacity and decreases urge incontinence. Oxybutynin is the most commonly used and its effectiveness is attributed to a combination of anticholinergic (M3-selective receptor subtype antagonism), local anaesthetic, calcium-channel-blocking activity in addition to antispasmodic properties.

Oestrogen deficiency leads to urethral hypermobility. Use of oestrogen derivatives therefore, usually applied locally, up-regulates expression of alpha-adrenergic receptors in the urethral wall and surrounding pelvic floor and so provides urethral support when these receptors are stimulated. An example is estradiol.

Tricyclic antidepressants for example imipramine increase bladder outflow resistance and also act on the bladder to inhibit contractions. Imipramine can be used synergistically with oxybutynin, however the side effects may not be well tolerated.

THE NEUROPATHIC BLADDER

J Moore

Patients with predominantly urge incontinence may benefit from Botulinum A toxin injections, neuromodulation, detrusor myomectomy or bladder augmentation. Injections of Botulinum A toxin directly into the detrusor have been shown to have good symptomatic effect. The injections are usually administered via flexible cystoscopy and repeated at 6-12 monthly intervals to maintain the response. Pharmacologically, it blocks presynaptic release of acetycholine and the neuromuscular junction of parasympathetic nerves supplying the detrusor.

Karsenty et al. (2008) carried out a systematic review of 18 articles on the efficacy of Botulinum A in bladder overactivity of neurogenic origin. They conclude that Botulinum Toxin A produces clinically significant improvement in symptoms and improvement in urodynamic parameters. In addition, it is well tolerated. They suggest that further randomised control studies are necessary to assess duration of effect and interval between treatments[1].

Intravesical capsaicin has also been shown to reduce detrusor overactivity.

Neuromodulation (also referred to as a bladder pacemaker) is a method of directly stimulating muscles involved in lower urinary tract function by means of electrodes placed on select nerves. A nerve stimulator is implanted on the efferent fibres of S2-S4 and an external radiotransmitter is activated when voiding is desired.

Bladder augmentation (clam augmentation cystoplasty) involves opening the bladder and repairing the defect with bowel, usually ileum. This impairs detrusor contraction, reduces Pdet and increases bladder capacity.

In patients with predominantly stress incontinence, management options include urethral bulking, tapes/slings and artificial urinary sphincters (AUS).

In the most extreme cases, urinary diversion may be the last option available when other options have failed and to redeem the patient's quality of life.

In conclusion the physiology of the lower urinary tract and the investigation and management of patients with neurogenic lower urinary tract dysfunction is complicated. The difficulty lies in the breadth of presentations requiring different approaches to management. However, it is not uncommon to encounter, patient with a neuropathic cause for symptoms. It is both challenging and satisfying to investigate, manage and ultimately improve long term morbidity and quality of life. These patients are likely to be longstanding and have progressive symptoms. Adopting an empathetic approach to disabling symptoms is essential to establish a doctor/patient relationship and develop management strategies.

EMQs for the Neuropathic Bladder

Theme: Bladder and Sphincter function in neurological disorders

Options

a) High pressure sphincter, high pressure bladder

b) High pressure sphincter, low pressure bladder

c) Low pressure sphincter, high pressure bladder

d) Low pressure sphincter, low pressure bladder

e) Detrusor sphincter dyssynergia (DSD)

For each of the patients below, select the single most likely diagnosis from the options listed above. Each option may be used once, more than once or not at all.

1. A 36 year old lady who is wheel chair bound is referred by her GP who has treated her for UTI on 4 occasions in the last 6 months. She complains of urinary incontinence when transferring from her wheelchair or when coughing.

2. A 25 year old man with multiple sclerosis for the last 5 years leaks urine involuntarily. On voluntary micturition he frequently only passes small volumes and has been treated recurrently for UTI by his GP. Recent blood tests have shown deteriorating renal function.

3. A 47 year old lady who previously suffered a spinal cord injury complains of increasing urinary incontinence. She has tried more frequent voiding but this has not improved the problem.

THE NEUROPATHIC BLADDER

J Moore

Answers

1. D – This patient describes incontinence on increase of intra-abdominal pressure. At rest, the detrusor is areflexic and does not generate enough pressure to empty and therefore urinary stasis predisposes to recurrent UTIs.

2. E – In this case, there is a high pressure bladder and sphincter which puts back pressure on the kidneys causing renal failure and as the situation worsens hydronephrosis will develop. In DSD, the sphincter pressure is higher than the bladder during voiding and therefore the bladder cannot empty efficiently.

3. C – In this case the bladder is constantly squeezing against a non-resistant sphincter so even small amounts of urine in the bladder will be able to pass.

Theme: Management of the Neuropathic Bladder

Options

a) Intermittent Self Catheterization
b) Urethral long term catheter
c) Suprapubic catheter
d) Transvaginal tape (TVT)
e) Consideration for an Artificial Urinary sphincter

For each of the patients below, select the single most likely diagnosis from the options listed above. Each option may be used once, more than once or not at all.

1. A 57 year old woman who has suffered from urinary incontinence since a spinal cord injury 10 years ago. She is constantly wet. Trials of intermittent self catheterization and anticholinergic medications have failed to improve the symptoms.

2. A 34 year old man with spina bifida has had ongoing problems with poor bladder emptying. He has used intermittent self catheterization for some time and also had treatment with intravesical Botulinum toxin but the problem persists.

The Neuropathic Bladder.
Urology.

Answers

1. D – in this case, the problem is most likely to lie at the level of the sphincter (low pressure sphincter) as the scenario tells us that interventions directed at the bladder have failed to improve the situation. As the patient is middle-aged, catheterization could have complications over the long term and a more definitive procedure would be to place a TVT.

2. E – another young patient who requires a definitive management strategy to avoid complications of long term catheters. Use of a catheter in this patient may cause an acquired hypospadias ('kippering' of the penis) in the case of a urethral catheter, recurrent UTIs, catheter blockage, bladder stones or bladder cancer. An AUS can improve incontinence in over 60% of patients and as high as 90%

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TRACHEOSTOMIES: HOW TO SAVE YOUR NECK

A Gupta

Tracheostomies: How to Save Your Neck. Otorhinolaryngology & Neck Surgery.

Tracheostomy is one of the most common surgical procedures performed on critically ill pateints (1,2). Even if you are not working in an ENT or thoracic surgery firm there is a very high chance of encountering a patient who has one. This short article is aimed at giving a simple overveiw of essential knowlege regarding tacheostomies. Common questions in modern MRCS and DOHNS exams are:

1. What are the indications for a tracheostomy?

2. How is the procedure done?

3. What are the common complications?

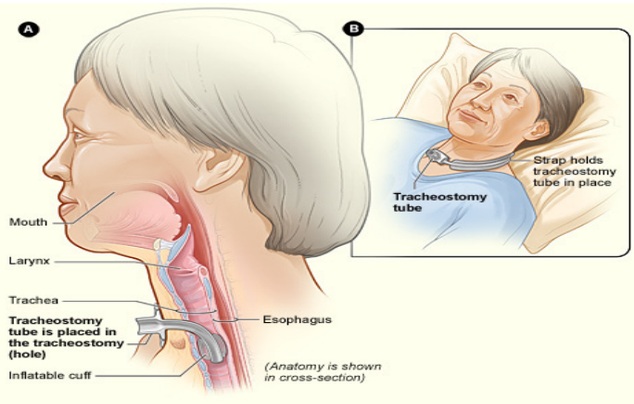
4. What are the different types of tubes that are used?

5. Emergency care?

Tabel 1 lists the indications for tacheostomy.

Indication	Examples
Protection of tracheobroncial tree	Head and neck surgery (oro-hypopharyngeal or laryngeal surgery) Neurological (myasthenia gravis or MS) Trauma (burns to face/ chest injury) Coma (Drug OD/head injury)
Relief of airway obstruction	Trauma (trachea, larynx, unstable facial fractures) Infection (acute epiglititis) Congenital (subglotic stenosis, haemangioma or severe laryngomalacia) Forgein body Bilateral vocal cord palsy
Respiratory assistance	Reduction of airway dead space (70%). Facilitate weaning off ventilation support Suction of secretions and tracheal toilet

Table 1: indications for tracheostomy (3)



Tracheostomy techniques

1. Surgical Tracheotomy

This is an open technique performed under either local or general anesthesia in an operating theatre. The procedure is shown in Box 1. It is important to give serious consideration to performing a tracheostomy under local anaesthesia when the airway is tenuous. In particular with stridor due to a laryngeal tumour, bilateral palsy, epiglotitis or arytenoid fixation as induction for a general anaesthesia can precipitate a crisis in which the airway can be difficult to maintain. There are a number of complications associated with an open surgical tacheotomy. These can be split into early and late (see table 2). There are no absolute contra-indications to a surgical tracheostomy, but a number of factors such as pateints with an obese neck, or severe cervical spondylosis do make it techniqually more difficult.

