123 Doc EDUCATION

FOUNDATION YEARS JOURNAL

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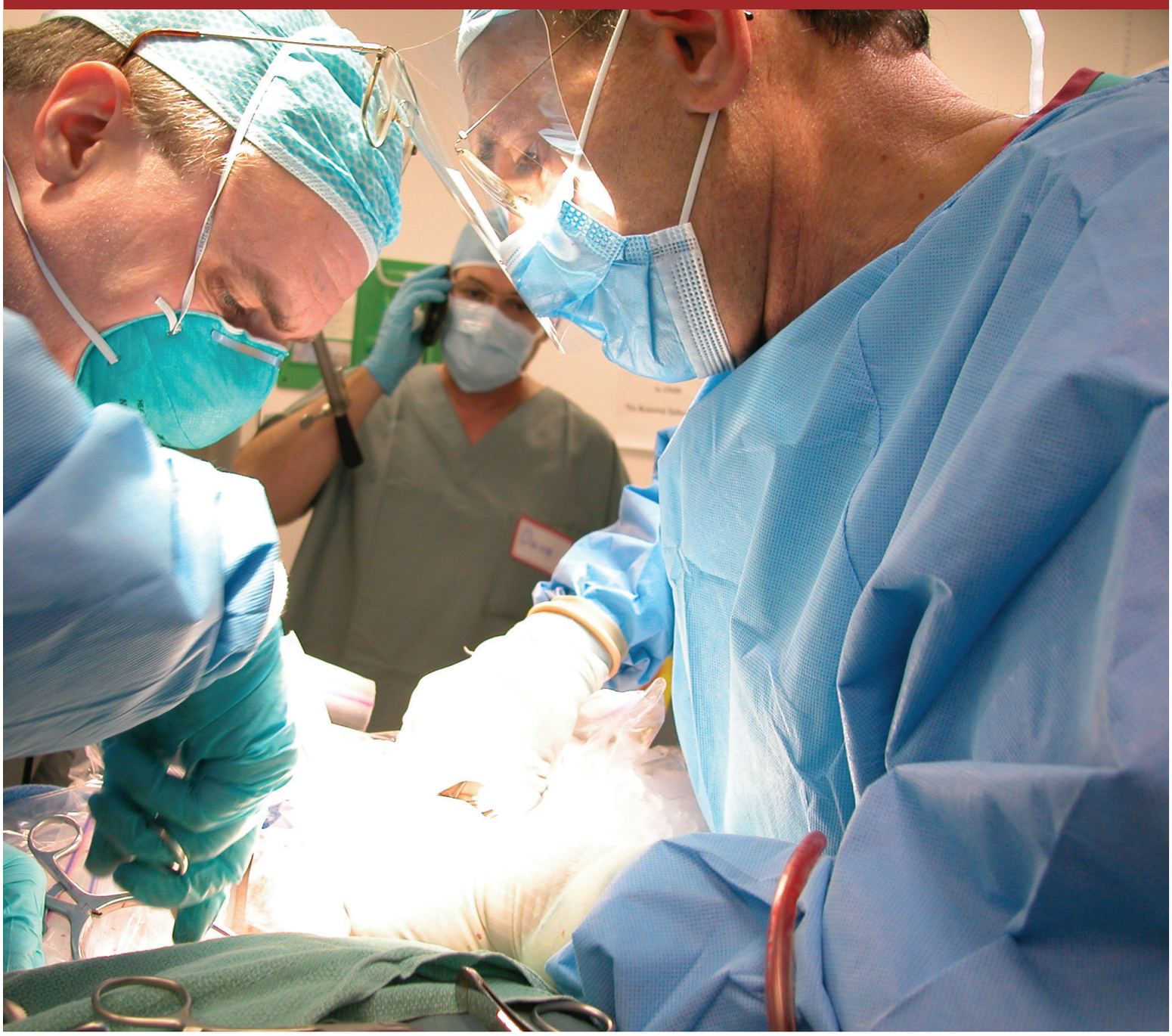

Contents

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Volume 4, Issue 1

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Volume 4, Issue 1: Surgery

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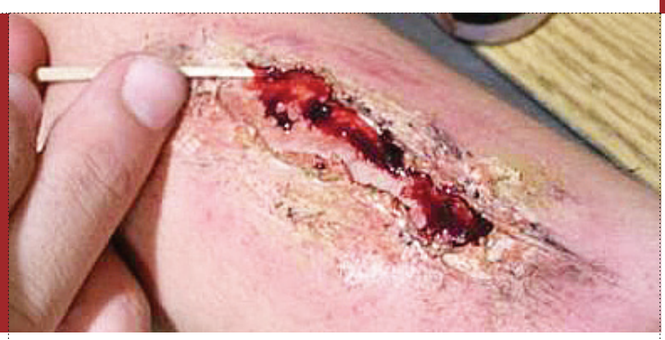
A HOUSE OFFICER'S GUIDE TO THE RECONSTRUCTIVE LADDER OF WOUND HEALING

Christopher Edward Hill and Thomas Macleod

Wound healing is a physiological process that aims to restore the anatomical and functional integrity of an acquired tissue defect.
Patient Management.



Introduction

Wound healing is a physiological process that aims to restore the anatomical and functional integrity of an acquired tissue defect.

The majority of wounds will heal on their own if given sufficient time, however, there are several surgical techniques that may be used to provide both quicker and more optimal wound closure.

The reconstructive ladder is an algorithm that helps select the most appropriate method of achieving wound closure. The ladder arranges the different methods of wound closure in hierarchical form from the simplest at the bottom to more complex techniques at the top.

When considering the closure of a particular wound, the lowest rung on the ladder (i.e. healing by secondary intention) should always be considered first and only if this is felt to be inappropriate is the next rung of the ladder (i.e. direct closure) considered, and so on.

This article provides an overview of the various methods of wound closure, their relative advantages and disadvantages and how to choose between them.

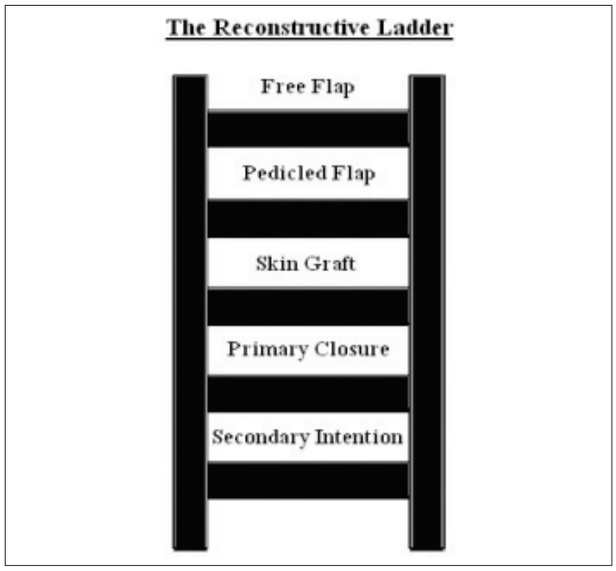


Figure 1: The reconstructive ladder.

Healing by secondary intention

Healing by secondary intention is the process of allowing an unopposed wound to pass through the different phases of wound healing with wound closure being achieved by a combination of wound contraction, keratinocyte migration and epithelialisation.

If the edges of a wound cannot be physically opposed (without undue tension) and there are no important structures in the base of the wound that may suffer as a result of exposure to the environment then healing by secondary intention should be considered.

Advantage:

- Avoids inherent risks associated with a surgical procedure.

Disadvantages:

- Generally takes longer to heal compared to other methods.
- Leaves the underlying tissues in the base of the wound exposed to the environment with potential for desiccation and infection.
- Final healed result may be inferior both from an aesthetic and functional point of view.

Examples:

- Pressure sores and leg ulcers.

Healing by primary intention

Healing by primary intention is the process of opposing the edges of a wound (usually by mechanical means) and again allowing the wound to pass through the phases of wound healing.

If the edges of the wound can be physically opposed without undue tension and the base of the wound is "clean" (i.e. free of dead, devitalised and infected tissue) then healing by primary intention should be considered. The wound is usually held closed by some form of mechanical means (e.g. sutures, steri-strips, skin staples or glue).

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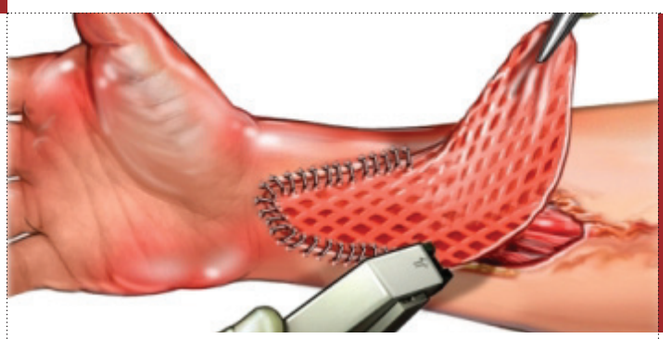
Christopher Edward Hill and Thomas Macleod



Skin grafts consist of epidermis together with a variable amount of underlying dermis.
Patient Management.

Advantages:
- Shorter time to achieve wound closure.
- Usually provides a better aesthetic and functional result compared to healing by secondary intention.

Disadvantage:
- Usually requires surgical intervention.

Example:
- Elective surgical incision.

Healing by tertiary intention, (delayed primary closure)

Healing by tertiary intention describes the process of debriding a wound of dead, devitalised and infected tissue and then leaving the wound for a period of time (typically 48 hours) before considering subsequent closure. The time interval allows any subclinical or further ischaemic tissue to declare itself and if necessary, for the wound to undergo further debridement.

Examples:
- Animal or human bite.

If a wound cannot be left to heal on its own and cannot be closed directly then consideration should be given to grafts and flaps, i.e. tissue that is transferred from another area of the body (a donor site) to cover the wound in question (recipient site).

Skin grafts

If the edges of the wound cannot be physically opposed and the wound bed itself is sufficiently vascular, then a skin graft should be considered as a suitable method of wound closure.

A skin graft is a piece of skin area that is harvested (removed) from one area of the body and used to cover a wound at another site. Once the skin is completely detached from the body it has no intrinsic blood supply and must then acquire a new blood supply from capillaries in the base of the recipient wound. In some circumstances a non-vascular bed may need to be converted to a suitably vascular bed to allow application of a skin graft. Using vacuum suction dressings, which is a technique that applies negative pressure to a wound in order to improve the vascularity may help achieve this[1].

Skin grafts consist of epidermis together with a variable amount of underlying dermis. In general, the greater the percentage of donor site dermis that is taken the more the skin graft will maintain the properties of normal skin, however, as the graft becomes thicker its metabolic demands increase and it is less likely to survive on all but extremely vascular wound beds. Full thickness skin grafts consist of epidermis and all of the underlying dermis, whereas split thickness skin grafts contain some but not all the underlying dermis.

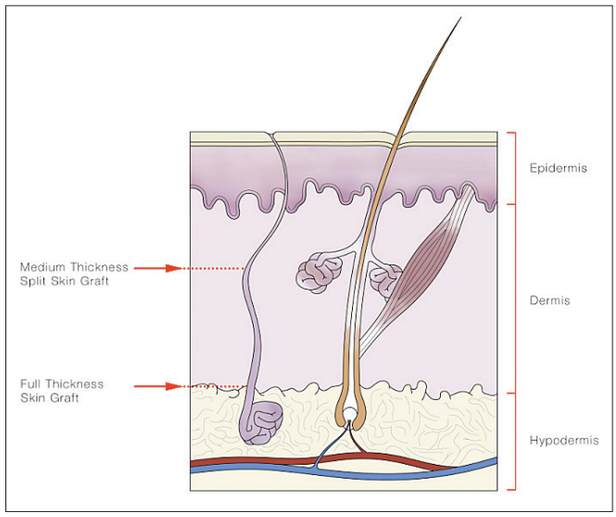


Figure 2: Cross-section of skin demonstrating levels of full thickness and split thickness skin grafts.

The donor site of a split thickness skin graft behaves like a "graze" and is usually able to re-epithelialise within 2 weeks from pluripotential stem cells (associated with hair follicles and sweat glands) in the residual dermis. A full thickness skin graft leaves no residual dermis, just subcutaneous fat and therefore must heal by secondary intention, but is usually closed directly. Donor sites for full thickness skin grafts are usually placed in discrete areas with skin laxity to allow closure and a hidden scar (e.g. post auricular sulcus or groin crease). Split thickness skin grafts are usually harvested from broad, flat areas with thick dermis to allow ease of harvest and to ensure that some dermis can be left to allow re-epithelialisation (e.g. thigh and buttock).

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Split thickness:

Advantages:
- Broader range of application, tolerating less hospitable graft sites.
- Can be left as a continuous sheet or passed through a meshing machine which cuts holes into it producing a net like appearance and allowing the graft to cover a larger area.

Disadvantages:
- Contract during healing, and will not grow with an individual.
- Provide a less satisfactory cosmetic appearance, as they often lack the pigmentation and hair growth of the surrounding tissue, and graft meshing can lead to a "pebbled" appearance upon healing.

Full thickness:

Advantages:
- Retain the characteristics of normal skin, including colour and texture.
- Suffer less secondary contraction when healing compared with split thickness grafts, which is particularly important when covering areas on the face, hands or over joints.

Disadvantages:
- Leaves no residual dermis at donor site, which requires subsequent closure.

Examples:
- Full thickness skin grafts are typically used to replace missing skin following excision of skin malignancies and to replace scarred tissue which is causing functional impairment.
- Split thickness skin grafts are typically used to cover large wounds, such as debrided burns.

Flaps

In its simplest form, a flap is a piece of tissue that remains attached to the body by its blood supply. As a flap has its own intrinsic blood supply, it is not dependant on the recipient wound bed for its survival. Flaps may be classified on the basis of the relationship between the donor area and recipient wound. The flap may be raised next to the wound (local flap), in the same part of the body (regional flap) or in a different part of the body (distant flap). Flaps of various composition can be raised (e.g. skin only, skin and facia, muscle only etc.), but all have an attachment to the body that provides the necessary blood supply.

If the edges of a wound cannot be physically opposed and the wound bed is unsuitable for a skin graft then either the wound bed must be optimised (e.g. application of VAC dressing) to allow application of a skin graft, or a flap should be considered. Consideration should be given for local flaps, then regional flaps and then distant flaps that will achieve wound closure. Flaps are also chosen instead of grafts if they will offer a better aesthetic or functional result.

Advantage:
- Provide robust soft tissue coverage of a non-vascular wound.

Disadvantages:
- Requires a surgical procedure with associated risks.
- Donor site morbidity.

Example:
- A strip of skin and fascia can be raised on the lower leg and used to cover an avascular defect – this is known as a fasciocutaneous flap. By lifting up a fasciocutaneous flap to cover a defect, this exposes the underlying muscle or periosteum at the donor site and this usually requires a skin graft to provide cover.

Free Flap

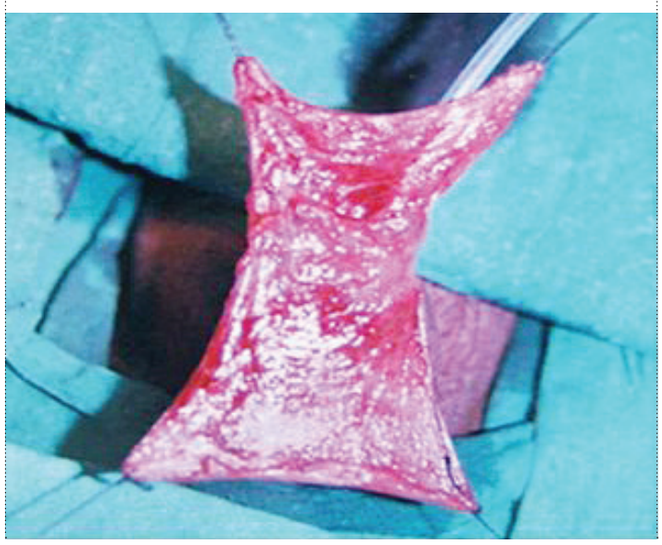
If the flap has an identifiable artery and vein, its blood supply can be temporarily interrupted as it is detached from the body and then reattached at the recipient wound by microsurgical anastomosis to blood vessels in the area. This is the basis of a "free" flap.

If a wound cannot be left, closed or grafted and there is no suitable local, regional or distant flaps that are suitable for wound coverage then a free flap should be considered.

Advantage:
- Provide robust soft tissue coverage of a wound where no local, regional or distant flaps are suitable.

Disadvantages:
- Requires an extensive surgical procedure with associated risks.
- Donor site morbidity.

Example:
- Free latissimus dorsi flap that is detached from its blood supply in the axilla and then reattached to blood vessels in the leg to allow muscle coverage of large tibial defects.



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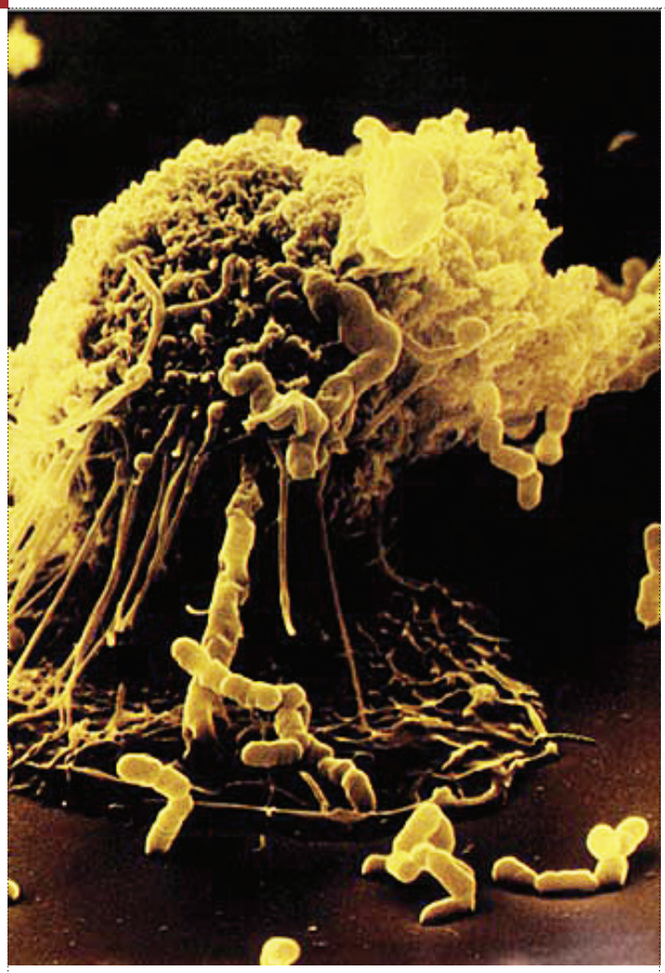

Acknowledgements

The Authors would like to thank Leo Arnold (Graphics Designer) for his assistance in the production of Figure 2.