Early Complicatons	Late Complications
Haemorrhage/Haematoma	Delayed haemorrhage
Tube displacement	Granuloma formation at stoma site

Box 1 : Surgical tracheostomy procedure (3)

Position : *Moderate neck extension and sand bags under shoulders*
Incision: *Transverse, mid way between cricothyroid cartilage and suprasternal notch*
Procedure: *Cut though skin and superficial fascia. Split pre-tracheal fascia longitudinally. Retract the strap muscle laterally. The isthmus of the thyroid may occasionally need to be retracted. The cartilage of the trachea will now be visible. A window is created between the 2nd and 4th tracheal rings and the tracheostomy tube inserted as the ET tube is removed. It is important to preserve the first ring.*
Closure: *Stay sutures secure the tracheostomy tube as well as Velcro straps placed around the neck help to keep the tube in position. The skin edges are close but not tightly around the tube otherwise surgical emphysema may occur.*
Post-op : CXR, nurse upright if possible, with regular suction

Obstruction	Infection (cellulitis/pulmonary)
Pneumothorax	Tube displacement
Pneumomediastinum	Tracheal stenosis
Surgical emphysema	Subglottic stenosis
Aspiration	Tracheomalacia
Decannulation	Fistula formation (tracheo-oesaphageal/tracheocutanoues/tracho-arterial)
	Vocal cord palsy

Table 2 : Complications of open surgical tracheostomy (3,4)

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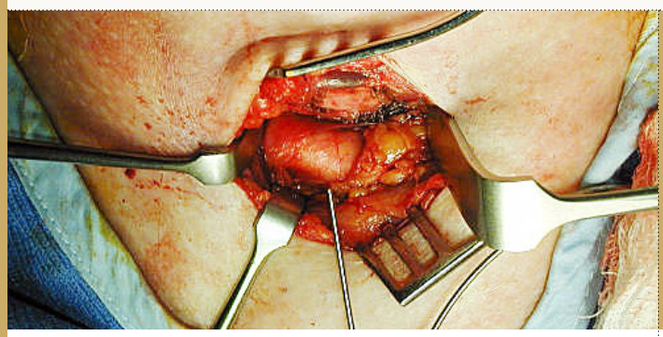
A Gupta



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2. Percutaneous Tracheostomy

This technique is being increasingly used in an ITU setting as an elective procedure to aid weaning off mechanical ventilation. A number of studies have found no significant improvment by using surgical techniques, with percuteanous techniques being associated with fewer complications (5). Percutaneous tracheostomy is becoming more popular as it is more time and cost effective compared with surgical techniques, furthermore it reduces the need for critically ill pateints to be transfered out of the ICU. Percutaneous trachemostoy was first described in 1955 by Sheldon et al (6). In recent times percutaneous dilatational tracheostomy (PDT) has become the most popular (7). The procedure is outlined in box 2. Complictions are similar to those of surgical tracheostomy. The relatice contraindications are a very thick set neck or short neck.

Box 2 : PDT procudure

3. Emergency cricothyroidotmoy and needle cricothyroidotomy

If attempts at naso/orotracheal intubation fail then either cricothyroidomy or needle cricothyroidotmy can be attempted depending on equipment and expertese present. Both of these proceudres are outlined in the Advanced Trauma Life Support course manual. Using either technique does not result in a definative airway and a surgical tracheosomy will be required in both situations once the patient is stablised(3).

Position : *Neck extended and sand/fluid bag under shoulders. The patient must be appropriately anesthetised and anatomical landmarks drawn on. Local anaesthetic with adrenaline infiltrated into the overlying skin.*
Incision: *1cm transverse incision between the 2nd and 3rd tracheal rings.*
Procedure : *An introducer needle and syringe are advanced at 45 degrees to the skin until air is aspirated from the trachea. A guide wire is passed down the needle and two dilators are used. Once the second dilator is in place the guide wire is removed and another set of dilators are passed over the first until a large enough opening is made to fit an appropriate tracheostomy tube. Make sure plenty of lubricating jelly is used and if the skin impedes placement, the incision can be extended slightly. As the tracheostomy tube is place into the trachea (under direct vision), the endotracheal tube can be removed and the ventilator connected.*
Closure : *Tapes and ties to secure the tube.*
Post-op : Listen for breath sounds, CXR

4. Mini Tracheostomy

Mini tracheostmy involves placement of a small tracheostomy tube or large bore cannula via the cricothyroid membrane. The tube is well tollerated with minimal sedation and allows speech and coughing. Effective tracheal toilet is the main indication. The technique is similar to that of an emergency cricotyroidotomy. It is absolutely essential to have good extension of the neck in order to tense the cricothyroid membane(3).

Types of tracheostmomy tubes

There are many types of tracheostomy tubes used. They can be broadly split into the following catogries

1. Lumen (Single or Double)

Single lumen tracheostomy tubes maximise the inner lumen and so decrease airway resistance. They are for short-term use but can become blocked with secretions. Single lumen tubes must be replaced every 5 to 7 days

Double lumen tracheostomy tubes have an inner lumen which is removeable. This makes it easier to clear secretions but the functional diameter is less so there is slightly greater resistance increasing the work of breathing. Double lumen tubes can stay in place for upto 29 days

2. Cuff (Cuffed or Uncuffed)

Uncuffed tubes allow air to move into the upper airway around the tracheostomy tube. They are sutible for long-term use and if the tracheostomy tube becomes blocked they allow respiration to continue via the upper airways. Because there is no cuff there is a much lower incidence of traceal mucosal damage. Uncuffed tubes are not suitable for patients who are at risk of aspiration or still require ventilation.

Cuffed tubes form a seal with the tracheal wall, allowing air to move solely through the tracheostomy. These tubes are used in pateints who require ventilation or who are at risk of aspiration. Problems are encoutered if the tube becomes blocked as this results in complete airway obstruction. In addition, prolonged pressure of the cuff on the surrounding tracheal tissue can lead to stenosis, erosions or ulcers. Adult tracheostomy tubes need to be deflated every 4-6 hours to reduce the likelihood of necrosis and scar formation leading to subglottic stenosis.

3. Fenestrated

If a tube is fenestrated it means that there is a hole in top of the tube as it bends between the horizontal and vertcal planethe (several small holes or one big hole). The hole/s allow air the move into the upper airway. Fenestrated tubes can reduce the work of breathing and allow vocalisation. These tubes are used when a patient is ready to be weaned off temporary trachiostomies or those who are unable to tolerate a speaking valve. There is a risk of aspiration and also granuloma formation at the site of the fenestrations.

TRACHEOSTOMIES: HOW TO SAVE YOUR NECK

A Gupta

4. Adjustable

These tubes allow the lengh of the tube to be changed and are used for patients with atypical anatomy or 'deep set tracheas'. At present, these only come in a single lumen. There is a high risk of decanulation when adjusting the tube length.

You may come across metal tubes (Silver Nigus). These are old fashioned tubes but are still used (usually patient's preference). Treat them as you would single lumen uncuffed tubes.

Common Oncall Bleeps

1. "Doctor, doctor the tube has got blocked"
or "Doctor the patient is very short of breath"

If this happens within a week of the tracheostomy being done this can be very serious as the tract is not well formed and removing the tube may lead to collapse. If there is an inner tube the first action is to remove this. Remove any debris and gently pass a suction tube down the tracheosomty to clear any hard secretion. When passing a suction tube be gentle and if resistance is met then do not be forceful. If the pateint begins to cough violently then you may have reached the carina. If things are no better then consider removing the whole tube. At this point it would be a good idea to use a tracheal dilator in place of the tube to keep the tracheostomy patent.

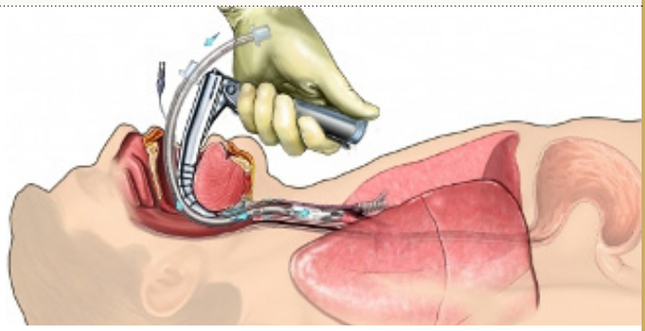
In some patients there will remain an airway, all-be-it tenuous, through the larynx (NOT in a laryngectomy patient!) and this might be enough until more experienced help arrives. If dilators do not provide any airway then it is worth trying to reinsert a tracheostomy tube in the right place (this is done by passing it in the HORIZONTAL plane until the end of the introducer hits the back wall of the trachea and it naturally feeds down the trechea – to place the tracheostomy tube in the vertical plane runs the risk of causing a false passage.

This is easy to do, particularly in the first few days after a tracheostomy.
Lastly an attempt can be made to obtain an airway using endotracheal intubation. Rarely bronchoscopy is needed to create an airway and remove a foreign body or crust if this has blocked the airway more distally.

A crash team should be called earlier rather than later as one option may be to obtain an airway using endotracheal intubation.

Remember to consider other causes of dyspnea as the tracheostomy is not always the cause, i.e lower respiratract infections.

While waiting for and help you can put the patient on oxygen via a tracheostomy mask.



2. "Doctor, doctor the tube has fallen out"

A crash team should be called earlier rather than later as one option may be to obtain an airway using endotracheal intubation.

When approaching a patient who is decannulated, it is important to stay calm. Ask the pateint to breath normally through the stoma or even through their nose and mouth. If the tube has recently come out it maybe be a simple case of putting it back. See the above section on how to do this. If this doesn't work , then a tube that is one size smaller can be used. If this fails, a tracheal dilator can be placed in the stoma to keep it patent and senior help should be sought.