Recommended reading

Black JM (1996) Surgical Options in Wound Healing. *Crit Care Nurs Clin North* Am. June, 8(2):169–182.

Colwell AS, Orgill DP (2006) Five Steps for Managing Complex Wounds: how employing the reconstructive ladder can prevent complications. *Contemporary Surgery,* 4th April, vol. 62.

Levin LS (1993) The reconstructive ladder. An orthoplastic approach. *Orthop Clin North Am.* July, 24(3):393–409.

McGregor AD, McGregor IA (2000) *Fundamental Techniques of Plastic Surgery: And Their Surgical Applications.* 10th edn. Churchill Livingstone.

Radovan C (1984) Tissue Expansion in Soft-Tissue Reconstruction. *Plast Reconstr Surg,* October, 74(4):482–492.

Taylor B, Bayat A (2002) Basic Plastic Surgery Techniques and Principles: Wound Healing. *Student BMJ,* 11:271–273.

Turner AJ, Parkhouse N (2006) Revisiting the reconstructive ladder. *Plast Reconstr Surg,* July, 118(1):267–268.

References

[1.] Argenta LC, Morykwas MJ, Marks MW, Defranzo AJ, Molnar JA, David LR (2006) Vacuum-assisted Closure: *State of clinic art. Plast Reconstr Surg,* June, 117(7 suppl):127S–142S.

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POST-OPERATIVE PROBLEMS

Jon Maskill

Surgery and anaesthesia together put significant stresses on the patient, the response to which is sometimes unpredictable. Good Clinical Care.

Surgery and anaesthesia together put significant stresses on the patient, the response to which is sometimes unpredictable. This article discusses some of the more common problems that may be encountered by junior doctors looking after patients who have very recently undergone surgery.

Early warning scoring systems are often employed to flag up patients with post-operative problems. If a patient is "scoring" then it is likely that they are manifesting the early features of reversible organ failure. Intervention at this stage is more effective and cheaper than waiting for the patient to deteriorate further and relying on organ support on the intensive care unit.

Airway

Airway obstruction can result from foreign bodies (blood clots, gauze swabs or dislodged false teeth), laryngospasm (spasm of the vocal cords) or laryngeal oedema. The result is stridor – an inspiratory noise often accompanied by see-saw movements of the chest and abdomen. If the obstruction is severe then there is little or no noise. Fully awake patients are often very distressed.

Laryngospasm rarely occurs more than 1 hour post-operatively unless in response to a foreign body which has started to irritate the vocal cords. The onset is sudden.

Laryngeal oedema can occur after ENT surgery to the upper airway or after a traumatic intubation. Unless there is oedema while in the recovery room the onset tends to be gradual.

Management

- Try to calm the patient allowing them to sit in whatever position they find most comfortable.
- Summon immediate help, preferably from an anaesthetist.
- Use suction to remove any visible material under direct vision only.
- Administer oxygen via a facemask.

Breathing

Breathing difficulties can occur at anytime following surgery (see Table 1). Establishing a diagnosis should occur concurrently with supporting the patient's respiratory system. Residual neuromuscular blockade will usually manifest itself in the recovery room and will be dealt with by recovery staff and an anaesthetist.

Immediate <1 hr	Early 1 to 24 hrs	Late 24 hrs +
Pneumothorax	Atelectasis	Pneumonia
Pulmonary oedema	Pulmonary oedema	Pulmonary oedema
Lobar collapse		Pulmonary embolism
Aspiration		ARDS
Residual neuromuscular blockade		

Table 1: Causes of breathing difficulties according to time of likely presentation.

History and examination

Try to determine what the patient is usually like. Do they have chronic heart or kidney disease that may predispose them to pulmonary oedema? Do they have long-standing COPD? Did the anaesthetist insert a central line during the operation and possibly create a pneumothorax? They may have a productive cough and a high temperature suggestive of pneumonia. Perhaps they underwent lower limb surgery a week ago and now have the pleuritic chest pain typical of a pulmonary embolism. Has the patient been unable to take deep breaths due to abdominal pain? They may have atelectasis. Are both sides of the chest the same on examination?

Investigations

These may include:

- Chest X-ray
- 12 lead ECG
- Arterial Blood Gases
- Echocardiogram
- Blood and sputum cultures.

Gather as many clues in the history, examination and investigations as you can to form a working diagnosis.

POST-OPERATIVE PROBLEMS

Jon Maskill

Treatment

• Oxygen should be administered to all patients with breathing difficulties. Monitor their response with a pulse oximeter. In patients with severe COPD a target saturation of 92% is often adequate. In other patients you should aim to achieve 95%. Ensure that the oxygen flow rate is appropriate for the type of mask you are using.

• Infective causes of breathing difficulties should be treated with intravenous antibiotics once cultures have been sent as guided by the microbiologist.

• Any post-operative pneumothorax should be drained if breathing difficulties ensue.

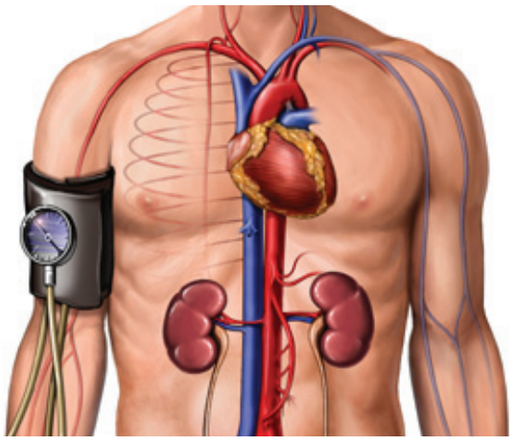
• Patients with abdominal pain avoid coughing and taking deep breaths. This results in atelectasis and occasionally lobar collapse. Adequate analgesia, physiotherapy and mobilisation can help to re-expand collapsed segments and improve oxygenation.

• Pulmonary oedema can be the result of fluid overload, left ventricular failure or ARDS (lung inflammation causing capillary leakage). Fluid overload is unusual in patients without either cardiac or renal disease. Take care to find out exactly how much fluid has been given and how much has been lost. Left ventricular failure may be due to a peri-operative myocardial infarction. ARDS is more likely if the patient has been critically ill or has received a massive blood transfusion. Intravenous furosemide (20mg to 40mg) may induce a diuresis and improve oxygenation. Cardiology input is often necessary.

• Despite thromboembolic prophylaxis, pulmonary embolism remains a significant cause of morbidity and mortality in post-operative patients. Usually this will occur several days following surgery (particularly pelvic and lower limb operations). A CT pulmonary angiogram should be considered if pulmonary embolism is suspected. Treatment is with either high–dose, low molecular weight heparin (LMWH) or an infusion of unfractionated heparin (UFH) followed by oral warfarin. The use of thrombolytic agents in these patients is controversial.

Circulation

Hypotension is relatively common in post-operative patients. Usually the cause is hypovolaemia but not always. While 250ml of intravenous crystalloid is rarely going to cause problems as a first line response, attending the patient and considering the options will benefit the patient in the medium to long term. The common causes of hypotension are elaborated in Table 2 along with suggested responses.



	Causes	Diagnosis	Treatment
Low left ventricular preload	Visible fluid losses (vomit, faeces, bleeding in drains) Invisible losses (bowel secretions, internal bleeding)	Usually tachycardia, poor capillary return, low volume of concentrated urine. May have full drains, distended abdomen, obvious gut losses	Intravenous fluids and/or blood products Investigate and treat the cause
Poor contractility	Myocardial infarction Pre-existing left ventricular failure	Usually tachycardia, poor capillary return, low volume of concentrated urine. History. ECG. Chest X-ray. Echocardiogram	Further monitoring (CVP, arterial line) Cardiology referral
Brady and Tachyar-rhythmias	Electrolyte imbalance Pre-existing dys-rhythmia	Pulse rate and rhythm. ECG. History	Correct any electro-lyte imbalance. Introduction or reintroduction of antidysrhythmic medication
Low peripheral resistance	Epidural analgesia	Tachycardia and low volume of concentrated urine. Warm peripheries with a brisk capillary return. Pyrexia, high white cell count. Purulence around wound, can-nulae or central line insertion site. Epidural catheter in situ with vasodilata-tion confined to lower body and limbs	Appropriate bacte-riological cultures followed by intrave-nous antibiotics and correction of cause. Provided there are no features of end organ hypo-perfusion, epidural induced mild hypotension does not require treatment. Discuss with anaesthetist

Table 2: Causes, diagnosis and treatment of post-operative hypotension.

The kidney is considered under the heading "circulation" by virtue of its intimate relationship with blood pressure and circulating volume. When the circulating volume is high there is less tubular reabsorption of water, the urine output increases and the circulating volume contracts. The opposite is true when the circulating volume is low. On top of the intrinsic renal mechanisms for controlling circulating volume, extrinsic factors (stress hormones and nephrotoxic drugs) exert their effects by resetting the limits within which the kidney functions. Urine volume is usually in excess of 0.5ml/kg/hr. Below this is oliguria and no output at all is anuria. Patients with cardiac failure or pre-existing renal disease are more likely to develop renal failure post-operatively.

POST-OPERATIVE PROBLEMS

Jon Maskill

In a typical post-laparotomy patient the urine output is generally high for the first few hours by virtue of the large volumes of fluid given in theatre by the anaesthetist. It gradually falls and gets more concentrated over the next 12 hours to between 0.5 and 1ml/kg/hr before slowly increasing to normal.
Good Clinical Care.

In a typical post-laparotomy patient the urine output is generally high for the first few hours by virtue of the large volumes of fluid given in theatre by the anaesthetist. It gradually falls and gets more concentrated over the next 12 hours to between 0.5 and 1ml/kg/hr before slowly increasing to normal. This ebb and flow is at least in part due to stress hormones released as a result of surgery. In patients with an inadequate circulating volume the relatively low output can turn to oliguria and then anuria if circulating volume is not restored. Early warning scoring systems include urine output so that oliguria is flagged before anuria and renal failure ensue.

Several hours of good urine output followed by anuria may be due to urinary catheter blockage. If this is suspected then flush the catheter. Sluggish capillary return, thirst, low JVP and concentrated urine suggest a contracted circulating volume. Hypotension (see Table 2) will result in oliguria whatever the circulating volume. The treatment of oliguria is to attend to the circulation and to withdraw any potentially nephrotoxic drugs (gentamicin, NSAIDS). Loop diuretics, such as furosemide, will only increase urine output if the blood pressure is adequate. However, reducing the circulating volume with a drug that is potentially nephrotoxic can worsen renal function. Hence, use furosemide only if you are certain that the blood pressure is adequate and the circulating volume needs to be reduced. As a rule of thumb, provided the urine output is adequate and diuretics have not been used, the blood pressure is also probably adequate.

Disability

Pain

Post-operative pain is very common. If a patient complains of pain, they probably are in pain. Analgesic requirements vary widely between patients. Reducing pain is best achieved by a combination of drugs each acting on a different part of the pain pathway. Table 3. illustrates some commonly employed analgesic drugs. Do not forget that the wound itself might not be the source of pain. Other causes include a sore throat or sinusitis from the NG tube, backache due to uncomfortable bedding, post-dural puncture headache following spinal anaesthesia, phlebitis due to cannula infection or irritant infusates, angina pectoris, etc.

	Dose and route (adults)	Comments
Paracetamol	1g every 6 hours. PO/IV/PR	For mild pain and as an adjunct to all other analgesics
NSAIDS	Several are available to be given orally with food or rectally. Newer injectable versions are being marketed	Good analgesia provided the patient is absorbing enterally and has no evidence of renal dysfunction. The potential for upper GI bleeding and renal failure limit use. COX-2 inhibitors have been linked with cardiac failure
Tramadol	100mg every 6 hours. PO/IV	Has analgesic properties above its mild morphine-like effect. Can be used in conjunction with paracetamol, NSAIDS and PCA morphine. Can cause nausea and confusion
Morphine	0.1–0.2mg/kg every 3–4 hours IV/Sc 0.2mg/kg every 3–4 hours PO (Oramorph)	Patients have very variable requirements. An ineffective dose in one patient may cause severe respiratory depression in another. Start low and titrate upwards. Maximum IV effect is after 15–20 minutes. Patient Controlled Analgesic (PCA) machines are only of use if the patient is pain-free first – they simply help to keep the analgesic level constant. Depressed respiratory rate and nausea are the main side effects. Histamine release is occasionally troublesome
Epidural	Sited by the anaesthetist. Up to 15ml/hr of local anaesthetic (often with an opiate) is delivered epidurally via a catheter	Extremely effective when working correctly. Asymmetrical or inadequate block height will require anaesthetic input. Persistent motor block after catheter removal or backache with neurological signs requires immediate anaesthetic advice

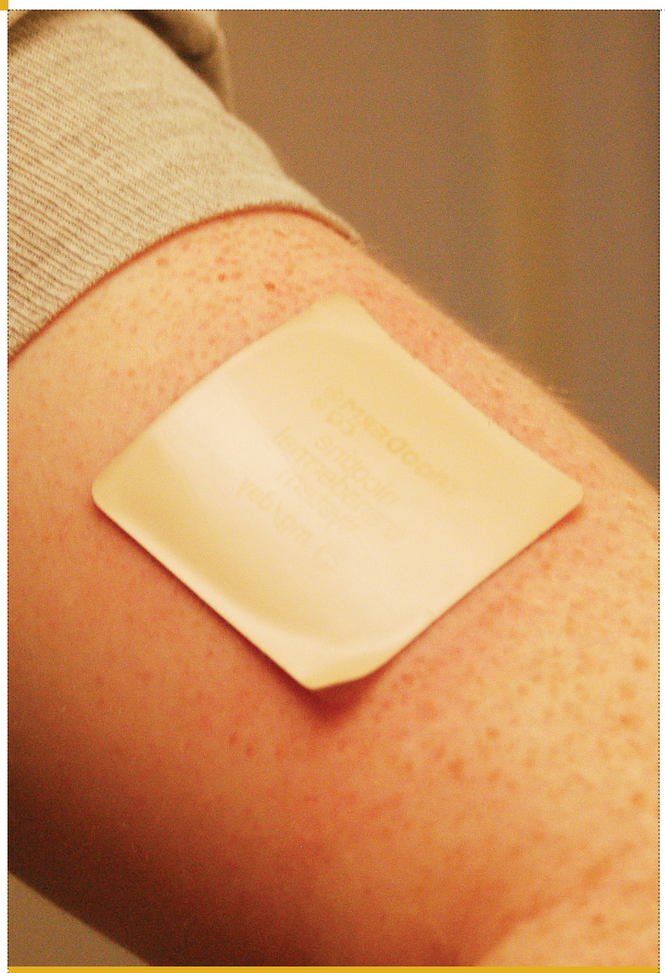
Table 3: Commonly used analgesics.

Delirium

Delirium is an acute, reversible organic mental syndrome with disorders of attention and cognitive function with increased or decreased psychomotor activity. Most common in the elderly and manifest particularly at night, delirium is a major problem post-operatively. While an unfamiliar environment and the effect of opiates account for the majority of cases there are a few causes that need to be excluded before simply prescribing a sedative. Hypoxia, pain, infection and withdrawal syndromes (see Table 4) must be identified and treated. Talking to the patient and providing an unambiguous environment may help in mild cases. Haloperidol 2–5mg orally (1–5mg i.v.) can be used to alleviate anxiety and make the patient easier to manage in more severe cases. Occasionally, up to 10mg midazolam i.v. in increments is given for dangerous motor activity.

POST-OPERATIVE PROBLEMS

Jon Maskill



	Comments
Alcohol	Tremor and agitation can lead to generalised seizures. i.v lorazepam 1–4mg can reduce anxiety and reduces the risk of seizures
Opioids	It is unusual for cessation of post-operative analgesics to result in withdrawal symptoms. In patients on long-term opioids, reintroduce them. Anaesthetic or chronic pain service input may be useful
Benzodiazepines	Careful reintroduction of usual medication or an intravenous substitute
Nicotine	Heavy smokers may benefit from a 21mg nicotine patch daily if other causes of agitation have been discounted. The patches may increase the risk of thrombosis
Antidepressants	Restart as soon as possible. If the enteral route is not available contact the pharmacist to discuss alternative routes
Recreational drugs	Other than the specific therapies for benzodiazepine or opioid withdrawal, benzodiazepines (lorazepam 1–4mg iv) may be of some benefit

Table 4: Specific withdrawal syndromes resulting in delirium.

Further Reading

1. Zakaria R, Handa A (2007) Student BMJ 15:337–382.
2. UKCPA Delirium (2006) **http://www.ics.ac.uk/icmprof/standards.asp?menuid=7**
3. Scottish Intercollegiate Guidelines Network (SIGN) number 77.

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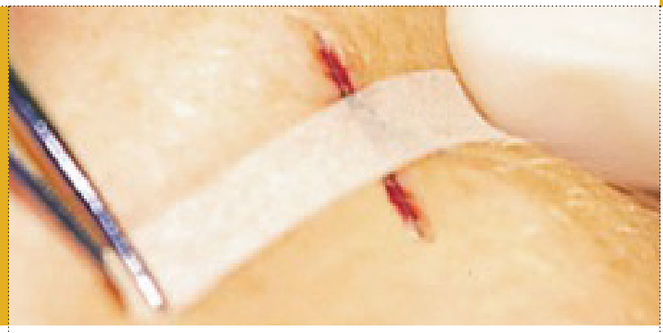
SURGICAL WOUNDS AND SURGICAL SITE INFECTIONS

Sarit Badiani and Sarah Addison

As a Foundation Year doctor it is essential to be familiar with the different types of surgical wounds encountered in a surgical unit and their routine care.
Good Clinical Care.



Introduction

As a Foundation Year doctor it is important to be familiar with the different types of surgical wounds encountered in a surgical unit and their routine care. Understanding the mechanisms for wound healing is essential to surgical practice as well as knowing that the principles of wound care are the same in all kinds of surgery.