3. "The site is bleeing"

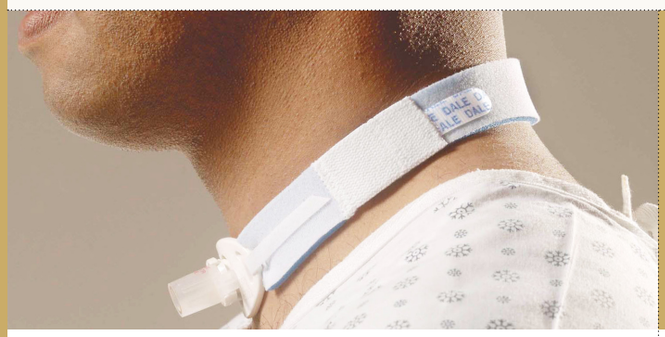
Bleeding can occur at anytime from immediatly post-operatively to months after placement of the tube. A small amount of bleeding around the cannula site post-operatively can be dealt with by pressure and packing. If the bleeding is heavy then an ABCDE approach must be used to assess the patient. Early bleeding can be from granulation tissue. If bleeding is not controlled by wound care and pressure then formal surgical exploration maybe required. In some cases of severe bleeding, hyperinflation of the tacheostomy cuff can have a tamponade effect but senior advice should always be sought. A late complication of prolonged use of cuffed tubes is formation of an arterial fistula. This is a rare but very serious complication.

4. CARDIAC ARREST!!

Treat the patient as any other. Do not remove the tracheostomy. Ventilation can be established by putting an Ambu bag directly onto the tracheostomy tube. If unable to ventilate, check the inner cannula is clear. If still unable to ventilate, try gentle suction of the trachea. If you are still not able to ventilate you will need to assess for oral intubation.

TRACHEOSTOMIES: HOW TO SAVE YOUR NECK

A Gupta



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Tracheostomy Questions.

Indications for Tracheosotomy
Below is a series of statements. Mark each true or false.

1. Trauma to the neck and face is a contraindication for tracheostomy insertion.

2. Tracheostomy can reduce airway dead space by upto 70%.

3. Tracheostomy is used in patients with myasthenia gravis.

4. Severe facial fractures are contraindication for tracheostomy insertion.

Answers

1. F 2. T 3. T 4. F

Tracheostomy EMQ

For the cases below choose the most appropriate answer. Each answer can be used once, more than once or not at all.

A. Needle cricothyroidotomy
B. Percutaneous tracheostomy
C. Mini Tracheostomy
D. Surgical Tracheostomy

1. A 38 year old man has been on the ITU for 6 days being intubated and ventilated after a severe asthma attack. He has been making good progress and it has been decided to start to wean him off his oxygen. Unfortunately there has been some difficulty doing this and he requires a tracheomstomy. Which type would be best?

2. A 57 year old obese man was involved in a road traffic incident and sustained a severe head injury. On admission he was incubated and ventilated. 10 days on, the ITU team require a tracheostomy to help wean him off his oxygen. Which technique would be best?

3. A 22 year old woman has been involved in a house fire. She has sustained severe burns to her face and thorax. On arrival to AE she is in respiratory distress and her O2 sats are 88% on 15L. The ITU SHO has failed to intubate the patient 3 times and an airway is urgently needed. Which tracheostomy should you use?

Answers

1. B – Cheap and easy to do
2. D – He will have a fat neck so best done in theatre
3. A – Emergency situation

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Author

Mr Ameet Gupta (MBBS, MRCS),
Kings Mill Hospital, Sutton-in-Ashfield,
Nottingham, NG17
Email: ameetgupta@nhs.net

DIAGNOSIS & MANAGEMENT OF MIDGUT MALROTATION IN INFANCY

M A Quail, R Dias

Diagnosis & Management Of Midgut Malrotation in Infancy.
Paediatric Surgery.

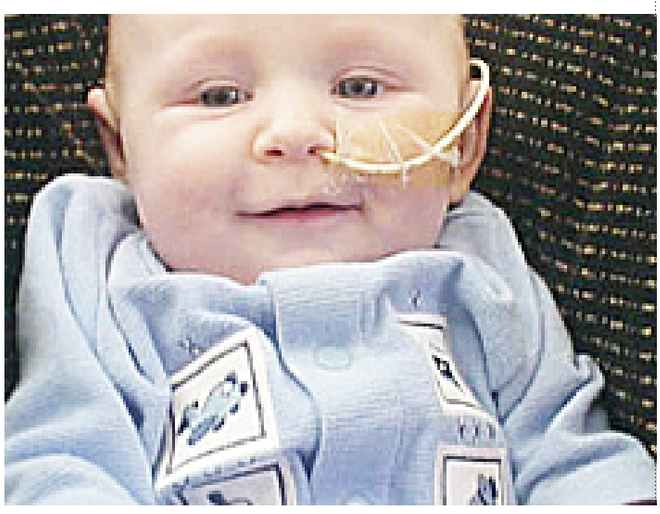
Abstract

In this report we present a clinical vignette of midgut malrotation in a newborn infant. The infant presented initially with non-bilious emesis, progressing to bilious emesis on the 5th day of life. The diagnosis was suspected following a Doppler ultrasound examination of the superior mesenteric vessels and confirmed by upper gastrointestinal (UGI) contrast study. The infant underwent an exploratory laparotomy where malrotation was confirmed and a Ladd's procedure was performed. The diagnosis and management of bilious vomiting is discussed, emphasising that green emesis in an infant should be managed emergently.

Clinical Vignette

A male term infant was reviewed on day 3 of life by the paediatric SHO on the postnatal ward. The infant's mother reported that the baby persistently vomited his feeds. There was no evidence of blood or bile in the vomitus and it was not described as projectile. The baby appeared well and abdominal examination was unremarkable. There was no significant antenatal or family history.

The baby was admitted to the neonatal unit for feeding observation. The infant was unable to feed successfully by breast or bottle and had hyperbilirubinaemia (unconjugated) presumed secondary to inadequate oral intake. A nasogastric tube (NGT) was inserted and the volume of each feed was reduced. However, despite these measures, the infant continued to vomit intermittently over the following 36 hours. The patient's mother and the nurse now reported that his vomit had a green colour to it.



An abdominal X-Ray was performed, but this was inconclusive. The duty radiologist performed an abdominal ultrasound with Doppler examination of the mesenteric vessels. This suggested that the infant's superior mesenteric vein (SMV) lay to the left of the superior mesenteric artery (SMA), (Figure 1).

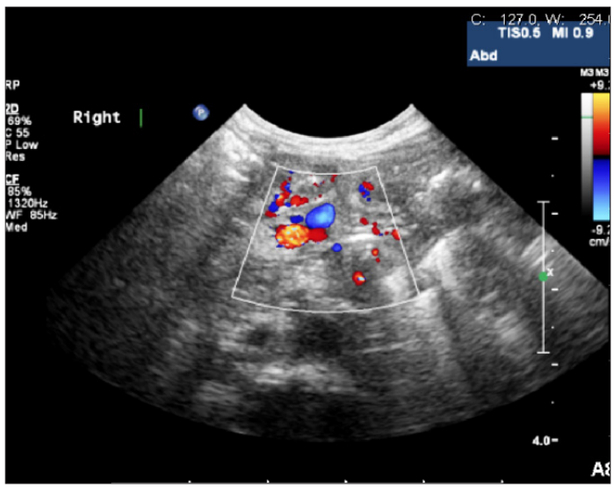


Figure 1. A Doppler ultrasound of the superior mesenteric vessels, showing the SMV (Blue) lying to the left of the SMA (Red).

Midgut malrotation was suspected due to the finding of bilious vomiting combined with the abnormal relationship between superior mesenteric vessels. An upper GI contrast study was performed with water-soluble contrast.

The contrast study demonstrated that the duodenum did not cross the left pedicle of L2. The duodenojejunal junction (DJJ) lay in the midline 2-3 cms inferior to the first part of the duodenum, indicative of midgut malrotation (Figure 2). The position of the DJJ is a critical component of the upper GI contrast study, and should be located to the left of the left vertical body pedicle at the level of the inferior margin of the duodenal bulb. ([1]) There was no radiological evidence of volvulus.

DIAGNOSIS & MANAGEMENT OF MIDGUT MALROTATION IN INFANCY

M A Quail, R Dias

Diagnosis & Management Of Midgut Malrotation in Infancys. Paediatric Surgery.

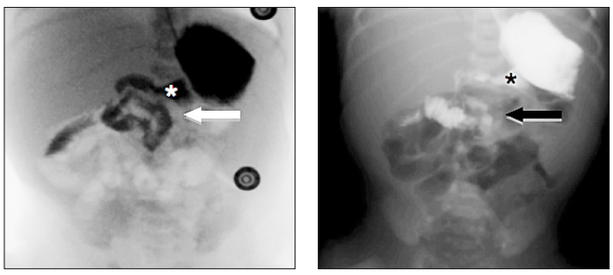


Figure 2. Upper GI contrast study. The position of the duodenojejunal junction (Arrow) relative to the duodenal bulb (Asterix) and vertebral bodies is demonstrated.

The baby was kept nil by mouth, the stomach was decompressed using a large bore NGT and IV fluids were commenced. The case was discussed with the paediatric surgical registrar who advised immediate transfer to their unit. The infant was reviewed and thereafter taken to theatre. At the time of laprotomy a malrotation without volvulus was noted. A Ladd's procedure and inversion appendectomy was performed.

Post operatively the baby was stable and was commenced on small volume enteral feeding 48 hrs after surgery. He tolerated this well and the feed volume was gradually increased. The infant's symptoms of vomiting did not return post operatively and he was discharged home on Day 15 of life.

Discussion

Intestinal malrotation is reported to occur in 1:500 live births and is due to aberration of the normal morphogenesis of the gut tube. An understanding of the embryology is useful and has been described elsewhere, however the explanations, which help describe the observed pathology, may not allow inference of the true underlying dysmorphogenesis. ([2,3]) Briefly, the embryological gut undergoes a series of rotations around the superior mesenteric artery before establishing final fixation points within the abdomen (Figure 3).

In malrotation both the duodenojejunal loop and caecocolic loop are abnormally fixated, having failed to complete their full rotation around the SMA axis, often with a high medially positioned caecum. Disordered attempts to fix the caecum lead to the observation of fibrous bands of tissue, 'Ladd's bands' extending from the caecum and right colon, crossing the duodenum to the retroperitoneum of the right upper quadrant. ([4]) These compressive bands, combined with torsion at the base of the midgut mesentery give rise to the clinical manifestations of obstruction and volvulus.

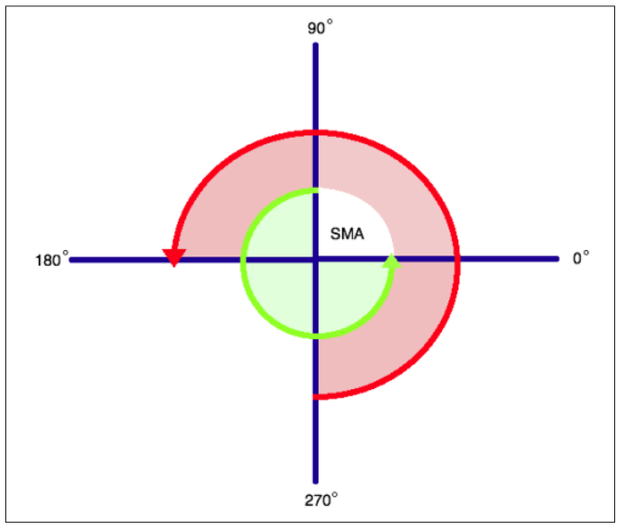


Figure 3. Normal rotation of the intestine during development around the superior mesenteric artery (SMA) axis. The caecocolic loop (Red) begins inferior to the SMA and the duodenojejunal loop (Green) begins superior to the SMA. Both loops undertake a 270° counter-directional rotation in stages (not shown) during development.

DIAGNOSIS & MANAGEMENT OF MIDGUT MALROTATION IN INFANCY

M A Quail, R Dias

Clinical Presentation

Up to 50% of cases of malrotation occur in the first week of life and 80% by one month. ([2, 5]) The classical presentation is that of an infant with bilious vomiting in 77% of cases, due to obstruction distal to the ligament of Trietz. ([5, 6]) Bilious vomiting is common in adults and its occurrence often elicits no concern from clinicians. This is not the case in infants, in whom the presence of bilious vomiting should be considered a surgical emergency until proven otherwise. The differential diagnosis of bilious emesis includes other surgical and non-surgical causes, including: Hirschsprung's disease, bowel atresia, meconium ileus, annular pancreas, milk inspissation, sepsis and rarely idiopathic bilious emesis. ([7, 8]) In an older child, intussusception should also be considered.

Alternative presentations for malrotation also include: non-bilious vomiting, diarrhoea, bloody stools, peritonitis and shock due to bowel infarction. ([2]) Older children and adults may also present with symptoms of malrotation, but symptoms may be insidious or intermittent, they may include failure to thrive, abdominal pain and vomiting. Patients' symptoms may have previously been ascribed to cyclical vomiting, recurrent abdominal pain, or even psychological problems. An incidental operative finding of chylous ascites in a neonate or child, often at herniotomy should also prompt the search for and exclusion of malrotation. ([9])

Clinical examination reveals an abdomen that is soft and nontender to palpation (50% of patients at presentation) until strangulation of bowel has occurred, after which, it becomes distended (36% at presentation), tender (14% at presentation) and stools may be bloodstained. ([4, 5]) The disorder of malrotation is associated with other problems of development including severe congenital heart disease, however it is often the congenital heart disease rather than malrotation, which first draws attention. The presence of digestive symptoms, in children with complex congenital heart disease, warrants prompt investigation for malrotation and volvulus. ([10])

Investigation

In an acutely unwell infant with peritonitis, time should not be wasted delaying surgical intervention to perform radiographic investigations as this may result in loss of viable gut and may ultimately result in a fatal outcome. In the case of a stable infant, an upper gastrointestinal (UGI) contrast study is the modality of choice. ([2, 11])

The UGI study can demonstrate the position of the duodenojejunal junction, which if normally situated can usually exclude malrotation. ([1]) However, there will remain a group of children in whom the diagnosis of malrotation will be difficult on the UGI study alone because of technical difficulties or because it can be difficult to differentiate some normal variations from true abnormalities of rotation. ([12]) Occassionally a lower GI contrast study may be needed to aid diagnosis by identifying the position of the caecum.

The plain abdominal film is occasionally abnormal but should not be relied upon to exclude malrotation, not least because typical signs of duodenal obstruction are often absent. ([12]) Similarly, although abdominal ultrasound can often provide supportive evidence regarding the relationship of superior mesenteric vessels (Figure 1), it should not be used as the primary or sole imaging modality as it misses the diagnosis in 15-30% of patients. However, if inverted superior mesenteric vessels are identified during any ultrasound examination, malrotation is likely. ([11])

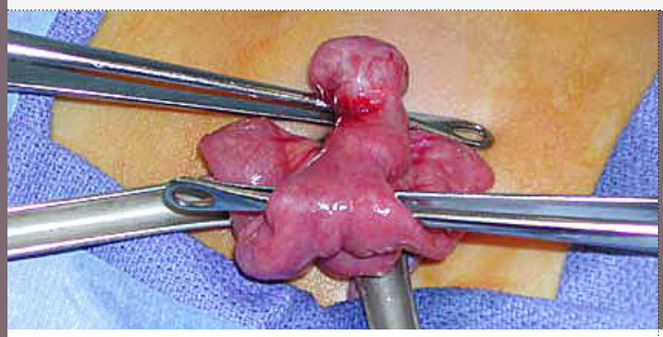
Surgical management

Unwell infants should be managed with appropriate input from neonatal and paediatric colleagues, with emphasis on effective stabilisation and urgent transfer. The surgical approach to malrotation with or without midgut volvulus is referred to as a "Ladd's procedure" ([6, 13]). Shew ([14]) has described the tenets of the procedure:

(1) Detorsion of the bowel when volvulus is present, (2) lysis of duodenal bands, (3) broadening the mesentery to separate the duodenum and caecum as far away as possible, (4) placement of the small bowel to the right side of the abdomen, and (5) placement of the colon to the left side of the abdomen. In the presence of midgut volvulus, the torsion pattern can be frequently difficult to determine by the appearance of the dilated bowel at the time of surgery. Invariably, the torsion is in a clockwise fashion from one to three full turns. Thereby, detorsion consists of "turning back the hands of time" with a counter-clockwise motion until the duodenum and caecum are in parallel. The Ladd's procedure in effect converts the patient from a state of malrotation to a state of broadened nonrotation, and not to that of normal rotation. At the completion of the Ladd's procedure, appendectomy is frequently performed, as the appendix will be abnormally positioned.

DIAGNOSIS & MANAGEMENT OF MIDGUT MALROTATION IN INFANCY

M A Quail, R Dias



Diagnosis & Management Of Midgut Malrotation in Infancys.
Paediatric Surgery.

In published case series of malrotation, evidence of volvulus at laparotomy ranged from 36-68%. ([5, 15]) It should be noted that the Ladd's procedure reduces, but does not eliminate the risk of late postoperative volvulus. The most common postoperative complication in one series was adhesive small bowel obstruction, occurring in 6%, most of which failed conservative management and required operative adhesiolysis. ([15]) The incidence in postoperative volvulus was 0.6%; previous studies have reported 2-7%, and certainly a past history of Ladd's procedure does not exclude recurrence. ([15])

Conclusion

Bilious emesis in a neonate is not analogous to that of an adult. A referral with this historical fact should prompt urgent investigation and management of intestinal obstruction with presumed intestinal malrotation and high suspicion of volvulus. Failure to diagnose and manage malrotation, which then progresses to volvulus, can result in the loss of the entire midgut. The investigation of choice for stable patients is UGI contrast study. The Ladd's procedure with good preoperative stabilisation is the definitive management for malrotation.

Multiple Choice Questions

(1) Correction of malrotation via Ladd's procedure eliminates the risk of volvulus.
T/F

(2) Bilious emesis in an infant often resolves without intervention.
T/F

(3) The most frequent postoperative complication following Ladd's procedure is recurrent volvulus.
T/F

(4) Detorsion of volvulus complicating malrotation most often requires rotation in a counter clockwise movement.
T/F

(5) Patients with malrotation often present with a normal clinical examination.
T/F

Answers

1. False - The Ladd's procedure reduces, but does not eliminate the incidence of postoperative volvulus.[1]

2. False - Bilious emesis is an ominous sign requiring emergent investigation and treatment for presumed intestinal obstruction.