Surgeons are responsible for making wounds but due to high turnover of patients in day case surgery and enhanced recovery programmes, post-operative care of wounds are largely managed by junior doctors, ward nurses, community nurses and specialist nurses (e.g. tissue viability nurses for complex wounds).

Types of wound healing

There are two types of wound healing – healing by primary intention or healing by secondary intention[1].

a. Primary intention healing: this is when there is no loss of tissue and the opposing layers of the wound are brought together by means of sutures allowing healing to occur by primary intention.

b. Secondary intention healing: this occurs when there may be loss of tissue thereby opposing layers of the wound cannot be brought together by means of sutures, allowing healing to take place by process of granulation.

The differences between primary and secondary intention healing are summarised in Table 1.

Primary Intention Healing	Secondary Intention Healing
No loss of tissue	Loss of tissue
Shorter healing process	Longer healing process
Smaller scar	Larger or hypertrophic scars
Clean or planned surgical incision	Dirty or traumatic wound

Table 1: Differences between primary and secondary intention healing.

Inspection of wounds

Evaluation of any wound must begin with careful inspection, examination of the patient and investigation into its etiology. Wounds that are especially resistant to healing with conventional measures may be due to a number of causes, including mechanical trauma, chemical injury, vascular compromise, infection, neoplasm, pressure and allergic reaction.

Careful inspection of the wound must be accurate and done at regular intervals. The caregiver must evaluate the wound, the surrounding skin and the overall health status of the patient[2]. When evaluating a wound, specific points should be considered, such as size, shape and healing process. A guide to the examination of a wound is summarised in Table 2.

What to Look for in a Wound	Details
Type of wound	Is it a surgically created wound? (i.e. clean wound or is it a traumatic wound?) (i.e. dirty wound?)
Site	Where is the wound? In limbs, usually described from a point nearest to the closest bony prominence. On the abdomen surgical incisions are commonly midline, transverse, sub-costal, etc.
Size	How big is the wound in centimetres (cm) or inches (in)?
Surrounding skin	Any presence of erythema?
Exudate	Any evidence of exudate formation. Type of discharge (e.g. bloody, serosanginous or haemoserous).
The patient	Patient's general health status. Obesity, diabetes mellitus, malnutrition are all increased risk factors for poor wound healing.
Other	Any sinuses, fistula, tracts or dehisence.

Table 2: How to inspect wound systematically.

SURGICAL WOUNDS AND SURGICAL SITE INFECTIONS

Sarit Badiani and Sarah Addison

Wound dressings

The choice of wound dressing should be aimed at promoting adequate healing, protect the wound and reduce the risk of infection. Wound dressings have certain properties and should be selected depending on the condition and type of wound involved. Some examples of essential properties of conventional wound dressings include[3].

1. Non-toxic, non-allergenic and non-sensitising.

2. Those that allow gaseous exchange – permeability to oxygen, carbon dioxide and water vapour.

3. Non-adherent and easily removable without causing trauma to the wound – adherence to underlying tissues can result in disturbance of granulation tissue leading to delayed healing.

4. Provide a barrier to microorganisms – reduce risk of infections.

5. Maintain a moist environment - promote wound healing. Table 3 summarises the different classification of wound dressings available and their uses[3, 4].

Type/ Classification of Wound Dressing	Properties of Wound Dressing and Best Use
Conventional Dressings	Non-toxic, non-allergenic and non-sensitising Allow gaseous exchange Non-adherent and easily removable without causing trauma to the wound Barrier to micro-organisms – reduce risk of infections
Hydrocolloids (e.g. Granuflex)	Occlusive Dressing Avoid in infection Over-granulation can occur Hydrocolloid absorbs excessive exudate Used for partial or full-thickness acute or chronic wounds
Alginates (e.g. Kaltostat)	Highly absorbent Medium to heavy exudating wounds Secondary cover required
Hydrogels (e.g. Intrasite gel)	High H_2O content and creates moist wound surface Debrides wound by hydration and autolysis Not suitable for heavily exudating wounds
Foam dressings (e.g. Lyofoam)	Moderately exudating wounds De-sloughs wounds by maintaining a moist environment

Table 3: Classification of wound dressings and their properties[3, 4].

Wound infection and its associate risk

The term now commonly used is surgical site infections (SSIs). It is defined as an infection occurring following a surgically created wound occurring within 30 days unless a foreign body is left in situ. The risk of a surgical site infection is dependent on the type of surgery involved and the aetiology of wound. SSIs are subdivided into three categories based on the depth of tissue involvement[5].

1. Superficial incisional SSIs.

2. Deep incisional SSIs – involving fascia and muscle.

3. Organ space SSIs – any anatomic structure opened or manipulated during the operation.

It was Barard and Gandon who classified the associated risk of surgical site infection depending on the type of surgery involved[6]. The rate of wound infection increases from clean to dirty wounds. The classification of types of surgical wounds and the associated risk of infection is summarised in Table 4. Managing a wound effectively reduces the risk of infection. Once an infection is identified it is important to act quickly and plan the appropriate management. Clinical symptoms and signs of infection largely depend on the severity, site and depth of infection. Superficial skin infections confined to the dermis usually present with surrounding erythema with or without purulent discharge. Pain is usually a late sign of infection and more commonly associated with deeper site infections within the subcutaneous tissue or deeper intra-abdominal infections.

Classification	Criteria	Example of Surgery	Percentage risk of SSI (%)
Clean	Elective Non-emergency Non-traumatic Primary closed wounds No break in epithelial surface Do not require antibiotic prophylaxis	Prosthesis placements Cardiac procedures	2
Clean-contaminated	Urgent or emergency case that is otherwise clean Elective opening of epithelium (i.e. appendicectomy) Minimal spillage	Head and Neck, Thoracic, Biliary and Genito-urinary procedures	10
Contaminated	Non-purulent inflammation, gross spillage of intestinal contents including biliary contents Penetrating trauma		
Dirty	Purulent inflammation Pre-operative perforation of respiratory, gastrointestinal or biliary contents	Colorectal surgery e.g. Hartmann's procedures	60

Table 4: Classification of types of surgical procedures and there associated risk of infection[6].

SURGICAL WOUNDS AND SURGICAL SITE INFECTIONS

Sarit Badiani and Sarah Addison

If an abscess cavity is suspected it is important to adequately drain the infection. This can be done on the ward by releasing some clips over the site of the collection or cutting a segment of sutures, this allows free drainage of the collection. In most circumstances draining a collection may be all that is required to treat wound infections.

Antibiotics alone are of little use when there is an abscess, as they do not penetrate the capsule of the abscess.

In cases where deeper infection is encountered a formal incision and drainage in theatre may be recommended. Antibiotics are usually started and need to be checked according the local trust protocol.

Wound dehiscence

This is defined as a breakdown of a post-operative wound most commonly involving abdominal wounds and occurs in up to 3% of patients[4]. Wound dehiscence can range from superficial dehiscence to deep dehiscence, involving all layers of tissue. It can often be alarming to see this for the first time as the patient will be in bed with an open wound usually with their bowels visibly peristalsing in front of you. Wound dehiscence usually occurs around the 5th post-operative day and is preceded by a serous discharge.

Factors leading to increased risk of wound dehiscence include[4].

1. Poor surgical technique.
2. Sutures are too close or too far apart.
3. Increased tension of closure.
4. Patient factors – obesity and co-morbidities, such as diabetes, malnutrition and malignancy, etc.
5. Underlying infection.

Risk factors associated with abdominal wound dehiscence are summarised in Table 5.

Local Factors	Regional Factors	Systemic Factors
Wound infection	Bowel oedema	Advanced Age
Haematoma	Deep intra-abdominal infection	Malnutrition
Seroma	Haemorrhage	Pulmonary disease
Poor surgical technique	Trauma	Steroids use
		Diabetes
		Neoadjuvant therapy

The treatment of wound dehiscence is achieved by treating the cause and prompt repair of the breakdown. Surgical plan of treating this would include:

1. Cover wound with warm saline soaked gauze.
2. Correction of electrolyte imbalance.
3. Surgical exploration – to rule out any underlying cause of dehiscence as well as resuturing of fascial layers.
4. Debridement of tissue if suspected tissue necrosis.

Summary

Wounds should be examined meticulously and regularly to reduce the risk of infections. It is important to be aware of the different types of surgical wounds and classification of surgery so as to be familiar with the percentage risk of surgical site infections. Wound dressings should be carefully selected according to its properties and depending on the type of would involved.

References

[1.] Mercandetti M, Cohen AJ. Wound Healing, Healing and Repair. **http://emedicine.medscape.com/article/1298129-overview**

[2.] Taub P, Harmaty M. Evaluation of wounds. **http://www.plasticsurgerypractice.com/issues/articles/2005-02_04.asp**

[3.] **http://www.surgical-tutor.org.uk/default-home.htm?specialities/plastic/dressings.htm~right**

[4.] Simpson A, Brooks A. *ABC of Tubes, Drains, Lines and Frames.* Chapter 7, pp. 40–43.

[5.] Harken A, Moore E, Abernarthy C. *Abernathy's Surgical Secrets.* Questions you will be asked.

[6.] Berard F, Gandon J (1994) Postoperative wound infections. *Annals of surgery,* 160:1–192.

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CAREERS FOCUS: FROM FOUNDATION YEARS TO SURGICAL TRAINING

Hannah Winter and Aneel Bhangu

Surgery has always been, and will always be, a very competitive specialty, with good reason.
Teaching & Training.

There have been large changes to the application process for surgery as well as surgical training itself over recent years, with more changes to come. Although the core principles of what you need to do to get into surgical training holds firm, there have been distinct differences in the pre-specialist training years too. Surgery has always been, and will always be, a very competitive specialty, with good reason.

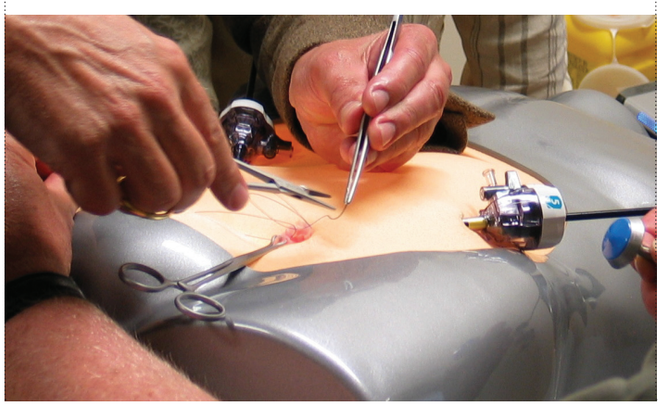
This article aims to give you a guide as to what you can do to get ahead of the surgical game as an FY1 and FY2. The first part of this process is to score maximum points in the application form to secure an interview. After this, the focus is placed on practising interview skills and knowing your CV inside out for the interview phase. In **bold** are the points you should be aiming to match from person specifications.

However, remember that you must balance all this with gaining clinical experience, which is fundamentally what FY jobs are about.

Which specialty?

Currently, specialties (including orthopaedics and neurosurgery) are now run through training where you enter in at Speciality Trainee 1 (ST1). Specialties (such as general or plastic surgery) remain uncoupled, where you enter at Core Training 1 (CT1) and then compete again at ST3.

Although not technically necessary for Foundation competencies, it will help to tailor your FY1/2 jobs to include some of the specialties you wish to apply for, be that in placements or projects you participate in. This will enable you to develop your **knowledge and commitment to the specialty.** During your placements, you must take any opportunity to develop your **basic surgical skills** in the operating theatre, thus improving your surgical logbook.



Audit

Much focus is placed upon audit. It is the **responsibility** of a doctor to continually audit and improve their practise and demonstrates commitment and good **organisational skills.** Before each application round (i.e. around January) you should be aiming to have carried out and presented two audits.

- A good audit in something non-surgical is better than a bad audit in something surgical.
- Audits on drug charts and DVT prophylaxis are typically audit for audit's sake, and are boring.
- For maximum marks, audit something that has affected you. You see management of a case that has affected you (positively or negatively).

You perform a literature review to see how things could have been done differently, so you audit local practise, and suggest areas for improvement and implement them; ideally you re-audit and **close the loop.** Here you score maximum marks (in your FY1/2 assessments, application forms and interviews), for both **reflection** and **audit** around one topic.

- Get the most from your audit. With a good quality and original audit, you can submit it for your hospital's audit **prize,** present it and get it published.

Research

Getting involved with **research** can be difficult with a 4-month post, but you should understand that it is important to help **develop** your chosen specialty and keep your **knowledge current.** Either get involved with something already going on or extend an audit into research territory, although you will need the correct ethical approval to do this. Research that does not involve live subjects or patients is easier (e.g. case note or biochemistry results reviews), as formal ethical approval is not needed (but still seek advice).

Papers and presentations

Getting a publication shows good **organisation** and **written communication skills.** Articles to submit for publication: case reports; audits; career advice; clinical reviews; and research. Try and get something into a **peer reviewed journal** for maximum impact. All of these too can be presented locally, regionally, nationally and internationally – look out for conferences via society websites, but remember you will probably have to fund yourself.

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Training

Start and maintain a logbook (e.g. **www.elogbook.org, www.iscp.ac.uk**). Surgical trainees have to do this, and so showing it at an interview will show a good **understanding of specialty,** as well as development of your **technical skills.**

Courses and exams

The courses which will increase your **technical ability** and show **commitment** to surgery are **Basic Surgical Skills, Advanced Trauma Life Support** and **Care of the Critically Ill Surgical Patient.** You can do these as an FY2, although to get them in before applications, you will have to do them between August and December (bear in mind ATLS has a waiting list). These will be self-funded and you may need to use your annual leave to complete them. These courses are compulsory for the completion of core surgical training.

The Foundation STEP course (Surgical Training Education Programme) is a distance learning programme available to all Foundation trainees, but particularly those interested in a career in surgery. It consists of web-based learning resources and printed modules to include surgical principles and technical skills, ethics and law, communication skills and perioperative management of patients. There are day events held at the Royal College of Surgeons, London, for those enrolled on the course to help participants with portfolio documentation, reflective practise and interview skills. This will also be self-funded.

For the last few years, FY2s have been able to sit the **MRCS Part A** (MCQs). This shows a very high level of **commitment to your specialty,** and should be at the top of your application forms. You will have to sit it early in the academic year to get the result for application time (September at the latest).

Teaching

Teaching is a basic responsibility of a doctor. As an FY2, your deanery may provide time for you to complete a **postgraduate award,** one module of which is teaching. Organise a **teaching course** for medical students and involve other FY1/2s. Producing a rota shows **organisational and managerial skills.** Ask for a letter of recognition from the clinical sub-dean of your hospital to put in your portfolio as **evidence of your teaching ability.**

Application preparation

Although the above will help you develop as a doctor, it is tailored towards your applications, the following aspects of which you should ensure you have achieved:

• **Portfolio** – the best are collated and polished from a very early stage. They contain all of the evidence in a logical and presentable order, including **more than** the basic number of DOPS/ mini-CEXs, etc. Be prepared to talk about any part of your portfolio in detail, including each and every form. Your divisions should include: CV; logbook (consolidation sheet); assessments and appraisals (including DOPS, Mini CEX, 360° assessment, etc.); exams, courses and certificates; teaching and education; research; audits; presentations; publications; reflective practise; management experience; continued development; and any other information that may support you as a candidate, be it personal or professional.

• **Forms** – the applications forms can be daunting. Download them and start early. White space questions will need some time to edit and re-edit; show them to your ST1s, consultants and educational supervisors, but only take on the changes that you like. There is a "knack" to filling out these forms, so make sure that you answer the question and make constant reference to the person specification, from which the mark-schemes are drawn up.

• **Interview** – from an early stage, ask your ST1/2s what came up in their interview to get a grasp of the type of questions asked. The last interview many of you will have had was to get into medical school and so you will need lots of practice. Your reflective diary is paramount in answering some questions during interviews. Many scenarios will be centred around your experience in, for example, an awkward situation, a disagreement with a colleague or your involvement within a multidisciplinary team. Read your reflective diary to remind you of patients and situations before going in for your interview.

• **Curriculum vitae** – maintain a good CV, put it right at the front of your portfolio, be proud of it, and give it to your consultants so they can provide you with references. Know it inside out! The first question you will be asked is "talk me through your CV".

What if I don't get the job?

Don't panic. Do you still want to do it? Then spend some time developing your skills. **Anatomy demonstrating** for a year is always a very positive career move; it deepens your **knowledge of basic clinical sciences** and is a full-time **teaching** job to be proud of. It's early for a **research degree,** but is still possible; ask around and look at the websites of academic surgical departments for more information. If you do "step away" for a year, ensure that you keep current by carrying out an audit with the local surgical department. Try to avoid career gaps. Good luck!

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HOW TO ASSESS PATIENTS WITH BREAST PROBLEMS

Ian S Fentiman

Evaluation of patients with breast problems exemplifies the need for good communication skills, competent examination and clinical judgement so that the majority of patients can be safely and effectively reassured and the minority of individuals with malignancy identified. Good Medical Practice.

Relevance to the curriculum

1. Good clinical practice

1.1 Demonstrates the knowledge, attitude, behaviours, skills and competencies to be able to take a history and examine patients, prescribe safely and keep an accurate and relevant medical record.

1.2 Demonstrates appropriate time management and organisational decision-making.

2. Relationship with patients and communication skills.

2.1 Demonstrates the knowledge, skills, attitudes and behaviours to be able to communicate effectively with patients, relatives and colleagues.

Introduction

Evaluation of patients with breast problems exemplifies the need for good communication skills, competent examination and clinical judgement so that the majority of patients can be safely and effectively reassured and the minority of individuals with malignancy identified.

Many patients attending "one-stop breast clinics" are very worried, and sometimes disproportionally to their likelihood of having breast cancer. Hence it is very important that the consultation is carried out in a calm and unhurried manner, despite the throng of others waiting to be seen. If this does not happen, rather than being a one-off visit, many patients with benign conditions will keep returning for the reassurance that eluded them when first seen.

Triple assessment (clinical examination, imaging and tissue diagnosis) forms the basis for evaluation of women with breast lumps or localised nodularity. Most patients attending "one-stop clinics" will not meet these criteria and many can be reassured and discharged without any further investigations after a normal clinical examination.