3. False - Small bowel obstruction due to adhesions is more common. Post operative volvulus is rare.[2]

4. True - The torsion is invariably in a clockwise pattern of 1-3 turns, requiring counter-clockwise detorsion.[3]

5. True - The abdomen is soft and non tender in 50% of patients at presentation. Abdominal signs appear when strangulation has occurred causing abdominal distension and tenderness in 36% and 14% of patients at presentation respectively.[4]

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DIAGNOSIS & MANAGEMENT OF MIDGUT MALROTATION IN INFANCY

M A Quail, R Dias

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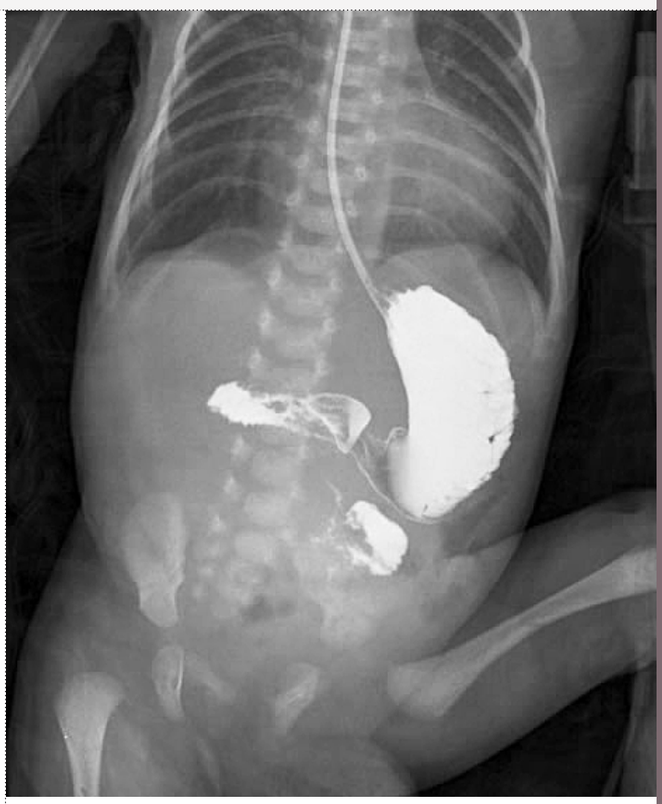

Author

Michael A. Quail, BSc (hons), MB ChB (hons), MRCPCH, MRCPE 1 & Ryan J. Dias, MB BS, DCH, MRCPCH. [2]
1. Cardiac Unit, Great Ormond Street Hospital for Children, NHS Trust, London
2. Neonatal Unit, Northwick Park Hospital, NHS Trust, Harrow

Corresponding Author

Dr. Michael Quail
Great Ormond Street Hospital, Cardiac Unit,
Great Ormond Street, London WC1N 3JH
Email: mquail@ucl.ac.uk

CASE-BASED DISCUSSION: CAUDA EQUINA SYNDROME

R Afinowi, R Sinha

Case-based Discussion: Cauda Equina Syndrome. Neurosurgery.

Abstract

Cauda Equina Syndrome is a relatively rare neurological syndrome presenting clinically with the hallmark symptoms of bladder, bowel and sexual dysfunction, most often resulting from compression of nerve roots within the lumbosacral spinal canal. Conversely, in spite of its relative infrequency, the personal, social and medico-legal implications for patients and medical practitioners alike are significant. Here we present a case of insidious onset, hence lacking the impact of an acute sudden presentation, cauda equina syndrome aiming to highlight the importance of recognizing this condition.

Case Vignette

Mrs CE is a normally fit and well 35 year-old who had seen her GP over a 5-month period complaining of low back pain and left sided sciatica of insidious onset. She now presents to the Emergency Department of her local hospital complaining of intolerable pain and urinary incontinence (dribbling) of 3 days duration. On referral to the on-call orthopaedic team for evaluation, she was found to have weakness of left ankle plantar flexion and an absent ankle jerk reflex, perianal numbness, loss of anal tone and voluntary contraction, and urinary retention with overflow incontinence (post-micturition residual volume 1000 ml).

Cauda Equina Syndrome (CES) describes a condition of altered bladder, bowel or sexual function with perianal or perineal "saddle" anaesthesia arising from compression of nerve roots within the lumbosacral spinal canal i.e. the cauda equina.

Incidence and Impact

CES is a relatively rare condition, the incidence in UK population studies has been quoted to be between 1 in 33,000 to 1 in 100,000[1-3]. Despite this, the potential for long-term disability from urinary, bowel, lower limb and sexual dysfunction can cause significant disruption to patients' personal, social and working lives. This is especially true for patients with CES who encounter delays in the diagnosis and management of their condition, during which time nerve root compression may be ongoing, resulting in permanent disability. Consequently, CES has a very prominent medico-legal profile, with damages settlements ranging up to £584,000[3]. Consequently, it follows that clinicians armed with a sound understanding of the condition, its clinical presentation and the need for urgent management are best placed to prevent the potentially disastrous sequelae for the patient.

Anatomy and Aetiopathogenesis

The cauda equina, its name derived from the Latin "horse's tail", is made up of terminal nerve roots held together in a common dural sac within the lumbosacral spinal canal. The spinal cord itself terminates as the conus medullaris, at the T12 to L2 vertebral level (most commonly L1), from where it is in continuity with the filum terminale. The cauda equina is hence a collection of the L1 to S5 peripheral nerve roots within the spinal canal and lesions of these roots present clinically with signs of a lower motor neuron lesion, affecting the bladder, bowel, perineal region and lower limbs to varying degrees[4, 5].

The commonest cited cause of CES is mechanical compression by a space-occupying lesion within the spinal canal, notably a prolapsed or herniated lumbar intervertebral disc (most commonly L4/5 or L5/S1). In addition, spinal canal stenosis of any cause may predispose to this condition[6].

The cauda equina appears to be particularly susceptible to mechanical compression for a number of reasons. The nerve roots within the spinal canal poses only a single layer of protective connective tissue (endoneurium) offering a relative lack of protection when compared to peripheral nerve roots. In addition, there is experimental evidence of a relative area of hypovascularity at the proximal portion of the cauda equina predisposing to ischaemic injury. The arterial blood supply to nerve roots was found to be from proximal and distal radicular arteries which formed an anastomosis in the proximal third of the nerve root suggesting a greater susceptibility to alterations of blood flow and consequent ischaemic damage in this anatomic area of the nerve root[4, 7]. It has also been demonstrated that mechanical compression of the nerve root reduces nutrient delivery by a combination of reduced blood flow and reduced diffusion from the surrounding cerebrospinal fluid.[8, 9]

CASE-BASED DISCUSSION: CAUDA EQUINA SYNDROME

R Afinowi, R Sinha

Causes of Cauda Equina Syndrome

- Lumbar intervertebral disc prolapse or herniation. Commonly L4/5, L5/S1
- Spinal canal stenosis: congenital, acquired, degenerative
- Trauma: Fractures, subluxation
- Neoplasms: Primary and metastatic tumours
- Myeloma
- Cystic lesions
- Vascular anomalies and lesions
- Infection: osteomyelitis, vertebral collapse, abscess, tuberculosis
- Iatrogenic: Spinal surgery, spinal anaesthesia, haematoma, gelfoam or epidural grafts, durotomy, spinal manipulation

Clinical Features

It is not uncommon for patients who develop a clinical CES to present with a background history of back pain and sciatica with or without documented established lower limb sensorimotor disturbance. The hallmark of a clinical diagnosis of CES is the onset of bladder, bowel or sexual dysfunction.

Three patterns of clinical presentation have been described[10, 11]:

- Clinical CES as the chief presenting feature of an acute lumbar intervertebral disc prolapse
- Sudden bowel, bladder or sexual dysfunction on a background of chronic back pain and sciatica
- Insidious and of gradual onset and progression, usually with back pain and sciatica

A useful clinical classification of prognostic importance is defining cauda equina syndrome as complete (CES-R) or incomplete (CES-I) based on the presence of established painless urinary retention with overflow incontinence and perineal 'saddle' sensory deficit.[2, 12]

Key Clinical Features

- Bladder dysfunction – Ranges from hesitancy, reduced sensation and poor stream (CES-I) to established painless urinary retention with overflow incontinence (CES-R)
- Perineal "saddle" sensory impairment – Uni- or bilateral hypoaesthesia or complete saddle anaesthesia to pin-prick testing
- Bowel dysfunction – Reduced or loss of anal tone, loss of voluntary contraction, incontinence, constipation
- Loss of bulbocavernosus reflex, anal "wink" reflex *
- Sexual dysfunction – Impotence, priapism

Associated Features:

- Back pain – Can be acute or chronic
- Sciatica – Uni- or bilateral or alternating (depending on the centrality /laterality of the compression)
- Flaccid lower limb motor weakness
- Diminished or absent deep tendon reflexes
- Lower limb sensory deficit

*The authors recommend assessing if a patient senses the passage and presence of a urinary catheter, as in deed, a catheter should be passed to allow for assessment and initial treatment of the painless urinary retention associated with established CES. Traction on the catheter can be used to assess the bulbocavernosus reflex (classically elicited by stimulating the glans penis or clitoris).

Case Vignette Part 2

An MRI of her lumbosacral spine was arranged within the hour. This showed extrusion of the L4/L5 intervertebral disc within the spinal canal. She was referred to the regional neurosurgical centre where she underwent an urgent decompressive laminectomy with discectomy. Mrs CE's bladder and bowel dysfunction did not resolve post-operatively. She is currently undergoing specialist urological rehabilitation and her future remains uncertain.

Investigation

A clinical suspicion of CES merits confirmatory imaging to facilitate urgent decompressive surgery[13]. Magnetic Resonance Imaging (MRI) is the investigation of choice defining nerve roots, cerebrospinal fluid space, soft tissue within the canal and space occupying lesions causing root compression[1].

CT and myelography are alternatives particularly where MRI is technically not feasible or unavailable[14, 15].

Management

A confirmed diagnosis of cauda equina syndrome warrants urgent specialist referral to a spinal or neurosurgical team for decompressive surgery of reversible causes[16, 17].

CASE-BASED DISCUSSION: CAUDA EQUINA SYNDROME

R Afinowi, R Sinha

The most common surgery performed is decompression of an intervertebral disc prolapse by microdiscectomy or laminectomy and discectomy. Particular care is taken to minimise handling (such as excessive traction by instruments) and avoid further damage to already compromised nerve roots. Other surgical interventions are dependent upon the aetiology at presentation[6].

Outcome

Outcome after decompressive surgery for disc prolapse is influenced by several factors, most importantly the pre-operative severity of symptoms (CES-I vs. CES-R). Complete cauda equina syndrome with established urinary retention (CES-R) is thought to be associated with a poor functional outcome, independent of the timing of surgery[2, 18, 19].

Controversies

The timing and urgency of surgery remains somewhat controversial, with further research limited by ethical and methodological issues[6,16]. Despite a widespread trend towards urgent surgery, it has been suggested that urgent surgery in patients with CES-R confers no benefit in terms of recovery of sphincter function and indeed, poses further hazards through performing a technically demanding procedure in a less than optimal out-of-hours setting. Urgent surgery however was advocated for CES-I[2,12].

This is in contrast to other studies which demonstrated outcome to be independent of the timing of surgery and the severity at clinical presentation (CES-R vs. CES-I) to be a poor indicator of outcome[18,19]. Other studies and re-analysis have suggested that the timing of surgery does confer benefits with a favourable outcome for both types of cauda equina syndrome if surgery is performed within 24 hours of the onset of symptoms[16, 20, 21] with others limiting this observation to predominantly CES-I[10]. The above illustrate discrepancy in the literature with regards to the timing of and benefit of surgery across the spectrum of cauda equina presentation. Despite the discrepancies highlighted, keeping a high index of clinical suspicion when confronted with the presenting features to maximise early diagnosis and subsequent time for definitive treatment at a neurosurgical/spinal unit seems the unifying principle in managing the burden of this syndrome.

Case-based Discussion: Cauda Equina Syndrome. Neurosurgery.

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CASE-BASED DISCUSSION: CAUDA EQUINA SYNDROME

R Afinowi, R Sinha

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MCQs

1. Prolapse lumbar intervertebral discs most commonly occur at
A. L1/2 and L2/3
B. L4/5 and L5/S1
C. L1/2 and L3/4
D. L2/3 and L3/4
E. L1/2 and L4/5

2. The hallmark of the clinical diagnosis of cauda equina syndrome is
A. Bladder dysfunction
B. Bowel dysfunction
C. Sexual dysfunction
D. Bladder and bowel dysfunction
E. Any of the above

3. Complete cauda equina syndrome is distinguished from incomplete cauda equina syndrome by the presence of
A. Painful urinary retention and sciatica
B. Painless urinary retention and sciatica
C. Painful urinary retention and perineal sensory deficit
D. Painless urinary retention and perineal sensory deficit
E. Painful urinary retention only

4. The investigation of choice for cauda equina syndrome is
A. MRI
B. CT
C. Myelography
D. Plain film Xray
E. Technetium-99 Bone scan

5. All of the following can cause cauda equina syndrome except
A. Lumbar disc prolapse
B. Trauma
C. Multiple Sclerosis
D. Neoplastic lesions
E. Tuberculosis

Answers

1. B
2. E
3. D
4. A
5. C

Authors

Rasheed Afinowi MSc MRCS
Core Surgical Trainee (Trauma & Orthopaedics)
Yorkshire Deanery

Rohitashwa Sinha MRCS
Neurosurgery Trainee
East of England Deanery

Correspondence to

Rasheed Afinowi
11 St James Court
Lock Keepers Court
Hull HU9 1QJ
R.Afinowi@doctors.org.uk
+44 796 1068 760

HOW TO PREPARE FOR MRCS PART B: SOME HELPFUL TIPS

A Titchener, P Kodumuri, N Sheikh, D Nikkhah

How to prepare for MRCS Part B: Some helpful Tips. Career Focus.

Recent successful candidates Andrew Titchener, Nomaan Sheikh, Preetham Kodumuri and Dariush Nikkhah give their tips on how to approach the second part of the MRCS exam.

The MRCS Part B is a daunting prospect, but hopefully these tips will help smooth the experience. Even before work begins it is essential to be familiar with the aims of the revision; examining the marking system and guidance material on the respective Royal College websites is key. Similarly it is important to remember that passes must be obtained in all topics and assessment domains, so concentrating on weaker areas is more important than polishing well known material.

A good anatomy atlas is an excellent aid for the prosection stations. I also spent a day with a fellow candidate in my local medical school's dissection room; this was great for viva practice. Clarifying and practicing a 'routine' for each type of physical examination station can be done with a colleague. Making this process automatic aids concentration on formulating a diagnosis.

On the day make sure you remember to use the alcohol gel at each patient station, and look around the cubicle to see if there is any special equipment you need to ask to use e.g. doppler probe. In the physical examination stations it is sometimes difficult to know how much history taking is required; I chose to ask a few pointed questions before proceeding. Time management is critical especially in the unmanned stations where it is easy to run out of time. Finally I would certainly advise dressing smartly but a jacket is not required as it cannot be taken into the exam room.

Andrew G Titchener MA MRCS AHEA

Any OSCE based examination can be a daunting prospect, the MRCS part B is no exception. The key to success is in knowing what the examiners are looking for and getting plenty of practice. The core curriculum is available at the Royal College website.

A helpful tip is to find other colleagues who are undertaking the exam at the same time, a small group of three of you can then practise set clinical examination techniques and related questions for all the stations with real patients or amongst each other. Amongst our little group we found it useful to give each other little tutorials on specific topics, reducing personal revision time, and gauging our understanding of the subjects.

If you start revising early enough, organise time with consultants in pre-arranged clinics, asking them to critique your clinical examination, diagnosis and management plans. Liaise with your local anatomy department and spend time looking at prosections with the aid of any good anatomy atlas.

During the exam itself, dress smartly but be ready to roll up your sleeves and get rid of your tie. Always use the alcohol gel at every station and look closely at any available equipment as it can help in your examination, for example; a tape measure lying near the patient, suggests measuring leg length in a hip examination. Always greet the mock patients and gain consent, it might sound obvious but it is easy to forget in an exam situation.

Nomaan Sheikh MRCS

HOW TO PREPARE FOR MRCS PART B: SOME HELPFUL TIPS

A Titchener, P Kodumuri, N Sheikh, D Nikkhah

My part B preparation involved 3 key areas.

1. Background (self directed) reading: My reading was focused on clinical examinations, differential diagnoses of common clinical cases and brushing up on Anatomy (definitely mandatory!). Aclands videos helped me immensely in the exam when we had to identify the structures in prosections with markers.

2. Preparation in a small group: I was fortunate to be in a group of three where we examined repeatedly which helped me finish my clinical examination stations immensely. Your presentation skills improve remarkably when you practice and present repeatedly.

3. Attending a course: Although debatable, I attended a course in Leeds which I would say helped me brush up my basic examination skills thoroughly. One of the easy ways to fail an exam is making simple mistakes.

Furthermore, use all the time you have in preparation and revisions rather than reading up new things a week before the exam.

As one of the examiners summed it up on the day of my exam - "It is a very easy exam to pass, but also a very easy exam to fail.

Preetham Kodumuri MBBS MRCS

As the MRCS part B is an OSCE I personally found it easier to practise with colleagues. We devised a weekly revision group that covered anatomy, surgical pathology, surgical skills, patient safety, surgical science and critical care. This helped me gauge my level of knowledge and ascertain what I needed to improve upon in the months prior to the exam.

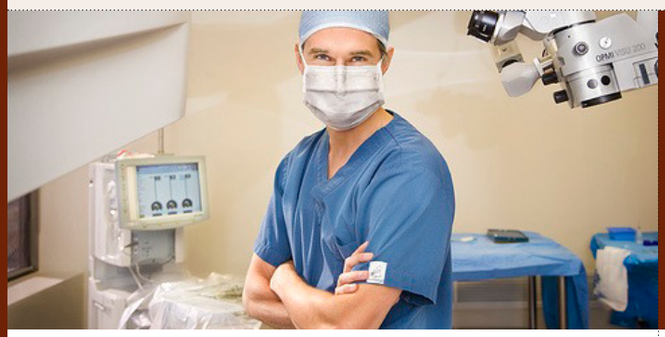
To pass the exam you must have an understanding of the basic principles of surgery. There is no need to read obscure and complicated texts without knowing the fundamentals. Anatomy is a key component for any surgeon and I found it hard to revise from the textbook. Interactive DVDs such as Acland's series on Human Anatomy were extremely simple and easy to watch. In terms of examination and communication skills it is still best to attend outpatient clinics where your consultants can give you important feedback on your style.