History

In most breast clinics patients are asked to complete a questionnaire before being seen, or this forms part of the clinician's history. Stripping away non-essentials and only using key questions as follows:

1. What is the problem and how long has it been present?
2. Any previous breast problems?
3. Any serious illness or major operations?
4. Any regular medications?
5. Any family history of breast cancer?
6. If still having periods, when did the last one start?
7. If postmenopausal, when did this occur?
8. Any children and is so were they breast fed?

What is the problem and how long has it been present?

If the patient says that she has a lump, laterality and duration are determined in addition to whether the size has changed since discovery. Is the lump painful? Although malignant lumps are classically described as painless, about one in ten will be associated with a burning sensation.

The most frequent complaint is painful lumpiness with the patient describing either a lump or thickening. Typically these patients are in their early 30s: those complaining of breast pain without a lump or lumpiness are asked whether the pain is cyclical, is it always in the same place, is it worse on exercise and does it radiate?

With a nipple discharge, is this unilateral or bilateral? Is there any associated lump? What is the colour? If the patient states that the discharge is bloody, is it bright or dark brown like old blood? Is there associated nipple retraction: if so is this long standing or recent and is there any associated pain or discomfort?

Any previous breast problems?

Prime importance is attached to a prior diagnosis of breast cancer, raising the possibility of recurrence or metastasis, but not excluding the possibility of a new benign problem, such as a cyst. Some give a past history of "cysts" which on closer questioning turn out to be lumps that resolved spontaneously. If a previous lump disappeared after drainage of fluid then it was a true cyst. Women who have had multiple cysts aspirated are at three to fourfold increased risk of breast cancer so any new lumps develop require careful evaluation[1].

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Increasing numbers of women pay for breast augmentation or reduction mammoplasty and the alteration in anatomy consequent upon both procedures may give rise to concern about new lumps that are often iatrogenic rather than spontaneous in origin. Nevertheless, because many individuals have now reached an age when breast cancer risk is significant, careful evaluation is necessary to avoid missing malignancy.

Some give a long history of milky discharge usually continuing after lactation. If associated with amenorrhoea or oligomenorrhoea, investigation for possible hyperprolactinaemia from a pituitary adenoma or microadenoma is warranted. Galactorrhoea amid normal menstrual cycles is likely to represent physiological incomplete involution.

Any serious illness or major operations?

Other than giving an indication of general health, there are certain breast conditions more likely to be associated with non-breast problems. A common example is the patient complaining of breast pain with a prior history of arthritis or musculo-skeletal disorder. Subsequent examination will often reveal the pain arises from the rib cage rather than the breast.

A recent history of hysterectomy can be associated with breast symptoms due to inadvertent ovarian artery damage causing endocrine dysfunction. After oophorectomy subsequent hormone replacement therapy (HRT) may induce localised nodularity or cyst formation.

Diabetes may produce a specific mastopathy but most patients with type 2 diabetes are overweight and postmenopausal, placing them at increased risk of breast cancer due to endogenous hyperoestrogenaemia[2].

More problematic are patients with dementia who may present with advanced breast cancer. Such individuals lack mental capacity and so treatment decisions will have to be made in line with the Mental Capacity Act (2005). Luckily, most have oestrogen receptor positive (ER+ve) tumours so that the option of systemic endocrine therapy will be available rather than extensive surgery.

Any regular medications?

This question often yields important clues as to the patient's general health and many claiming no previous serious illnesses will list medications for diabetes angina and atrial fibrillation (such as warfarin). It is customary to ask about oral contraceptive use: previously use tells little but recently starting or changing may produce moderate breast enlargement or tenderness.

Those women taking HRT, now a minority of the postmenopausal, are at some increased risk of breast cancer, depending on age and duration but the most important aspect that should be considered is that irrespective of age, they have de facto premenopausal breasts. Hence lumps may be cystic in women who are in apparently the wrong age group.

Any family history of breast cancer?

Breast cancer affects one in nine women but <10% of breast cancers arise as a result of an inherited mutation so many women needlessly worry because a relative has had the disease. The main mutations so far discovered are in the BRCA1 and BRCA2 genes located respectively on chromosomes 17q21[3] and 13q12-q13[4]. Both encode for proteins involved in DNA repair so the failure to repair DNA adducts leads to early onset breast cancer in mutation carriers.

Potential carriers usually give a history of breast cancer being diagnosed before age 40 in at least one first degree relation and often second degree relatives. Additionally, some relations will also have had ovarian cancer. In the absence of a first degree relative with early onset breast cancer, it is unlikely that the individual will prove to be a mutation carrier. Most of these patients need reassurance, not surveillance.

If still having periods, when did the last one start?

Premenopausal women suffer from a variety of benign conditions, ranging from physiological premenstrual breast discomfort to severe cyclical mastalgia which may eventually, for a few, become constant and have a profound impact on their personal and reproductive life. Typically, severe mastalgia affects women in their 30s and while no underlying causes have yet been found, the condition does respond to a variety of endocrine manipulations including tamoxifen, danazol and bromocriptine.

Some patients with breast discomfort are in the earliest stage of pregnancy, which may be surmised on a basis of the last menstrual period under circumstances where the individual may not be aware of her achievement. Women nearing the menopause are likely to have irregular cycles which may also lead to breast discomfort that can be bilateral or unilateral.

If postmenopausal when did this occur?

The majority of breast cancers will be found among the postmenopausal but although the most likely explanation for a new lump, cysts and inflammatory lesions may also occur. Recent nipple inversion is also a presentation of malignancy but can also result from duct ectasia. Breast pain is rarely a manifestation of cancer in this group and the explanation is usually musculo-skeletal, and occasionally referred pain from angina or cholelithiasis.

Any children and if so were they breast fed?

Although epidemiological studies indicate that parity is protective up to age 30 and thereafter increases breast cancer risk, in absolute terms the effect is small. Most breast cancer cases have no discernible risk factors and many had their first baby before age 30 and breastfed[5].

Those who are lactating when seen may have various complaints. These include blood-stained milk, often at the commencement of breast feeding and which settles spontaneously, or a lump. There are three likely explanations for the latter. A galactocoele tends to be a well-circumscribed lump often rapid in onset and sometimes painful. Often the "lump" is simply an area of nodularity and very occasionally it proves to be malignant.

HOW TO ASSESS PATIENTS WITH BREAST PROBLEMS

Ian S Fentiman

Examination

Ideally the examination is carried out in a warm, well-lit room. The patient is asked to undress down to the waist and lies on a couch with the head end elevated to 45^0 so that she is not lying flat, which can cause distress for some older patients.

The examination comprises various stages:

- Inspection with the arms by the side.
- Inspection with the arms elevated.
- Palpation of the unaffected breast.
- Palpation of the symptomatic breast.
- Palpation of the unaffected breast in the semi-lateral position.
- Palpation of the symptomatic breast in the semi-lateral position.
- Palpation for metastatic disease, when appropriate.

Inspection with the arms by the side

At first glance, is there gross asymmetry or difference in height of the nipple areola complex? Frequently the left breast is slightly larger than the right, which may be the reason for the slightly greater frequency of left-sided cancers in both females and males[6]. The nipples are inspected for evidence of inversion (Paget's disease) or the presence of skin papillomas. Are there any skin changes, such as scars, peau d'orange, dimpling or discoloration. At this point the patient is asked, when appropriate, to indicate the lump that she then feels to recheck that area for evidence of swelling or dimpling.

Inspection with the arms elevated

The patient's arms are then gently held at the wrists and she is asked to lift the arms. The site of the lump is carefully observed to determine whether this action reveals skin dimpling. Elevation of the arms leads to visualisation of the axilla and, depending on breast size, the inframammary folds, but the breast tissue may need to be lifted manually to display this region. In patients who had breast augmentation, the scars from the implant insertions may be difficult to see and may be inframammary, circumareolar or axillary. Women with prior breast cancer may have mastectomy scars or if breast conserving surgery has been performed: the scars may be more subtle, particularly if sentinel node biopsy has been performed, but there may be residual blue discoloration from patent blue dye of small skin tattoo marks for radiotherapy alignment.

Palpation of the unaffected breast

There are several methods of breast palpation: the essential aspect is a methodical, thorough and apparently unhurried examination. One such technique is to divide the breast into a series of vertical columns, about 3cm wide, in the mind's eye. Starting at the lower inner aspect, the breast tissue is palpated with the tips of the middle three fingers of the dominant hand, using the non-dominant hand to push and pull the breast tissue so that there is as little as possible between the examining fingers and the chest wall. After palpating up and down these columns the nipple areola complex will be reached and this needs to be gently pushed horizontally and vertically to determine whether there are any small sub-areolar masses.

The palpation continues laterally to the axillary tail. The arm on that side is gently supported by the examiner's ipsilateral hand so that the suspensory ligament of the axilla is relaxed enabling the contralateral hand to palpate the axillary contents.

Palpation of the symptomatic breast

The same technique is then employed to examine the symptomatic breast. If a lump is found the following need to be recorded:

- Size (horizontal and vertical dimension in mm).
- Location.
- Mobility (attachment ± to skin and pectoralis major muscle).
- Margins (well or poorly defined).
- Texture (hard, firm or soft).

Dimensions of the tumour are measured with callipers and recorded in mm, not as "walnut-sized", "pea-sized" or some such non-quantitative description. The location is recorded in terms of quadrant, using imaginary vertical and horizontal lines through the midpoint of the nipple, giving upper outer, lower outer, lower inner and upper inner quadrants. When this is not possible because the lump is at a junction of 2 quadrants, its position is described as upper central aspect, lateral, subareolar and so on. Periareolar lesions can be described in terms of the clock face.

Mobility in relation to the skin is determined by immediate dimpling on arm elevation or consequent to manual movement of the lump, demonstrating fibrosis of the ligaments of Astley Cooper. There may be overlying peau d'orange overlying the lump, but this may be sited inferiorly. Such changes may be subtle and only detectable in a good light.

HOW TO ASSESS PATIENTS WITH BREAST PROBLEMS

Ian S Fentiman

Breast examination.
Good Medical Practice.

Palpation of the unaffected breast in the semi-lateral position

The patient is then asked to turn half on her side towards the non-affected side and her arm is placed above the head, resting on the pillow. This manoeuvre enables re-palpation of the breast and often reveals lumps in the upper outer quadrant that were undetected in the supine position. Failure to detect such lumps may result in litigation that is both humiliating and expensive.

The breast is re-examined, based on imaginary vertical or oblique columns so that a thorough second palpation has been performed. The axilla is also re-palpated with the patient in this position.

Palpation of the symptomatic breast in the semi-lateral position

The patient is asked to turn half on her side in the opposite direction so that the symptomatic breast can be re-examined in a similar manner. Dimensions of palpable lumps and axillary nodes are rechecked and recorded and any other palpable breast masses are noted.

If the patient is complaining of pain and clinical examination has not revealed any lumps, the final step is to determine the site of the pain. In most cases, as the breast tissue falls away from the ribs it will be possible to place a finger on the rib from which the pain is emanating. This may produce an involuntary movement as the rib is touched which is pathognomic of rib cage pain.

If the patient is complaining of a nipple discharge, an attempt is made to express this and if unsuccessful the patient is asked to try. When a patient has a palpable lump and a discharge, almost invariably determining the nature of the lump explains the discharge and the diagnosis can be made by FNAC or excision biopsy. In the absence of a lump, the most important first line investigation is testing the discharge for haemoglobin (Hb), using standard urine-testing sticks. If no haemoglobin is present, surgical intervention is almost never necessary. Irrespective of the colour, if Hb+ discharge emanates from one duct, patients at Guy's Hospital are advised to undergo microdochectomy. In a series of 284 microdochectomies, all those with cancer had Hb+ discharge[7].

Palpation for metastatic disease, when appropriate

If there is clinical suspicion of malignancy, the clinical status of the axillary nodes will have been determined already as part of the routine examination. Another potential regional site of metastasis is the supraclavicular fossa (SCF). Both SCF are examined from behind with the patient sitting up and the presence of palpable nodes is measured and recorded.

The abdomen is palpated particularly for hepatomegaly and the lungs are percussed to exclude gross pleural effusion. Although these aspects of the clinical examination are enshrined in the evaluation of breast cancer, they rarely yield positive signs except in those relatively rare patients with locally advanced disease.

Outcome of clinical evaluation

At the conclusion of the clinical evaluation the examiner assigns the patient to one of five groups, depending upon clinical suspicion. These are E (evaluation) 1–5 and definitions of these are given in Error! Reference source not found.Table 1. For patient classified as E1; if there is localised nodularity in a woman aged <40, an ultrasound is usually ordered (although its function is largely to act as a placebo). Most of these patients need reassurance and are discharged. For patients with apparent fibroadenomas aged ≥25, FNAC is performed during ultrasound to confirm the diagnosis.

Younger women (<25) with ultrasound confirmed fibroadenomas, do not need to have a tissue diagnosis made. All patients with suspected cancer need to have both ultrasound and mammography, together with a core biopsy. Additionally ultrasound of the axilla is performed and if an axillary abnormality is present, FNAC is performed. Those with a malignancy and a normal axillary ultrasound are suitable for sentinel node biopsy.

HOW TO ASSESS PATIENTS WITH BREAST PROBLEMS

Ian S Fentiman

E	Definition	Mammos	U/S	FNAC	Core
1	No lump	≥40 years	Local nodularity	No	No
2	Benign lump	≥40 years	Yes	Yes	No
3	Equivocal lump	≥40 years	Yes	No	Yes
4	Suspicious lump	Any age	Yes	No	Yes
5	Malignant lump	Any age	Yes	No	Yes

Table 1: E (evaluations 1–5 and definitions. Protocols for investigation of breast abnormalities may vary from unit to unit, but the core investigations performed in all centres are shown in bold.

Communication

If at the end of the clinical evaluation and the examiner is of the opinion that the lump is malignant, it is best to share this with the patient and explain that various investigations will be needed to confirm the diagnosis. At this point protocols diverge. In some units, the lump is subjected to fine needle aspiration cytology after imaging, which is reported immediately. The patient is seen again, given the diagnosis and often a treatment plan is outlined.

In my unit, we do not use FNAC to make a diagnosis of malignancy but instead get a core biopsy performed during imaging. The patient, who then sees both the consultant and the breast care nurse, is warned that she probably does have breast cancer but the pathology results will be available in a few days. She is asked to return, with a friend or relative, for the definitive diagnosis and a treatment plan that will have been discussed by the multidisciplinary team before she is next seen in the results clinic.

This slight delay enables the patient to start the process of coming to terms with the diagnosis and allows for a full discussion of a treatment plan since receptor status (oestrogen, progesterone and HER2) will be available from immunohistochemistry of the core biopsy. Also almost all patients respond to the potential diagnosis by bringing a relative or friend. This provides the benefit of support from the companion, who, it is hoped will be able to listen more dispassionately and remember what has been said so that areas of uncertainty can be better understood.

Conclusions

Careful history taking, thorough clinical examination and targeted selection of investigations will enable the majority of women with benign breast problems to be seen and reassured at a "one-stop clinic". For those with suspected cancer, a calm sympathetic response will make the difficulties of the diagnosis and treatment easier to understand with a greater likelihood of the patient accepting optimal local and systemic treatment.

References

1. Dixon JM, McDonald C, Elton RA, Miller WR (1999) Risk of breast cancer in women with palpable breast cysts: a prospective study. *Lancet* 353:1742–1745.

2. Key TJA, Pike MC (1988) The role of oestrogens and progestagens in the epidemiology and prevention of breast cancer. *Eur J Cancer Clin Oncol* 24:29–43.

3. Miki Y, Swensen J, Shattuck-Eidens D, et al. (1994) A strong candidate for the breast and ovarian cancer susceptibility gene BRCA1. *Science* 266:66–71.

4. Wooster R, Bignell G, Lancaster G, et al. (1995) Identification of the breast cancer gene BRCA2. *Nature* 378:789–791.

5. Salber EJ, Trichopoulos D, MacMahon B (1969) Lactation and reproductive histories of breast cancer patients in Boston, 1965–1966. *J Natl Cancer Inst* 43:1013–1024.

6. Fentiman IS, Fourquet A, Hortobagyi G (2006) Male breast cancer. *Lancet* 367:595–601.

7. Chaudary MA, Millis RR, Davies GC, et al. (1989) Nipple discharge. The diagnostic value of testing for occult blood. *Ann Surg* 124:253–255.

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CASE REPORT PATHOLOGICAL FRACTURES: INVESTIGATING UNKNOWN PRIMARY TUMOURS

Aneel Bhangu and Richard Dias

A 60-year-old man had noticed some mild pain and localised swelling around his left hip for 3 months. He had been losing some weight and was a lifelong smoker. One morning, he stood up from a chair and heard a loud crack from his left hip. He was in immediate pain and was unable to mobilise. An ambulance brought him to hospital where an X-ray was taken (see Figure 1). A subsequent scan performed is shown in Figure 2.

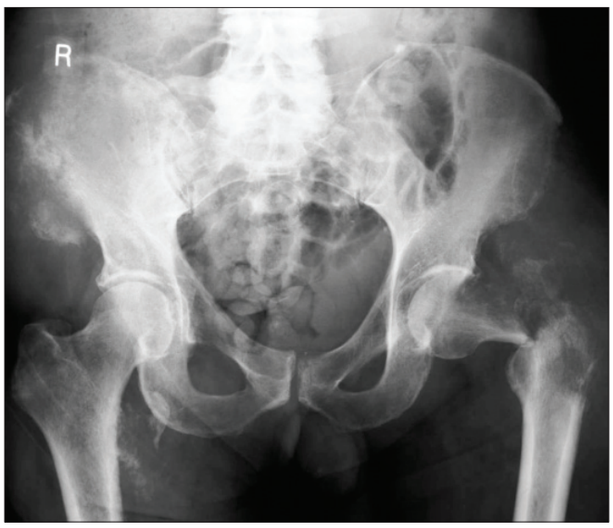


Figure 1: The first X-ray of a patient's left hip.

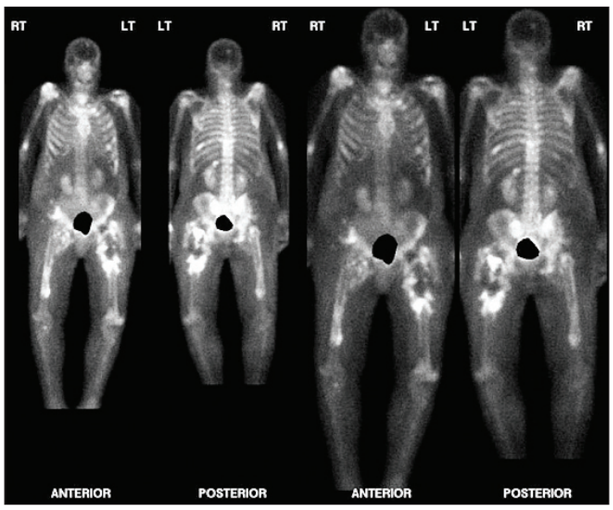


Figure 2: A series of X-rays.