The information you learnt in the written MRCS part A will prove useful in the OSCE. From your textbooks you should provide yourself with a standardized framework for clinical examination and questions. I found Basic Science for the MRCS by Andrew Raftery an informative, succinct and easy to read text. The broad reference guide consisting of two PASTEST revision volumes for MRCS written by Catherine Parchment Smith also helped consolidate my knowledge base.

Dariush Nikkhah BM MRCS

ACADEMIC CAREERS IN SURGICAL PRACTICE: THE VIEWS OF AN ACADEMIC CLINICAL FELLOW & NIHR CLINICAL LECTURER

J Smith, D Humes



Academic careers in surgical practice: The views of an Academic Clinical Fellow and NIHR Clinical Lecturer. Current Training Issues.

Introduction

This review offers a practical guide to how an interested surgical trainee may pursue an academic career. Traditionally there have been two types of surgical trainee who have entered research; (i) those wishing to develop their Curriculum Vitae (CV), and (ii) those with an interest in pursuing a long term academic career.

There are common pathways for both groups once they enter a period of research. This article will focus solely on the trainee component of developing an academic career up to the Clinical Lecturer stage.

Routes into research

There are currently two main routes into an academic career in surgery (see Fig 1).

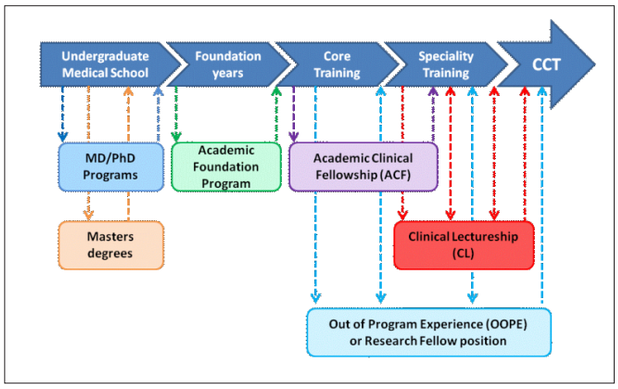


Figure 1: Overview of Current Academic Programs (Based on diagrams from 'Rough Guide' The UK Foundation Programme Office 6 and NIHR Research Careers Pathway

(1) Progressing through an Academic Foundation program to an Academic Clinical Fellowship (ACF) and then to an Academic Clinical Lecturer (CL) post. This pathway was designed to structure Academic training in light of the Walport report[1] and these posted are managed by the National Institute for Health research (NIHR).

(2) Obtaining an independent Research Fellow post and taking an Out Of Programme Experience (OOPE) during and/or between core (CT) and specialty training (ST).

What are the NIHR funded Integrated Academic Training posts[1]?

The NIHR posts allow entry into research at an early stage of training. They are defined research rotations as part of the Foundation program and beyond[2] [3] [4].

What is an ACF? [2]

Academic Clinical Fellowships (ACFs) are combined clinical and academic programs for junior trainees during their early years of speciality training (CT1 and above). ACFs spend 25% of their time in academia and 75% in clinical training. In reality, this equates to 3 months per year or roughly 1 to 2 days per week in academic work. The ACF lasts for 3 years, with the main objective being to acquire academic skills and/or preliminary data to support a successful funding application to a research body such as the Medical Research Council or Wellcome Trust.

In order to support the trainee, the ACF posts advertised as speciality specific are run-through posts with a national training number. If the trainee fails to obtain research funding, or a place on a higher degree program, after 3 years, they will return to normal clinical training if all clinical competencies have been achieved. However, if the ACF succeeds in obtaining a PhD or MD, there is a further opportunity to pursue the integrated pathway by applying in open competition for a Clinical Lectureship.

What is a CL? [2]

Clinical Lectureships (CLs) are 4 years long and involve spending 50% of time in specialist clinical training and 50% in research or medical education training. They are aimed at trainees who are advanced in their specialty training, have already obtained a higher degree and who show an excellent potential for a career in academic medicine. The CL is designed to allow trainees time to develop their research skills and apply for funding to support further post-doctoral or educational training.

ACADEMIC CAREERS IN SURGICAL PRACTICE: THE VIEWS OF AN ACADEMIC CLINICAL FELLOW & NIHR CLINICAL LECTURER

J Smith, D Humes

What are the alternatives to the Integrated Academic Training Program?

The more 'traditional' route of entry to research usually involves an interested trainee approaching an Academic or researcher in a field of interest. This is normally between core and specialty training or after a few years of specialty training. The trainee develops a project and, either acquires a research fellow job, or applies for an Out of Programme Experience (OOPE) and/or funding to undertake a higher degree.

It is still possible to undertake research in this way and to apply for and be successful with many research funding bodies. However, obtaining funding is a highly competitive process, whichever path you take.

Academic placements are not numerous and are competitive. To apply for an academic position one needs to develop an exceptional CV. However, before embarking on an academic career it is important to be aware of what it entails.

What are the benefits of pursuing an academic career? See Figure 2

The excitement of developing a research question, performing the study and then obtained the results is extremely rewarding. You develop a set of research specific and generic transferable skills. There is a period of time to mature and to learn to work independently. The opportunity to contribute to the wider pool of scientific knowledge and advance medical practice is achievable and your work may lead to changes in practice or the eventual development of new methods of diagnosis or treatment. It may also have the fringe benefit by helping to establish yourself in a chosen field. This may help future job applications. However, this will not happen for everyone.

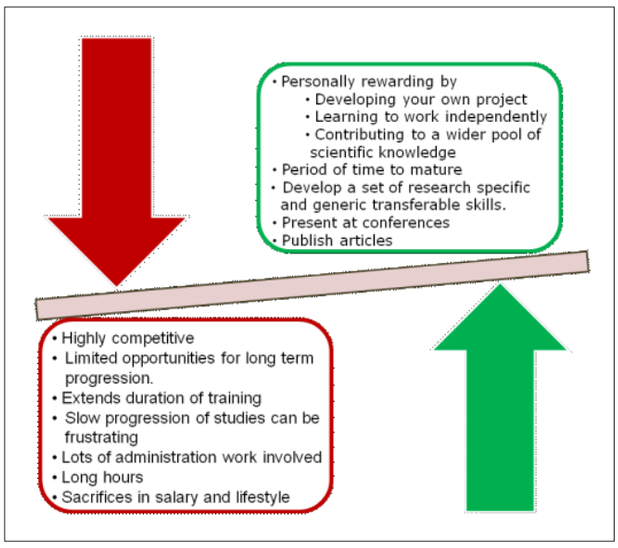


Figure 2: Advantages and Disadvantages of Research

What are the problems in pursuing an Academic Career?

It is competitive, with limited opportunities for long term progression in research. Your clinical training may be extended. It can be frustrating setting up studies, moving into an environment when things do not happen quickly and dealing with the day-to-day administration of projects. Do not underestimate the amount of work involved in projects, ethics applications or higher degrees. It involves working long hours with sacrifices in salary and lifestyle. Only you can decide if the benefits outweigh the disadvantages.

Clinical training issues? [5]

As an academic you need to meet the same standards as a clinical trainee. The same number of work-based assessments have to be completed in only 75% or less of the time. The organisational skills learnt in research will help to achieve this. For more advanced trainees, surgical and on call experience can be difficult to achieve with the result that the duration of training may be lengthened.

So, if research is for you, then the next stage is to think about the best time to integrate research into your clinical career. It is important to be aware that guidance on when and what goals you need to have achieved from a period of research for your future career, are constantly updating. If you are interested in research as a career or purely to improve your future job prospects, it is important you get up to date advice. You should discuss your plans with the JCST (Joint Committee on Surgical Training, http://www.jcst.org/), the Royal College of Surgeons and your Clinical Programme director early on. They can give advice on the best time to apply during your career, especially for Out Of Programme Experience or Clinical Lectureships. Even if they advise waiting until you have increased clinical experience, this time can be usefully spent developing research protocols, ethics applications and improving your CV, to make you more competitive when you do apply.