Questions

1. What does Figure 1 show?

2. How will you investigate?

3. What is Figure 2 and what does it show?

4. What are the treatment options?

Answers

1. There is a fracture of the left proximal femur, through a lytic lesion. Further bony destruction is seen in the right iliac crest. This is a *pathological fracture,* and due to the presentation and history it is most likely caused by metastatic bone disease which is secondary to an unknown primary tumour.

2. All patients should be fully assessed with a thorough history, examination and then simple and more complex investigations.

• **History:** weight loss; tiredness; bone pain (indicating possible locations of metastatic deposits); haemoptysis; haematemesis; malena; blood per rectum; tenesmus; haematuria; dysuria; and breast lumps. Bone pain is important to identify metastatic deposits, as prophylactic fixation prior to fracture is more desirable and results in less morbidity.

• **Examination:** cachexia; pallor; lymphadenopathy; respiratory system (although may be normal with lung cancer); abdominal system and digital rectal examination (for masses, prostate and blood); breasts; spine (for painful vertebrae).

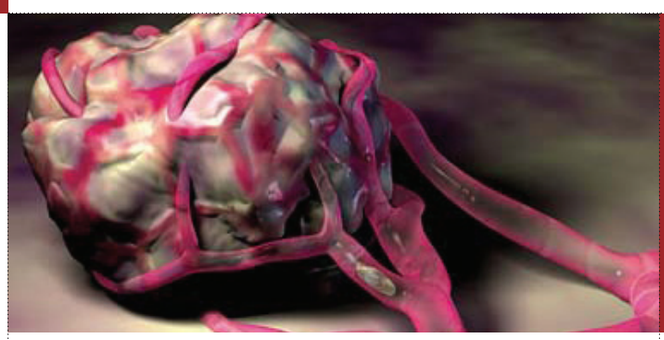
Investigations

• **Simple:** FBC for anaemia; U&E for renal function; bone profile and calcium (may be raised due to bone displacement from metastatic lesions); liver function tests (although these are non-specific); urine dipstick; chest X-ray (for lung lesions); and mammogram (if suspected). Most tumour markers lack the sensitivity and specificity to be used as screening tools[1]. Full length femur X-rays are needed.

• **More complex:** CT chest/abdomen/pelvis is a good place to start to identify solid tumours. Endoscopy (upper and lower GI) can be used to investigate suspected GI cancers. Bone scan, ultrasound and MRI (local area, e.g. pelvis) are used in addition to CT to assess metastatic spread.

CASE REPORT PATHOLOGICAL FRACTURES: INVESTIGATING UNKNOWN PRIMARY TUMOURS

Aneel Bhangu and Richard Dias



The patient presenting with widespread metastasis and an unknown primary is a situation you are likely to come across as a junior doctor.
Patient Management.

3. This is a nuclear bone scan, showing "hot spots" in the left hip, right femur, pelvis, vertebrae, humerus and ribs. Bone scans use radioactively labelled technicium which is taken up by cells with high turnover (e.g. malignant cells) and so shows hot-spots when using a gamma camera.

4. Pathological fractures require treatment of both the fracture and underlying disease process. Patents presenting with fractures are initially stabilised with ABC principles (they may have sustained injuries elsewhere or may be severely dehydrated), and then consideration is given to either *surgical* or *non-surgical* treatment. Surgical treatments of pathological fractures include internal fixation (such as intramedullary nails) or arthoplasty. Furthermore, *prophylactic fixation* of impending fractures may be necessary to prevent further fractures. Non-surgical treatments may be suitable for patients who are too unwell for an operation, or where an operation potentially may cause more harm than good. In our patient's case, due to extensive destruction of bone a massive joint replacement was undertaken (an *endoprosthesis,* Figure 3).

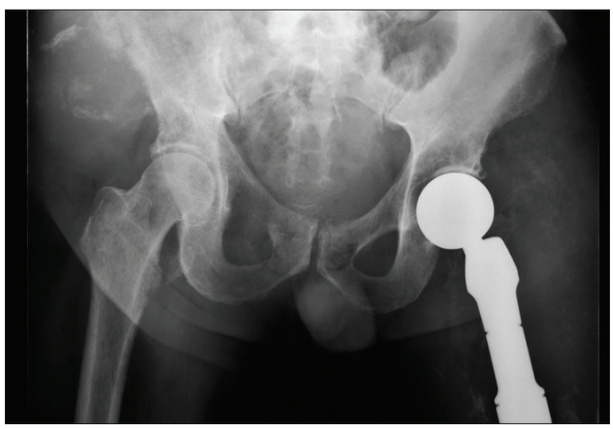


Figure 3: Massive endoprosthesis of the left proximal femur. Margins of clearance could not be achieved and so this was a palliative procedure.

Discussion

The patient presenting with widespread metastasis and an unknown primary is a situation you are likely to come across as a junior doctor. This type of patient presents to a wide variety of specialties, whether it be orthopaedic surgeons, general surgeons or any of the medical specialties. Due to the complexity and poor prognosis, care under the multidisciplinary team is vital.

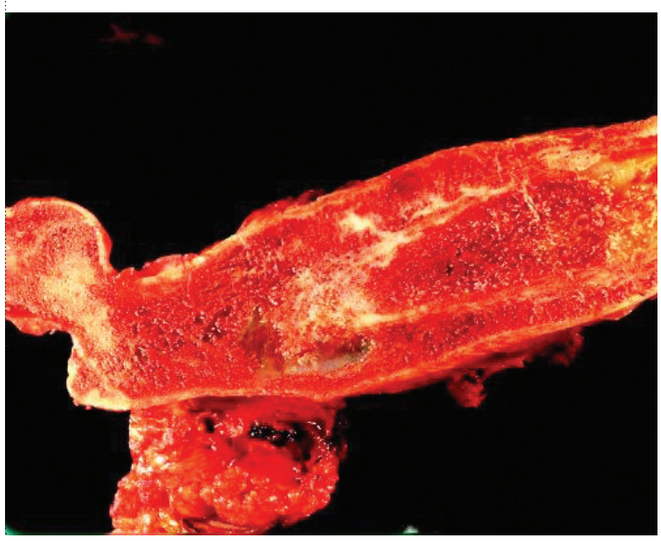
Pathological fractures

Pathological fractures are low energy fractures through abnormal bone which has been weakened due to another disease process. Bony metastases are the most common cause, with other causes including fibrous dysplasia, primary hyperparathyroidism, Paget's disease and bone cysts[2].

Bone tumours

The vast majority of bony malignancies are *secondary* tumours (>90%); *primary* tumours are uncommon, and include osteosarcomas, Ewing's sarcomas and chondrosarcomas[3]. Prostate cancer typically produces sclerotic bone lesions (increased bone production) whereas the other tumours typically produce lytic lesions (bony destruction, Figure 1). Multiple myeloma is a malignant condition which can also predispose to pathological fractures.

The five primary cancers which commonly metastasise to bone are breast, prostate, lung, kidney and thyroid. The five most common locations for metastases are bone, brain, breast, liver and lung.



CASE REPORT PATHOLOGICAL FRACTURES: INVESTIGATING UNKNOWN PRIMARY TUMOURS

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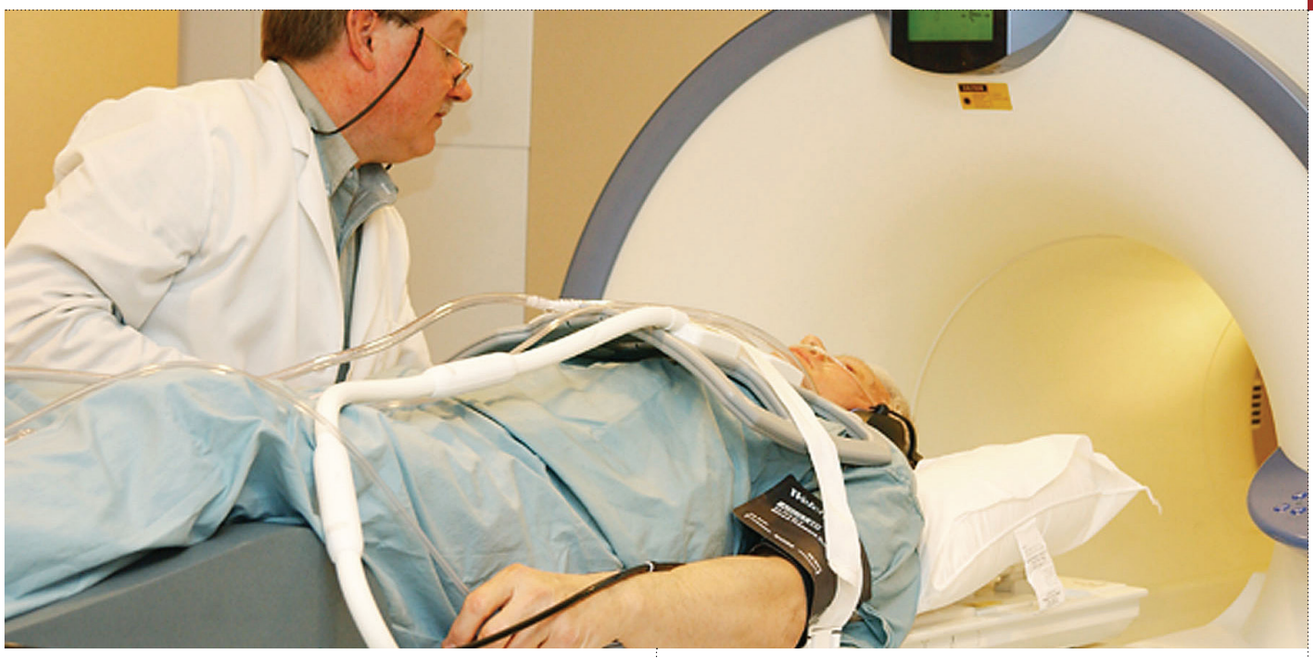

Our patient

A CT scan of his chest, abdomen and pelvis, which showed lesions in the lung, liver and bones, but no clear primary. Histology from the femoral head revealed a mucoid producing tumour which fits with a likely primary of lung cancer. Adjunctive radiotherapy to his hips was planned, but the patient rapidly declined and died within 2 months of presentation.

Unknown primaries

It is useful to determine the primary tumour, as this gives information about prognosis and can guide the need and type of radiochemotherapy. There are some situations where a primary tumour with limited metastases can be treated to cure, such as colorectal cancer with a small liver secondaries; this type of treatment is rapidly improving and becoming an option for more patients[4]. However, when metastases are widespread, as in our patient's case, prognosis is generally poor[5]. Sometimes the primary tumour will never be found, although palliative therapies can still be used if the patient can tolerate them.

References

1. Duffy MJ (2001) Clinical uses of tumour markers: a critical review. *Crit Rev Clin Lab Sci.* June, 38(3):225–262

2. Dent JA (1997) Fractures in the elderly. In: JA Dent (ed.) *Churchill's mastery of medicine and surgery.* New York: Churchill and Livingstone, p. 27.

3. Gebhard M, Mankin HJ (1994) Management of bone tumours. In: PJ Morris and RA Malt, *Oxford Textbook of Surgery,* vol. 2. Oxford: Oxford University Press, pp. 2460–2463.

4. Pozzo C, Barone C, Kemeny NE (2008) Advances in neo-adjuvant therapy for colorectal cancer with liver metastases. *Cancer Treat Rev.* June, 34(4):293–301.

5. Wair JC (1994) Benign and malignant tumours of the lung. In: PJ Morris and RA Malt, *Oxford Textbook of Surgery,* vol. 2. Oxford: Oxford University Press, pp.1895–1912.

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SCROTAL SWELLINGS: A JUNIOR DOCTOR'S DIAGNOSTIC DILEMMA

Amr Hawary and Ian Pearce

Scrotal swellings are a common presenting complaint which, if assessed and investigated in a logical way should not pose a significant diagnostic dilemma.
Good Clinical Care.

Introduction

Scrotal swellings are a common presenting complaint which, if assessed and investigated in a logical way should not pose a significant diagnostic dilemma.

As always good assessment begins with a thorough history, not only focused but also including any possible systemic features (e.g. weight loss and backache).

Patients with scrotal swellings should be examined in both the erect and supine positions and the non-affected testis should be examined first.

The first critical question the clinician must answer in order to be successful in reaching the correct diagnosis is whether the swelling truly originates from the scrotal contents or whether the swelling occupies the scrotum but has a higher origin in the groin. Put simply, "Can you get above the swelling?" If the answer to this question is no then the swelling is not truly scrotal in origin and is most likely to be an inguinoscrotal hernia (Indirect inguinal hernia).

This review addresses, the true testicular swellings or swellings you can get above, which are commonly encountered in surgical practise which include: hydrocoele; varicocoele; epididymal cyst; testicular tumours; testicular torsion; torsion of testicular appendix; and epididymo-orchitis.

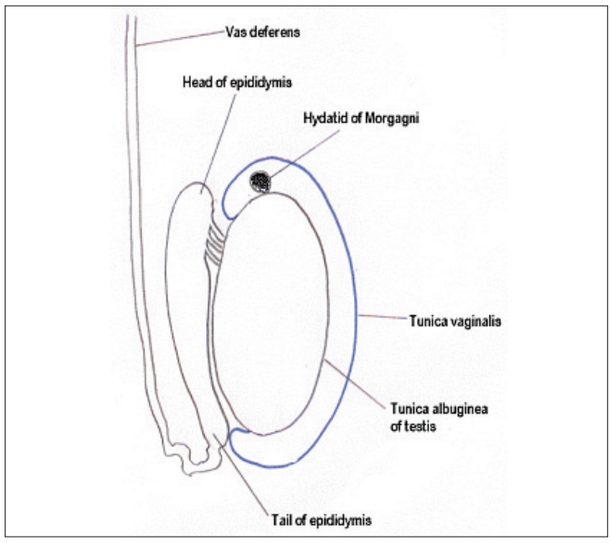


Figure 1: Testicular anatomy.

Hydrocoele

A Hydrocoele is a fluid collection in the serous space between the tunica albuginea and the tunica vaginalis that surrounds the testis and may be congenital or acquired.

Congenital hydrocoele

The processus vaginalis is an out pouching of the peritoneum which descends with the testes via the inguinal canal into the scrotum at around the 28th week of gestation. During infancy and childhood this patent connection between the scrotum and peritoneal cavity gradually closes over. Failure to close results in a patent processus vaginalis with fluid descending from the peritoneal cavity into the space between the tunica vaginalis and albuginea. This is known as a communicating hydrocoele, its incidence is between 2–5% with more than 90% of these congenital hydrocoeles resolving during the first year of life as a result of the spontaneous closure of the processus vaginalis. They usually present with the classical history of a fluctuating scrotal swelling increasing throughout the day but absent upon waking as a consequence of fluid shift back into the peritoneal cavity while supine.

Those congenital hydrocoeles without a patent processus vaginalis are termed non-communicating and present with a constant scrotal swelling with similar characteristics to those found in the adult population.

A variant of the congenital hydrocoele is a hydrocoele of the cord, in this type of hydrocoele, the distal end of the processus vaginalis closes correctly while the mid portion remains patent. The proximal end may be open or closed in this type of hydrocoele and this will dictate whether or not the swelling fluctuates with any change in body position.

Acquired hydrocoele

Acquired hydrocoeles usually occur due to an imbalance between fluid production and fluid absorption within the tunica vaginalis, (Primary) but may also occur as a consequence of intra-scrotal pathology, e.g. malignancy, trauma or infection (Secondary).

SCROTAL SWELLINGS: A JUNIOR DOCTOR'S DIAGNOSTIC DILEMMA

Amr Hawary and Ian Pearce

Presentation

Acquired hydrocoeles usually present as a painless large diffuse scrotal swelling, which may be of gradual or acute presentation depending upon the presence or otherwise of any underlying pathology.

A dull ache due to the weight of the hydrocoele may be present and if very large, the swelling may be visible through the patients clothing or even interfere with the ability to perform sexual intercourse. Some patients present because they simply cannot accept the cosmetic appearance of their scrotal swelling.

The majority of hydrocoeles are of gradual onset signalling their primary benign nature but those presenting acutely are usually secondary to testicular pathology (e.g. malignancy, trauma, etc.).

Examination

The scrotal swelling is usually soft and non-tender with no signs of infection or inflammation, (unless the hydrocoele is secondary and of acute onset). It is possible to get above the swelling but the testis cannot be palpated separately from it.

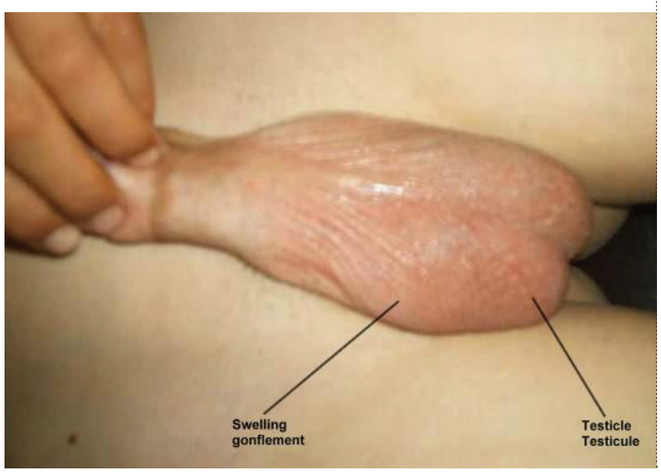
The testis itself may be palpable if the hydrocoele is lax but it is not uncommon for the underlying testis to be incompletely palpable preventing adequate assessment or indeed to be completely impalpable due to the tense nature of some hydrocoeles.

Hydrocoeles transilluminate (light up in their entirety) when light is placed on the skin covering the swelling indicating its fluid content. This test, however, does not distinguish a hydrocoele from a large epididymal cyst.

Investigations

The diagnosis is usually reached based on history and clinical examination. If in doubt, or if the testis cannot be adequately assessed, a scrotal ultrasound scan should be requested.

With experience, clinical examination is as accurate as ultrasonography in diagnosing benign scrotal swellings.



Treatment

Conservative treatment

In many patients when the swelling is small and not interfering with daily activities, there is no need for surgical intervention and the patient can be advised to seek medical advice should the clinical picture change.

Aspiration and sclerotherapy

This entails aspiration of the Hydrocoele fluid and injection of sclerosing material, such as tetracycline. Such an approach is associated with high degree of recurrence and pain, and increases the risk of bleeding and infection. It is usually reserved for those patients who are symptomatic but unfit for surgery.

Surgical treatment

Surgery remains the mainstay treatment for those patients who are symptomatic. The indications for surgical treatment in adults are either a dull aching testicular pain, a huge scrotal swelling interfering with the quality of life or cosmesis alone. Many newborn hydrocoeles resolve before the age of 1 year, hence observation is usually appropriate until this age.

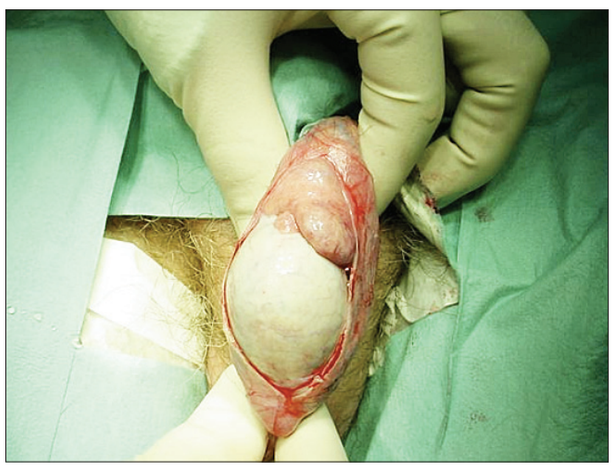
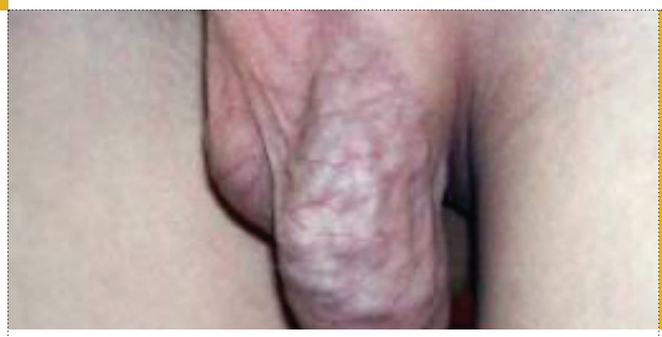


Figure 2: Scrotal hydrocoele.
Courtesy of Urology Department, Manchester Royal Infirmary

SCROTAL SWELLINGS: A JUNIOR DOCTOR'S DIAGNOSTIC DILEMMA

Amr Hawary and Ian Pearce



A varicocoele is an abnormal dilatation of the pampiniform venous plexus of veins within the spermatic cord and is found in approximately 15% of adult and adolescent men, with a marked left-sided predominance (85%).
Good Clinical Care.

Varicocoele

A varicocoele is an abnormal dilatation of the pampiniform venous plexus of veins within the spermatic cord and is found in approximately 15% of adult and adolescent men, with a marked left-sided predominance (85%).

Aetiology

Varicocoele formation is thought to be due to multiple primary factors:

- Higher venous pressure in the left renal vein (into which the left gonadal vein drains).
- Collateral venous anastomoses.
- Incompetent valves of the internal spermatic vein.

Presentation

Varicocoeles are usually asymptomatic but may present as a dull dragging discomfort and are usually referred as scrotal swellings, although a small minority are referred as a consequence of male factor infertility. The majority of patients with a varicocoele have normal fertility.

Examination

The patients must be examined in both the erect and supine positions, with and without the valsalva manoeuvre. The varicocoele is usually a painless compressible swelling that lies posterior or occasionally surrounding the testicle. The classical description of varicocoele is "the feeling of a bag of worms". It is very important that the testicles are properly examined paying special attention to testicular volume which may be reduced as a consequence of the varicocoele.

Varicocoeles should disappear in the supine position, failure to do so should raise the suspicion of a renal cell carcinoma compressing the renal/gonadal vein and mandates upper renal tract imaging (e.g. renal ultrasonography). A right-sided or acute onset varicocoele should also alert the clinician to the same possibility.

Classification

Grade 1: small varicocoele, only palpable with valsalva.
Grade 2: moderate size varicocoele, easily palpable without valsalva.
Grade 3: large varicocoele, visible through scrotal skin.

Investigations

When the clinical examination findings are inconclusive, high-resolution, colour-flow Doppler ultrasonography is the diagnostic method of choice.

Treatment

Indications for treatment of varicocoeles are:

- pain
- cosmesis
- unilateral testicular atrophy before puberty
- male factor infertility.

There are multiple approaches of varicocoele ablation all aimed at occlusion of the dilated veins. This may be done surgically, (either open or laparoscopically) or with radiological embolisation.

While varicocoele repair results in an improvement in semen parameters for those patients with male factor infertility and a clinical varicocoele, whether this brings with it an increase in the conception rate has not been conclusively proven as most studies terminate at 12 months and are, hence, too short for adequate evaluation.

The potential side effects of varicocoele repair include pain, infection, hydrocoele formation, (20%) and testicular atrophy secondary to ischaemia (1%).

Epididymal cyst

An epididymal cyst is a fluid filled sac arising from the epididymis, most commonly the head.

Presentation

Epididymal cysts are usually asymptomatic and rarely cause pain or discomfort with most cysts being either found incidentally by the patient or discovered on routine clinical examination. Occasionally the shear size of the cyst may stimulate patient referral on the basis of its cosmetic appearance.

Examination

Epididymal cysts are confined to the scrotum, it is possible to get above them to exclude a hernia and the testis is separately palpable allowing exclusion of a hydrocoele. They are typically painless, smooth and spherical swellings arising from the epididymis with a positive transillumination test.

Investigations

In the majority of patients history and clinical examination are sufficient to confidently reach a diagnosis. Scrotal ultrasound may be utilised if in doubt.

SCROTAL SWELLINGS: A JUNIOR DOCTOR'S DIAGNOSTIC DILEMMA

Amr Hawary and Ian Pearce

Treatment

Epididymal cyst excision is only indicated for pain or discomfort, cosmesis or patient anxiety.

Complications are similar as for hydrocoele repair.

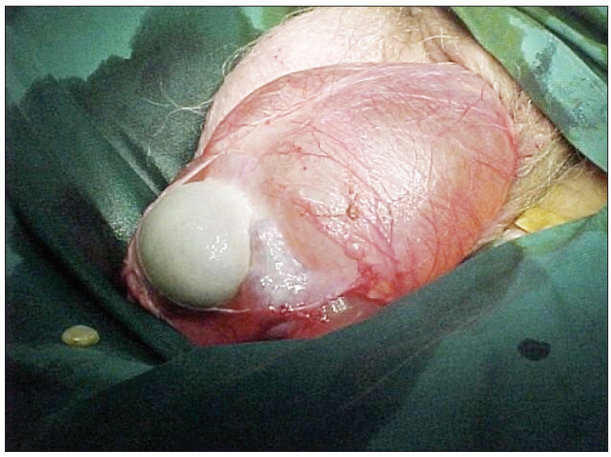


Figure 3: Epididymal cyst.
Courtesy of Urology Department, Manchester Royal Infirmary

Testicular tumours

Testicular malignancy is the most common cancer in men aged 20 to 35 years; it can be broadly classified to Seminomas and Non-Seminoma Germ Cell Tumours (NSGCT), or teratomas with some tumours having elements of both.

Risk Factors:

- criptorchidism or testicular maldescent
- family history
- malignancy in the contralateral testis
- Klinefelter's syndrome.

Presentation

Testicular tumours can present with different clinical scenarios:

- Feeling a painless hard swelling or lump in the testicle.
- Acute pain if bleeding into a tumour occurs. (15%).
- Secondary Hydrocoeles.
- Pain and discomfort or change of sensation in the testicle.
- Signs and symptoms of metastatic spread eg: Back ache, haemoptysis, and weight loss.

Examination

Examination should begin with a general, chest and abdominal examination, in particular assessing for the presence of para-aortic lymphadenopathy

Testicular examination will reveal a swelling confined to the scrotum, which is easy to get above. The swelling is painless, not separate from the testis and is usually hard and craggy in nature.

As always, the unaffected testis should be examined first.

Investigations

Whenever there is any clinical suspicion of testicular malignancy, an urgent scrotal ultrasound should be arranged to confirm or refute the diagnosis.

If this confirms the presence of a testicular tumour then the following should be arranged in addition to arranging an urgent radical orchiectomy via an inguinal approach:

- Serum Alpha-fetoprotein (AFP).
- Serum Beta-human chorionic gonadotropin (_-hCG).
- Serum Lactate dehydrogenase (LDH).
- Abdomino-thoracic CT scan.

Counselling

One of the greatest challenges that a junior doctor will face is breaking bad news.

Explaining to a young fit male that he has testicular cancer is a prime example of this. It is imperative that such news should be given in the presence of other supporting staff (e.g. specialist nurse) and preferably in the presence of a patient's relative, providing the patient consents to this.

The patient should be advised that with proper treatment the cure rate exceeds 90% but that adjuvant therapy may be required and this may impair fertility. Sperm banking should be offered to every patient pre-operatively together with the option of inserting a testicular prosthesis for cosmesis either immediately or following treatment completion.

Testicular torsion

Testicular torsion is a true urological emergency requiring urgent scrotal exploration. It usually occurs around puberty although it has been described in all age groups from infants (extravaginal torsion) up to adult life.

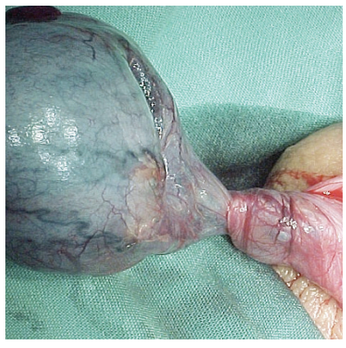
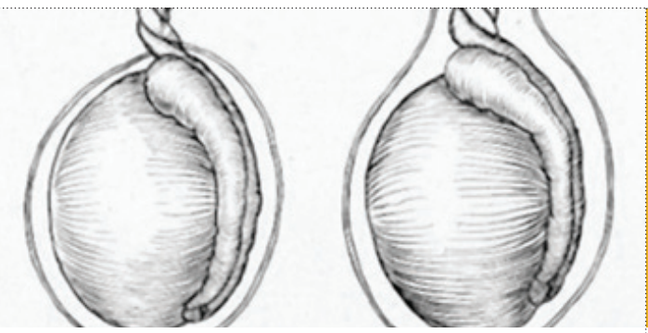


Fig 4 : Infarcted Testis

SCROTAL SWELLINGS: A JUNIOR DOCTOR'S DIAGNOSTIC DILEMMA

Amr Hawary and Ian Pearce



Extravaginal testicular torsion.
Good Clinical Care.

Predisposing factors

Classification

- Extravaginal testicular torsion in which the testis and its coverings twist in their entirety within the scrotum. Testicular torsion in neonates and intrauterine life is of this type.
- Intravaginal testicular torsion in which the testis twists within the confines of the tunica vaginalis. This type of testicular torsion is the type that occurs in nearly all other age groups.

Presentation

The diagnosis is usually based on history of acute, severe unilateral testicular pain which may be associated with a scrotal swelling. Lower abdominal pain and vomiting.

It must be borne in mind that some of these symptoms may be absent.

It is very important to have a proper history including history of testicular trauma or any other urinary symptoms. A history of lower urinary tract symptoms (e.g. dysuria) is more typical of infection rather than testicular torsion, but this is not conclusive and torsion should still be considered in this setting.

Examination

The diagnosis of testicular torsion is primarily a clinical one, with examination revealing a very tender, swollen testis which may be impossible to assess adequately due to pain. The testis may be high riding and the contralateral testis may have a classical bell clapper lie.

Investigations

Once a diagnosis has been made or is suspected there is no time to be lost in obtaining routine investigations. Venous oedema resulting in arterial insufficiency and ischaemia occurs within hours with testicular loss usually by 8 hours following acute testicular torsion. If possible without delay, colour Doppler ultrasonography of the testis gives valuable information regarding testicular blood flow. A torted testis usually shows no or poor perfusion.

Management

Immediate surgical exploration is the only treatment for testicular torsion. If torsion is confirmed and the testis is viable following de-rotation, bilateral orchidopexy with non-absorbable sutures should be performed. If the testis is not viable orchiectomy is performed with contralateral orchidopexy and if no torsion is discovered, the scrotum should be closed without further intervention.

Counselling

Patients should be counselled that failure to correct testicular torsion will result in testicular atrophy and loss and that if the testis is not viable following de-rotation, orchiectomy will be performed. While this reduces fertility, this reduction is usually compensated for by the female partner.

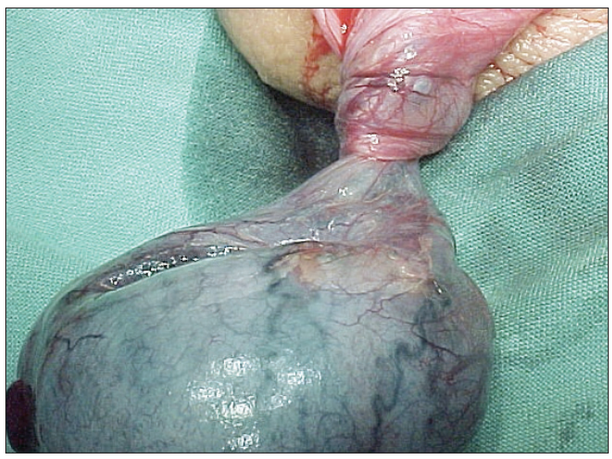


Figure 4: Testicular torsion.
Courtesy of Urology Department, Manchester Royal Infirmary

Torsion of testicular appendage (hydatid of Morgagni)

Torsion of a testicular appendix is a further cause of an acute scrotum in young boys with the peak age between 10 and 12 years old. The hydatid of Morgagni is an embryological remnant of the Mullerian ducts on the upper pole of the epididymis or the testis its self.

SCROTAL SWELLINGS: A JUNIOR DOCTOR'S DIAGNOSTIC DILEMMA

Amr Hawary and Ian Pearce

Presentation

Although pain is usually the presenting complaint, it is typically less severe and of more gradual onset than testicular torsion, however, it may be almost impossible to distinguish the two and the correct diagnosis may only be made during surgery.

Examination

Examination reveals a painful swollen testis. The tenderness is usually localised to the area of the appendage.

The haemorrhagic infarction of the hydatid of Morgagni may be seen as a localised area of discolouration at the upper pole of the testicle, the "blue dot sign".

Investigations

Like most scrotal swellings the diagnosis is usually made based on history and clinical findings, however, Doppler ultrasound scan may be utilised if in doubt.

Management

Scrotal exploration and excision of the infracted hydatid of Morgagni is the recommended treatment as this reduces pain and aids swift recovery. There are, however, no long-term adverse effects of conservative management.

Epididymo-orchitis

Epididymal infection progressing to involve the testis occurs as a result of retrograde ascending infection via the ejaculatory ducts and the vas deferens.

Aetiology

The causes of epididymo-orchitis vary with age. In infants and young patients one should suspect an underlying urology condition especially if the condition is recurrent (e.g. neuropathic bladder or vesico-ureteric reflux).

In young adults who are sexually active a sexually transmitted disease leading to epididymo-orchitis should be excluded before exploring other causes while in older men bladder outlet obstruction with accompanying incomplete emptying of the bladder is a likely aetiological factor.

Presentation

The patients may present with a poor general condition weakness, fevers and lethargy together with a longer history of gradual onset scrotal pain which may be accompanied with an array of urinary symptoms including, dysuria, foul smelling urine or even haematuria.

Examination

Examination reveals a swollen, tender and hard testis with signs of increased scrotal temperature and erythema. It may be difficult to differentiate from testicular torsion or even testicular tumours on scrotal examination.

Investigations

Urinalysis will usually shows proteinuria, pyuria and the presence of nitrites and formal analysis with microscopy will reveal bacteruria and pyuria. Urine cultures are typically positive once returned after 48 hours.

Common Pathogens include:

- *Klebsiella pneumoniae*
- *Proteus species*
- *Staphylococcus*
- *Pseudomonas*

Scrotal Ultrasonography will usually demonstrate hyperaemia and increased vascularity to the testis.

Management

Conservative treatment in the form of analgesia, bed rest and empirical antibiotics until a definitive urine culture and sensitivity is available, it is usually the mainstay of treatment. Surgical treatment is only resorted to if the diagnosis of testicular torsion cannot be excluded or to drain a scrotal or testicular abscess that may develop secondary to severe epididymo-orchitis. In such cases testicular ischaemia and atrophy may occur.

Take home messages:

- Thorough history taking is pivotal in reaching a proper diagnosis.
- Full examination of all systems is essential when examining a patient with a scrotal swelling.
- If suspecting testicular torsion consult your senior immediately.

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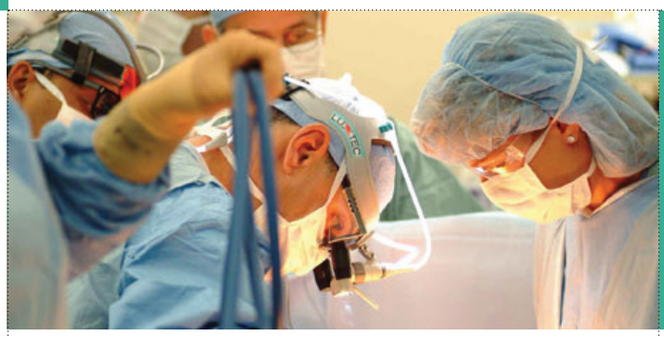
GUIDANCE ON HOW TO PREPARE AND PASS YOUR MRCS EXAMINATION

Hannah Winter



If you are intending to pursue a career in surgery and are a Foundation Year doctor, it would be wise for you to begin to look into the MRCS examination.
Teaching & Training.

Introduction

If you are intending to pursue a career in surgery and are a Foundation Year doctor, it would be wise for you to begin to look into the MRCS examination. Competition for placements is traditionally high and now is no exception. Completing part of the examination looks good on your curriculum vitae and demonstrates commitment and dedication to a career in surgery.