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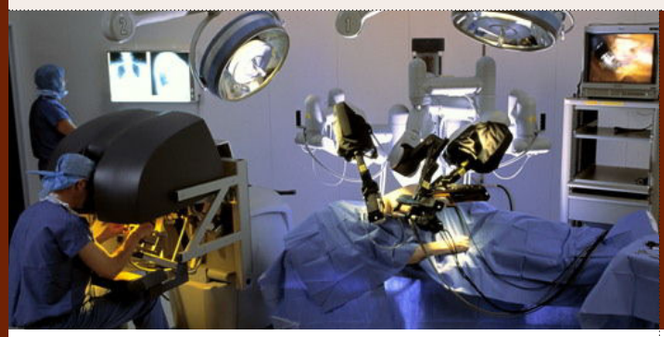

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Developing your CV to apply for a post

The key to developing a good CV is first to write a CV, and then have someone senior look at it. It is impossible to identify areas of weakness until you have produced a CV. Senior clinicians and academics can help you structure your CV, Identify weak areas and suggest how to improve them. All medical students should be encouraged to do this as early as possible to help in their professional development. Time invested in improving your CV will be well spent. Once you have a CV you can outline a plan of what goals you would like to achieve, and when you aim to achieve them and how you are going to do it. There are many opportunities to improve a CV (see figure 3)

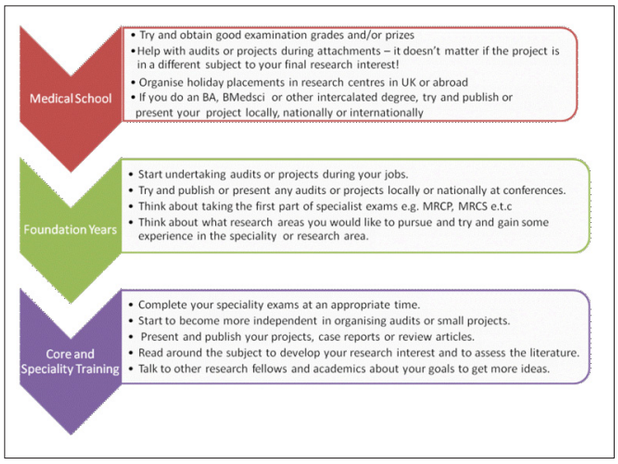

Figure 3: There are many ways to develop your CV at any stage

Once an academic position has been acquired, the next step is often to apply for funding. Some research positions come with funding, which has already been applied for by the project supervisor or is attached to the research project. However, this is the exception rather than the rule. Applying for funding can be a difficult process and success is not guaranteed. You need to focus on the areas on which funding bodies will be focused

(1) the Project

(2) the Research Institution or group

(3) You

The Project

How do you develop a Research Project?

Developing an appropriate research question is the key to good research. Identify an area of research, perform a thorough search of the literature and develop a question. Often the initial question may come from work already undertaken in the research group. Make a critical appraisal of the work that has been performed and ask:

How can I do this better? or How can I add to the understanding of this problem?

A good deal of time needs to be spent developing a research question and refining it. Do not underestimate the time needed to plan and read around the project. Developing the project should be achieved with help from supervisors and collaborators.

How to choose a research institution?

Choosing the right institution is very important. Ensure the institution has a good track record in the area of the proposed research and has the research environment that will support you as a novice researcher. Ideally the group you want to work for should have all the facilities and people to train you in the techniques you will be using. Ensure that the research infrastructure has taught components that lead to higher degrees which is an important factor in any subsequent funding application.

Choosing a Supervisor

This can be a difficult decision. Often a supervisor will be a leading Academic who has developed the project. It is important that a supervisor is approachable and amenable to work with. You are more likely to be successful if a supervisor has previously been successful.

Key questions to ask are:

(a) What have they published and where?
(b) How many students have they successfully supervised?
(c) What success have they had in winning external funding?
(d) Are they experts in the area of the proposed research?
(e) Do they have their own laboratory with local expertise in the proposed techniques?

ACADEMIC CAREERS IN SURGICAL PRACTICE: THE VIEWS OF AN ACADEMIC CLINICAL FELLOW & NIHR CLINICAL LECTURER

J Smith, D Humes

These are by no means faultless indicators but they will give you an idea as to how successful and how well supported you will be. You can usually research most of this information on-line.

How do you improve yourself?

To reach the stage of developing a research project you must have shown a willingness to participate in research. It also helps to have peer reviewed papers, and made presentations at institution and national meetings.

How to go about applying for funding?

This is one of the most daunting tasks. You need to leave plenty of time for writing the application that may involve several months of work, with many revisions. Do not underestimate the amount of time and work required to improve the application. See Figure 4.

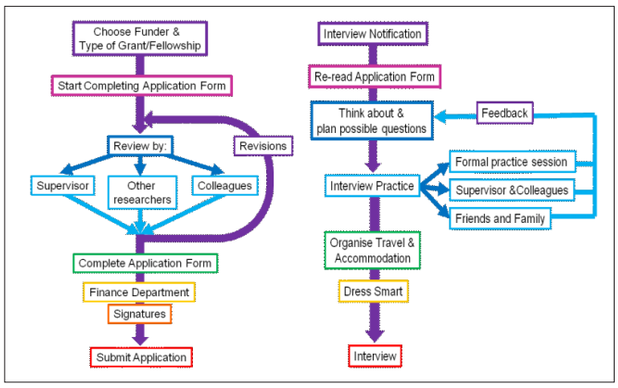

Figure 4: Time line of Funding application and interview

First, choose a funder. Whether the funder is Charity based or Research Councils will depend on the area of research. For any application, the research question should be clear, and should include the methods behind it. Ideally there should be some preliminary data included in the application. If you are an NIHR trainee, you could utilize some research time by collecting or analyzing this data and learning the techniques that will be needed to undertake the project.

Personal details, the institution's profile and the project need to be clear and precise. Leave time for supervisors and research collaborators to read and edit the application. An academic from outside the research group may be asked to peer review the application to ensure people from outside the research area can understand the proposed work. You are the driving force for the application, you need a plan and a strict time line to get all aspects of the application completed and reviewed. Time needs to be allocated for the application to be costed by the finance department, for referees to write an accompanying piece and for finding the other signatures, such as the Head of Department. Key people are often busy and may be away at crucial points. Advise them about the application in advance and make arrangements to collect important information or signatures. Be aware of what needs submitting.

Most applications are on line, but some funders still have a paper version. Make sure all the criteria for the application are fulfilled e.g. font, spacing, number of copies, DVD or CD copies. Importantly allocate time to send off the application.

This may seem very daunting and it is. It is important to talk to others who have been through a similar process and have or haven't been successful. They can help with insight into what problems and pitfalls can be avoided and may help by letting you view their applications.

The application is your gateway. If successful, the next level is the interview. If unsuccessful, don't be disheartened. They can provide insights into the project and give pointers as to how the application may be improved. The secret to success is often perseverance and responding to feedback.

The interview

The key to interviews is practice. Ask colleagues and supervisors about questions that may be asked and what the style of the interview is likely to be. Gather a list of potential questions that may be asked. Familiarize yourself with the application as it will be several months since it was sent it off. Try to predict areas that may need explaining. Write down or practise your answers to the questions – but try not to make them sound rehearsed. Try to organize an interview panel with supervisors and other academics. Practising with colleagues, friends and family can also help – even if they do not fully understand what the project is about.

On the interview day dress as you would do for any formal interview. Allocate plenty of time to reach the venue - perhaps stay in a hotel if the interview is in the morning. Check and/or book transport in advance. In the interview, do not be worried about the size of the room or the number of interviewers. Some panels may be only 2-4 interviewers but some can be 20 or more. If the panel is large, it is likely that only a few will be asking questions. The panel may consist of people who have an expertise in the area of the proposed research or proposed techniques. Be prepared to discuss the background, design, set up and analytical methods as well as yourself and your ambitions. Try not to be discouraged by stony expressions or difficult questions. The panel need to see enthusiasm for and understanding of the project, and that the objectives will be delivered. They are potentially going to give you tens of thousands of pounds – they need to be sure you, your project and your institution are a good investment.

ACADEMIC CAREERS IN SURGICAL PRACTICE: THE VIEWS OF AN ACADEMIC CLINICAL FELLOW & NIHR CLINICAL LECTURER

J Smith, D Humes

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Whatever happens try not to dwell on the negative aspects afterwards. To get an interview is a fantastic achievement. Note down the questions that you remember to help you in your next interview. Do this, even if you are successful because someone else may need your help and you may apply for other funding in the future. If you are successful, congratulations! If not persevere.

What happens if you do not secure funding?

There is a possibility that you may not get funding. Your academic time should still be useful, with you having gained many generic skills and gathered some data that can be publish and presented.

This article is meant as a practical guide to research written by two trainees one of whom is currently undertaking her PhD and another who has completed their PhD. It is by no means comprehensive but acts as a starting place from which you take a better understanding of what a career as an academic trainee may entail.

What other information is available about academic training and funding?

Below is a list a useful websites
http://www.academicmedicine.ac.uk/
http://www.clinicalacademicjobs.org
http://www.nihrtcc.nhs.uk/
http://www.wellcome.ac.uk/Funding/index.htm
http://www.mrc.ac.uk/Fundingopportunities/index.htm
http://www.rdfunding.org.uk/

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Authors

Miss. Jan Smith Wellcome Trust Research Fellow
Mr. David J Humes 1 Lecturer in Surgery
1 Nottingham Digestive Disease Centre and
Biomedical Research Unit, QMC Campus,
E Floor, West Block, Nottingham University Hospital NHS Trust,
Derby Road, Nottingham, NG7 2UH, UK

Corresponding author

Mr. David Humes
Lecturer in Surgery, Nottingham Digestive Disease Centre
and Biomedical Research Unit, Department of Surgery,
QMC Campus, E Floor, West Block,
Nottingham University Hospital NHS Trust,
Derby Road Nottingham NG7 2UH, UK
Tel: 0115 823 1143, Fax: 0115823 1160
Email: david.humes@nottingham.ac.uk
jan.smith@nottingham.ac.uk

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