The new examination is different to the traditional MRCS. There are guidance notes and a syllabus provided by the Royal College of Surgeons Great Britain and Ireland, which you should read thoroughly before undertaking the examination[1]. We hope that this article will add to the advice already available and help you to prepare, know what to expect and ultimately pass the exam.

Part A: MCQs and EMQs

This part of the examination is similar to that taken by the collegiate MRCS candidates. It is divided into Applied Basic Sciences and Principles of Surgery-in-General.

The papers last 2 hours each and take place on the same day with a break in-between for lunch. The Applied Basic Sciences paper consists of 135 multiple choice questions, each with one single best answer available from a choice of 5 options. The Principles of Surgery-in-General paper consists of 135 questions, which are extended matching questions with a variable number of options and clinical scenarios. You must pass both papers in order to pass the examination. There is no negative marking.

The best advice to get through Part A is to practise, practise, practise! There are numerous websites you can register with that have practise questions specifically aimed at the MRCS examination[2, 3]. Although these can be expensive, they are commonly used and considered a great help. Ideally, if you are organised and efficient, 3 months is an appropriate time to begin your revision. At this stage, register with the practise websites and borrow some books from the library. Those of most use will be practise question books and revision books on physiology, pathology and anatomy (see Figure 1). Start your revision with targeted revision books and then add to this with additional reading from larger text books.

If you are in a surgical placement, take note of your next days operating list. Read up the anatomy relevant to the cases listed and then test yourself and ask your seniors to question you in theatre. This is a great way to revise and consolidate what you have learnt the previous day. Use people around you who have the time and knowledge to help. Remember, they were once in the same position and will mostly be more than happy to help you out.

Part B: OSCE

The OSCE is a fast paced examination lasting 3 hours 30 minutes. While it does not feel that long, it is exhausting so make sure you sleep well the night before and eat a good breakfast. There are 20 stations; 12 manned stations, 4 unmanned and 4 rest stations. Each station lasts 9 minutes and in-between stations there is a minute for time to move.

The OSCE gives you the opportunity to specify your area of interest. While 12 stations are generic, 4 of the stations are related to the specialties of your choice. The specialties to choose from are: a) head and neck; b) trunk and thorax; c) limbs (including spine); and d) neurosciences. You are asked to select two options when submitting your application. Your first choice will arise as 1 station in anatomy and pathology, 1 in history taking and 1 in physical examination. Your second choice will arise only in your second physical examination station.

The marking system is set on 6 domains, based on good medical practise and 5 broad content areas. The domains include: knowledge; clinical skills; technical skills; communication; decision-making/problem-solving; and organisation/planning. The broad content areas include: anatomy/pathology; surgical skills/patient safety; communication skills; applied surgical science/critical care; and clinical skills.

Anatomy is the broadest subject examined in the OSCE. It is certainly the area that takes the longest time to revise and where candidates commonly begin their revision. Anatomy is best learnt from a core anatomy text book (see Figure 1). This will have the details that are required for the OSCE. Again, try to consolidate what you have learnt by coordinating your reading with up-coming theatre cases. The anatomy stations in the OSCE use cadaveric specimens. An anatomy atlas of prosections is therefore a very useful revision aid (see Figure 1).

GUIDANCE ON HOW TO PREPARE AND PASS YOUR MRCS EXAMINATION

Hannah Winter

Revising with friends is another useful revision method. Dividing areas of the syllabus and preparing presentations for each other, helps to reduce the workload and makes for a more interesting revision session. If you can get hold of a skeleton between you, this is a great way to learn the anatomy of bones and muscle attachments. Teaching medical students is another way to help with revision by further consolidating your knowledge and practising verbalising this knowledge to others.

Anatomy:	
1.	*Clinically Orientated Anatomy:* K.L.Moore and A.F.Dalley[4].
2.	*Instant Anatomy:* N.R.Borley and R.H.Whitaker[5].
3.	*Clinical Anatomy:* H.Ellis[6].
4.	*McMinn's Clinical Atlas of Human Anatomy:* P.H.Abrahams, J.Boon and J.D.Spratt[7].
Clinical examinations:	
1.	*Surgical Short Cases for the MRCS Clinical Examination:* C. Parchment Smith[8].
2.	*Clinical Cases and OSCEs in Surgery:* M. Ramachandran[9].
General revision aids:	
1.	*Surgical Critical Care Vivas:* M. Kunani[10].
2.	*Applied Surgical Physiology Vivas:* M. Kunani[11].
3.	*Surgical Pathology Revision:* D. Lowe[12].
4.	*Essential Revision Notes for Intercollegiate MRCS Books 1 and 2:* PasTest[13, 14].
5.	*Basic Science for the MRCS:* A. Raftery and M. Delbridge[15].

Figure 1: Books that are commonly used for MRCS revision.

To practise for your clinical stations there are a selection of books available (see Figure 1). However, these are intended to supplement clinical experience and nothing can be better than actually seeing and examining patients yourself. Attending outpatient clinics is a great opportunity to see interesting signs. There will often be clinics specifically for neck lumps and general orthopaedic clinics are helpful to learn how to examine joints correctly. The surgical day case department will often have patients with hernias, varicose veins and lumps and bumps listed for theatre that day. It is advisable to visit these patients prior to surgery, preferably with the consultant present who can then question you in an examination style. It is not uncommon in the OSCE to be asked to examine the cardiovascular or respiratory system where peri-operative management of patients can raise interesting discussion. An example of this is the peri-operative management of patients with metallic heart valves.

The CCrISP course, organised by the Royal College of Surgeons, may help to prepare for the communication skills stations. This course is good revision for critical care and physiology, but also includes sessions with role players and is useful for developing communication skills and becoming used to others watching and assessing you in consultations.

When you are in your OSCE, always be systematic in your approach to answers. Remember: management is mostly conservative, medical or surgical. Surgery is often laparoscopic or open. Fractures can be described as stable or unstable, simple or comminuted. Finally, good luck to you all. There will always be something you don't know and can't answer, but remain positive and be confident with all that you do know. Best of luck!

References

1. The Royal College of Surgeons of Great Britain and Ireland (2008) G*uidance Notes* (online). Available from: h**ttp://www.intercollegiatemrcs.org.uk/new/guidance_html** (accessed 16 October 2009).
2. PasTest (2009) *MRCS Part A Online Revision* (online). Available at: h**ttp://www.pastest.co.uk/public/MRCS-A-Online-Revision-7-73-t8.aspx** (accessed 16 October 2009).
3. 123Doc Education (2009) *Medical Education* (online). Available from: **http://www.123doc.com/** (accessed 16 October 2009).
4. Moore KL, Dalley AF (2005) *Clinically Orientated Anatomy.* 5th edn. Lippincott Williams and Wilkins.
5. Borley NR, Whitaker RH (2005) *Instant Anatomy.* 3rd edn. Wiley Blackwell.
6. Ellis H (2006) *Clinical Anatomy.* 11th edn. Wiley Blackwell.
7. Abrahams PH, Boon J, Spratt JD (2007) *McMinn's Clinical Atlas of Human Anatomy.* 6th edn. Mosby.
8. Parchment-Smith C (2002) *Surgical Short Cases for the MRCS Clinical Examination.* PasTest.
9. Ramachandran M (2002) *Clinical Cases and OSCEs in Surgery.* Churchill Livingstone.
10. Kanani M (2002) *Surgical Critical Care Vivas.* Cambridge University Press.
11. Kanani M (2004) *Applied Surgical Physiology Vivas.* Cambridge University Press.
12. Lowe D (2006) *Surgical Pathology Revision.* 2nd edn. Cambridge University Press.
13. Andrews S, Parchment-Smith C, Chalmers R (2006) *Essential Revision Notes for Intercollegiate MRCS: Book 1.* PasTest.
14. Parchment-Smith C (2006) *Essential Revision Notes for Intercollegiate MRCS: Book 2.* PasTest.
15. Raftery AT, Delbridge M (2006) *Basic Science for the MRCS.* Churchill Livingstone.

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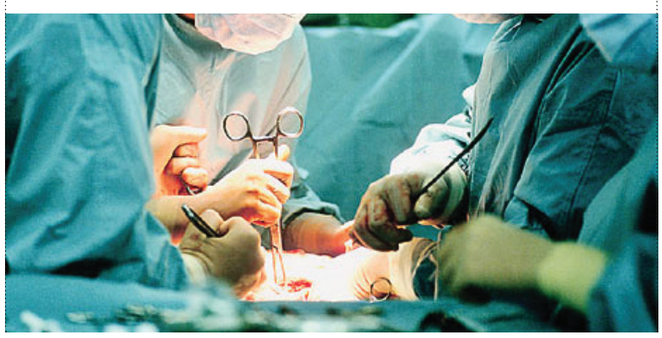
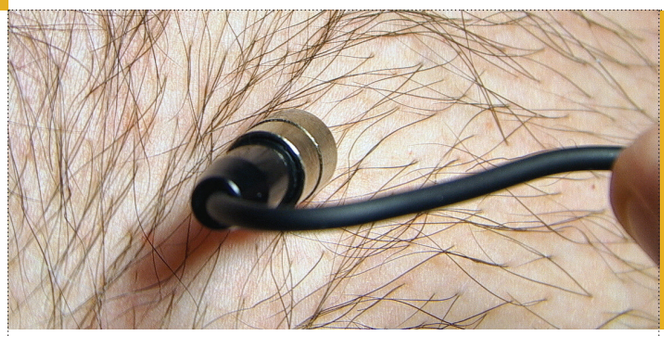


THE USE OF GASTROPROTECTION IN TRAUMA PATIENTS RECEIVING NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

Sunil Sharma, Gokulan Phoenix, James Chan, Graham Sleat and Alastair Graham



A clinical audit of trauma patients in a district general hospital was performed to assess and improve the adherence to evidence - based guidelines for gastroprotection when prescribing non-steroidal anti-inflammatory drugs (NSAIDs).
Good Clinical Care.

Abstract

Background

A clinical audit of trauma patients in a district general hospital was performed to assess and improve the adherence to evidence-based guidelines for gastroprotection when prescribing non-steroidal anti-inflammatory drugs (NSAIDs).

Methods

A clinical audit was conducted over an 18-week period to assess pre-intervention practice. Subsequently, changes including prescriber and pharmacist education were implemented and following this data was collected over a 9-week period to assess any change in performance. Case notes and prescription charts of all adults (aged 18 or more) commenced on regular NSAIDs on admission to the Trauma Unit were reviewed.

Patients were risk stratified according to the number of risk factors, which included age over 65 years, major co-morbidity, oral steroids, anticoagulation, history of upper gastrointestinal ulceration or bleeding and prescription above the generally recommended dose of NSAIDs. The use of gastroprotective drugs was recorded to measure adherence to evidence-based guidelines.

Results

A total of 644 patients were reviewed over the study period, 451 pre-intervention and 193 post-intervention. One hundred patients fulfilled the inclusion criteria pre-intervention and 49 post-intervention. The proportion of high-risk patients co-prescribed gastroprotection was 25.3% pre-intervention and 73.1% post-intervention. The likelihood of a patient receiving gastroprotection increased significantly with the presence of high risk as compared to background risk both pre- ($p=0.002$) and post-intervention ($p<0.001$).

Conclusions

- The majority of trauma admissions are at high risk for developing gastrointestinal haemorrhage.
- Initial adherence to national guidelines for safe prescription of NSAIDs in our trauma unit was low, but improved dramatically with intervention, including education of prescribers and pharmacists.
- Awareness of gastroprotection guidelines must be raised in trauma units to minimise the risk of GI complications.

Introduction

Non-steroidal anti-inflammatory drugs (NSAIDs) are effective analgesic and anti-inflammatory agents that have been recommended by the World Health Organisation for the first line management of pain. However, their side effect profile includes adverse gastrointestinal events[1–4].

The widespread use of NSAIDs, together with the high turnover of orthopaedic trauma patients means that the safe prescription of NSAIDs represents an important clinical and epidemiological issue.

There is much evidence to show that gastrointestinal toxicity is associated with chronic NSAID use, especially in the context of rheumatoid and osteoarthritis[5]. However, it has also been well documented that acute GI haemorrhage can occur as a result of short-term NSAID administration[6].

Guidelines for the safe prescription of NSAIDs have been developed by the American Rheumatological Society for pain control in patients who undergo elective orthopaedic surgery[7]. These recommend the use of gastroprotection for patients on NSAIDs who have one or more risk factors.

The purpose of this audit was to assess adherence to evidence-based national guidelines on gastroprotective strategies in new NSAID users in the orthopaedic trauma setting in a District General Hospital (Stoke Mandeville Hospital, Aylesbury), and to assess the association between risk factors and prescription of a gastroprotective agent.

STANDARD

American rheumatological society guidelines recommend the use of gastroprotection for patients on NSAIDs who have one or more risk factors[7].

THE USE OF GASTROPROTECTION IN TRAUMA PATIENTS RECEIVING NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

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Method

The audit population, obtained from the local Trauma and Orthopaedics admissions Access database (Microsoft Corporation, Seattle), consisted of emergency admissions under Trauma and Orthopaedics.

Case note and prescription chart review was carried out for all patients and inclusion criteria included: (1) minimum age of 18 years; and (2) commencement of NSAIDs at or during admission. Subjects on long-term NSAIDs and those who were commenced on NSAIDs on an irregular, as required, basis were excluded. Demographic, diagnostic, management information, type of NSAIDs, type of gastroprotection and upper GI event during admission were also recorded.

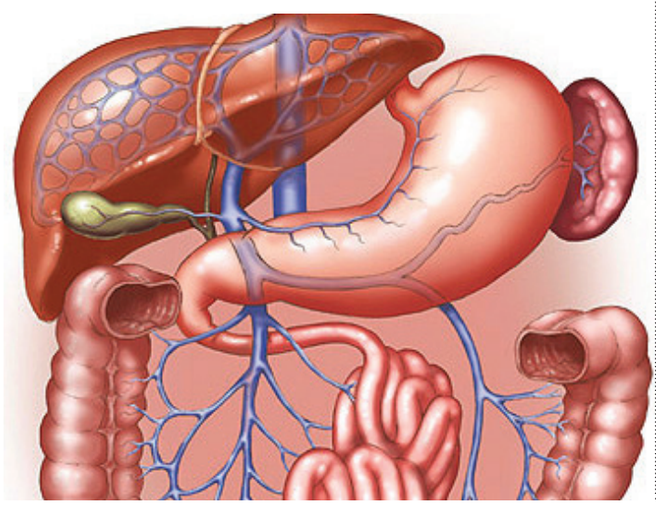
Patients were risk stratified according to the following[7-9]:

- age > 65 years
- major systemic co-morbidities
- past history of upper GI haemorrhage or peptic ulcer disease
- concomitant use of oral steroids
- concomitant use of anticoagulants
- high or twice normal dosage of NSAID.

Those with no risk factor were deemed to have a "background" risk of GI haemorrhage whereas those with one or more risk factors were at "high risk".

Evidence-based guidelines state that those at "high risk" should be prescribed gastroprotection (omeprazole 20mg) whereas those at "background risk" should not[7]. Adherence to these guidelines was recorded. Statistical data analysis involved the use of the Fishers Exact Test via the Simple Interactive Statistical Analysis package[10].

Pre-intervention, an audit was performed over an 18-week period between April and August 2008 inclusive (451 admissions). Various strategies aiming to improve practise were implemented (education of surgical prescribers and pharmacists, and posters to raise awareness of guidelines). Post-intervention, the same assessment of prescribing practice over a 9-week period between September and November 2008 (193 admissions) was performed.



Results

Pre-intervention

Of a total of 451 admissions, 100 patients (22%) satisfied the inclusion criteria, including 37 males and 63 females, giving a male:female ratio of 1:1.7. Of these patients, 75% (75 patients) met the definition of "high risk".

Of the 100 eligible patients in the audit, 25 patients (25%) had no risk factors, 15 patients (15%) had 1 risk factor, 29 patients (29%) had 2 risk factors, 24 patients (24%) had 3 risk factors, and 7 patients (7%) had 4 risk factors for adverse GI events.

Of the "high-risk" patients, 67 out of 75 patients (89%) had age ≥65 as a risk factor. Forty-nine out of 75 patients (65%) had concomitant use of anticoagulants, 38/75 patients (51%) significant co-morbidities, 10/75 patients (13%) concomitant prescription of glucocorticoids, and 5/75 patients (7%) history of peptic ulcer disease/GI bleeds.

Adherence rate to the national evidence-based guidelines for the prescription of gastroprotective agents with NSAIDs was 19/75 patients (25%)[7].

Sixty-seven out of 100 (67%) patients were prescribed diclofenac, and 33/100 (33%) patients were prescribed ibuprofen.

Of the patients who received gastroprotection, 16/19 patients (84%) received omeprazole 20mg once daily, 1/19 patients (5%) received lansoprazole 30mg once daily and 2/19 patients (11%) received ranitidine. No patient had documented intolerance of any gastroprotective agent.

The likelihood of a patient receiving gastroprotection significantly increased with the presence of high risk as compared to background risk (p=0.002). None of the patients with background risk were co-prescribed gastroprotective agents (see Table 1 and Graph 1).

Number of patients on gastroprotection pre-intervention				
		Gastroprotection		Total
		No	Yes	
Level of risk	Background risk	25	0	25
	High risk	56	19	75
Total		81	19	100

Table 1: Number of patients on gastroprotective therapy for each risk factor group pre-intervention.

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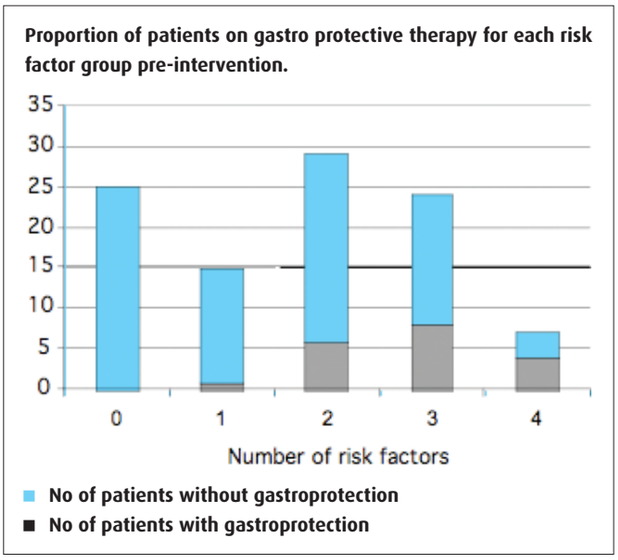


Graph 1: Proportion of patients on gastroprotective therapy for each risk factor group pre-intervention.

Post-intervention

Of a total of 193 admissions, 49 patients (25%) satisfied the inclusion criteria, including 28 males and 21 females giving a male:female ratio of 1.3:1. Twenty-six patients (53%) met the definition of "high risk".

Of the 49 eligible patients, 23 patients (47%) had no risk factor, 14 patients (29%) had one risk factor, 11 patients (22%) had two risk factors, and 1 patient had three risk factors (2%) for adverse GI events.

Of the "high-risk" patients, 20/26 patients (77%) had age ≥65 as a risk factor. One out of 26 patients (4%) had concomitant use of anticoagulants, 17/26 patients (65%) significant co-morbidities, and 3/26 patients (12%) concomitant prescription of glucocorticoids.

Adherence rate to the national evidence-based guidelines for the prescription of gastroprotective agents with NSAIDs was 19/26 patients (73%)[7].

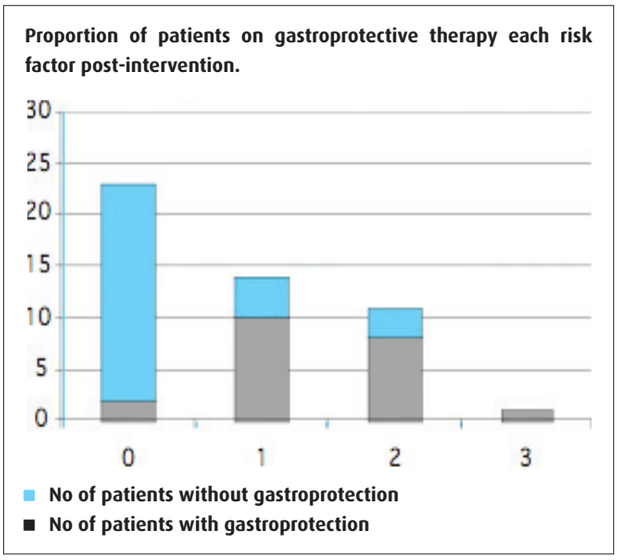
Thirty-six out of 49 (74%) patients were prescribed diclofenac, and 13/49 (27%) patients were prescribed ibuprofen.

Of the patients who received gastroprotection, 13/21 patients (62%) received omeprazole 20mg once daily, 8/21 patients (38%) received lansoprazole 30mg once daily and none received ranitidine. No patient had documented intolerance of any gastroprotective agent.

The likelihood of a patient receiving gastroprotection significantly increased with the presence of high risk as compared to background risk ($p<0.001$). Two of the patients with background risk were co-prescribed gastroprotective agents (see Table 2 and Graph 2).

Number of patients on gastroprotection post-intervention				
		Gastroprotection		Total
		No	Yes	
Level of risk	Background risk	21	2	23
	High risk	7	19	26
Total		28	21	49

Table 2: Number of patients on gastroprotective therapy for each risk factor group post-intervention.



Graph 2: Proportion of patients on gastroprotective therapy for each risk factor group post-intervention.

Discussion

The pre-intervention adherence to evidence-based guidelines for the safe prescription of NSAIDs in our trauma unit was low (25%). Safe prescription of NSAIDs is particularly important in this context because trauma patients tend to be elderly, which in itself, is a risk factor for upper GI haemorrhage (86% of patients in our audit were aged ≥65). Given the widespread use of NSAIDs, the incidence of orthopaedic trauma patients and the proportion of such patients with one or more risk factors, it is clear that the safe prescription of NSAIDs in this context is a major clinical issue.

Post-intervention, adherence to guidelines improved dramatically from 25% to 73%. Furthermore, a smaller proportion of NSAID receivers fell into the high-risk category from 75% to 57%. Ranitidine, which is not a recommended gastroprotective agent according to evidence-based guidelines, was prescribed in 11% of cases pre-intervention but was no longer used post-intervention[7].

THE USE OF GASTROPROTECTION IN TRAUMA PATIENTS RECEIVING NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

Sunil Sharma, Gokulan Phoenix, James Chan, Graham Sleat and Alastair Graham

In this audit 52% of patients had two or more risk factors. Although this audit was not powerful enough to allow quantitative analysis on the increase in risk of GI bleed for each risk factor or combination of risk factors, it would be logical to assume that multiple risk factors will place the patient at greater risk than a single risk factor. It might therefore be prudent to consider alternative analgesia in this subgroup of patients.

This audit was limited by the fact that the sample size was not powerful enough to demonstrate the relationship between NSAID prescription and acute GI bleed. However, there is sufficient evidence in the literature to show that NSAID use, both short term and chronic, can lead to upper GI haemorrhage[1].

The guidelines that we have used as the standard for this audit were based on elective orthopaedic patients, rather than the acute orthopaedic trauma setting. Assuming that the pathogenesis of upper GI bleed secondary to NSAIDs is consistent, these guidelines should apply to all patients receiving NSAIDs.

In view of the findings of this audit, we are currently in the process of updating the local Trust policy regarding the safe prescription of NSAIDs according to the evidence-based guidelines in collaboration with the local Pharmacy and Gastroenterology Departments. These results demonstrate that the intervention strategies implemented were effective and we urge other departments to pursue similar measures to minimise the risk of upper GI side effects in NSAID users.

Conclusions

The majority of trauma admissions are at high risk for developing gastrointestinal haemorrhage.

Initial adherence to national guidelines for safe prescription of NSAIDs in our trauma unit was low, but improved dramatically with intervention, including education of prescribers and pharmacists.

Awareness of gastroprotection guidelines must be raised in trauma units to minimise the risk of GI complications.

References

1. Hernández-Diaz S, García Rodriguez LA (2000) Association between non-steroidal anti-inflammatory drugs and upper gastrointestinal tract bleeding and perforation: An overview of epidemiological studies published in the 1990s. *Arch Intern Med* 160:2093–2099.
2. Allison MC, Howatson AG, Torrance CJ, Lee FD, Russell RI (1992) Gastrointestinal damage associated with the use of non-steroidal anti-inflammatory drugs. *N Engl J Med* 327:749–754.
3. Griffin MR, Yared A, Ray WA (2000) Non-steroidal anti-inflamatory drugs and acute renal failure in elderly persons. *Am J Epidemiol* 151:488–496.
4. Page J, Henry D (2000) Consumption of NSAIDs and the development of congestive heart failure in elderly patients: An under recognised public health problem. *Arch Intern Med* 160:777–784.
5. Gabriel SE, Jaaklimainen L, Bombadier C (1991) Risk for serious gastrointestinal complications related to use of non-steroidal anti-inflammatory drugs: A meta-analysis. *Ann Intern Med* 115:787.
6. Maiden L, Thjodleifsson B, Theodors A, Gonzalez J, Bjarnason I (2005) A quantitative analysis of NSAID-induced small bowel pathology by capsule enteroscopy. *Gastroenterology,* May, 128(5):1172–1178.
7. Recommendations for the medical management of osteoarthritis of the hip and knee. (2000 update) American College of Rheumatology Subcommittee on Osteoarthritis Guidelines. *Arthritis & Rheumatism* 43(9):1905–1915.
8. Fries JF, Williams CA, Bloch DA, Michel BA (1991) Non-steroidal anti-inflammatory drug-associated gastropathy: incidence and risk factor models. *Am J Med* 91:213–222.
9. Laine L (2001) Approaches to non-steroidal anti-inflammatory drug use in the high-risk patient. *Gastroenterology* 120:594–606.
10. **http://www.quantitativeskills.com/sisa/statistics/fishrhlp.htm** accessed on 24 March 2009.

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MANAGEMENT OF ACUTE PANCREATITIS

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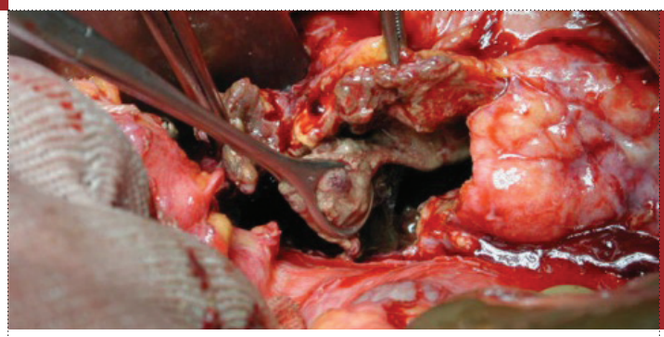


Acute pancreatitis (AP) is an inflammatory condition of the pancreatic gland whereby pancreatic enzymes autodigest the pancreas.
Patient Management.

Abstract

Acute pancreatitis (AP) is an inflammatory condition of the pancreatic gland whereby pancreatic enzymes autodigest the pancreas. Although the exact mechanisms by which this process occurs is unknown, the presence of gallstones and excessive alcohol consumption are responsible for approximately 75% of attacks in the UK. Individuals typically present with acute epigastric pain that radiates through to the back, nausea and vomiting are also prominent features. The diagnosis is confirmed by a raised serum amylase, however, if individuals present more than 3 days after the onset of pain, the amylase may be normal and serum lipase measurements may be more useful. The majority of people will have mild self-limiting disease that requires supportive care only. However, of those with AP approximately 20% go on to have a severe attack characterised by the systemic inflammatory response syndrome (SIRS), and multi-organ dysfunction (MOD). Identifying early those who are likely to have a severe attack has been the driving force behind the development of scoring systems aimed to predict early those who will develop a serious attack. Those with both local and systemic complications will need to receive their care in a high dependency or intensive care unit environment and ideally be managed by multidisciplinary teams. Mortality from AP has a bimodal distribution with approximately half of the deaths occurring in the first 14 days from SIRS and MOD, which fails to respond to treatment. The second peak occurs at 3 months with MOD secondary to sepsis from infected pancreatic necrosis.

Patient scenario

Mr AB, a 56-year-old gentleman presented to A&E with 4-week history of diarrhoea and vomiting. He was passing watery stool four times a day, with some blood on the toilet tissue, none mixed in with stool. He was vomiting small amounts but no blood was present. He also described severe epigastric pain radiating through to the back. His past medical history included hypertension, peptic ulcer disease, haemorrhoids and had been dependent on alcohol for the last 8 years. He is a non-smoker who lives alone. He had been drinking half a bottle of vodka a day for the last 4 months since loosing his job. His regular medications and clinical examination findings on admission are shown in Table 1.

Medications on admission	Clinical examination findings
	Airway patent
Omeprazole 40mg OD	Respiratory rate 22, O2 saturations 84% on air
Candesartan 8mg BD	Heart rate 118
Bisoprolol 10mg OD	Blood pressure 124/80
Ramipril 10mg OD	Urine output 15mls/hr
Simvastatin 40mg ON	Afebrile
Vitamin B 1 Tablet OD	GCS15
Thiamine 300mg OD	Clinically dehydrated, warm peripheries
Felodipine M/R 2.5mg OD	Generalised tender abdomen, not peritonitic

Table 1: Mr AB's medications and clinical examination findings on admission.

Introduction

Acute pancreatitis (AP) is a condition in which activated pancreatic enzymes autodigest the pancreatic gland[1]. The incidence of AP in the UK is between 150–420 cases per million population and appears to be increasing[2,3]. AP occurs equally in men and women and although it can occur at any age is most common in the fourth and fifth decades of life.

Aetiology

Approximately half the cases of AP are due to gallstones with alcohol abuse accounting for 20–25% of cases in the UK[4]. Viral infections, certain medications, for example, statins, steroids, furosemide, opiates, tetracyclines and valproic acid, metabolic conditions (such as hypercholesterolaemia and hypercalcaemia), systemic illnesses, post abdominal and cardiac surgery or ERCP, and trauma may also cause pancreatitis. In approximately 20% of cases no cause is found[4]. It is therefore important when a diagnosis of AP is being considered to ask in the history about:

- previous gallstones or symptoms typical of biliary colic
- alcohol intake
- drug intake
- exposure to viral causes or prodromal symptoms.

MANAGEMENT OF ACUTE PANCREATITIS

Heather M Sowden and Sonia Littlewood

Presentation

Patients commonly present with acute epigastric pain that may radiate through to the back, gradually increasing in severity, such that the patient has to lie still and breathe shallowly. Nausea and vomiting often accompany the pain. It is not uncommon for the patient to have eaten a large meal or have drunk alcohol before the pain began.

Diagnosis

The diagnosis of AP is usually reached by the combination of acute epigastric pain and vomiting together with increased levels of serum amylase[5]. It should be noted that amylase levels rise early in the attack and decline over the first 3-4 days so in a patient presenting 3 days after the onset of pain the amylase may be normal[6]. Amylase has a shorter half-life than lipase and as lipase may remain raised in the serum for longer and is pancreas specific, measurement of lipase in the diagnosis of acute pancreatitis may have a higher specificity for the condition[7]. Continued amylase testing is not recommended as it has no prognostic value and does not reflect the severity of the attack, its use is diagnostic only. Elevated urinary amylase also has diagnostic value. Obtaining a baseline C-reactive protein (CRP) and continued CRP measurements are useful. After 48 hours the CRP appears to be as useful as APACHE II score at predicting severity and reflecting either improvement or worsening of the patients condition[8]. Persistent elevation or continued elevation should prompt CT scanning.

Differential diagnosis and investigations

The differential diagnosis for individuals presenting with acute epigastric pain plus nausea and vomiting include: perforated viscus; acute cholecystitis; bowel obstruction; mesenteric ischaemia; renal colic; myocardial infarction; pneumonia; and diabetic ketoacidosis. Routine investigations that should be performed to exclude the above include:

- an ECG to look for myocardial infarction or ischaemia,
- an erect chest radiograph to look for signs of consolidation, pleural effusions or air under the diaphragm
- serum full blood count, white cell count, liver function tests, amylase and CRP *(Lipase if presenting >3 days after onset of symptoms)*
- arterial blood gas sampling
- urine analysis for blood and white blood cells and ĐHCG levels in females
- serum glucose or capillary glucose
- abdominal ultrasound to look for gallbladder inflammation, common bile duct dilatation and/or the presence of gallstones within the biliary tree.

CT angiography can be undertaken to rule out embolic mesenteric infarction when other causes have been ruled out. An abdominal radiograph should not routinely be performed.

Following the diagnosis of AP further investigations can be done to predict the severity of the attack, e.g. fasting serum calcium and lipids, etc.

Patient scenario

Mr AB was given IV fluids immediately, high flow oxygen, analgesia, pabrinex and DVT prophylaxis. Chest and abdominal radiographs were taken, demonstrating no air under the diaphragm or bowel obstruction or renal stones. The urine was dipped, and both stool and urine samples were sent for culture. Blood results included amylase 910 (0-180U/dl), urea 36.2 (2.5–6.7mmol/L), creatinine 442 (70–130mmol/L), capillary glucose 11.7 (3.5–5.5mmol/L), CRP 106, arterial blood gas demonstrated metabolic acidosis with a base excess of −18.7 but no hypoxia. Mr AB had a score of 3 according to the Glasgow criteria and transferred to a high dependency unit.

Risk stratification

AP can vary in severity from mild inflammation to haemorrhagic necrotic destruction of the whole gland. The latter scenario caries a very high mortality. For that reason, severity scoring systems were developed in an attempt to identify early those who were likely to have a severe attack and develop local and systemic complications as clinical assessment alone could not accurately predict this[9]. On admission the patient should be stratified according to risk and if deemed high risk they should be transferred to the facility in which optimisation of care can occur, this is an HDU/ICU setting. Two scoring systems are frequently used in AP and are specific to pancreatitis, the Ranson and Glasgow Imrie classifications. The APACHE II system which is also used correlates physiological variables with outcome.

Ranson's criteria is made up of eleven variables that have been shown to be prognostically significant in acute pancreatitis. A score of <3 predicting a mild attack, scores of >3 a severe attack and scores of >6 predicting death[10] (see Table 2).

Ranson's criteria	
ON ADMISSION	**AFTER 48 HOURS**
Age >55	Serum Calcium <2mmol/L
WCC >16,000 cells/mm3	Hematocrit fall >10% since admission
Blood glucose >11mmol/L	pO_2 <60mmHg
Serum AST >250 IU/L	Serum Urea increase >8mg/dL
Serum LDH >350 IU/L	Base deficit >4meq/L
	Estimated fluid sequestration >

Table 2: Ranson's criteria for prediction of severe acute pancreatitis. An overall score of >3 predicts severe acute pancreatitis.

The drawbacks of the Ranson criteria are that prediction cannot be made until 48 hours after admission, the criteria were developed on individuals with AP secondary to alcohol consumption and at intermediate scores (4–6) prediction of outcome is less accurate[11].

MANAGEMENT OF ACUTE PANCREATITIS

Heather M Sowden and Sonia Littlewood

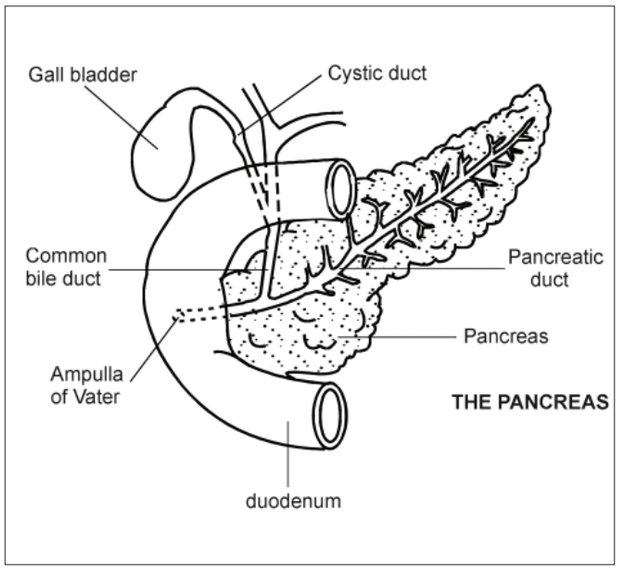
The Glasgow severity scoring system consists of nine variables validated on individuals with both alcoholic and gallstone pancreatitis and is as accurate as the Ranson criteria at predicting death from AP[12] (see Table 3). Similar to the Ranson criteria, a score of >3 predicts severe AP, with the drawback that it assesses the variables in the first 48 hours rather than on admission.

Glasgow prognostic score
Age >55 years
WBC >15 x109/L
Urea >16mmol/L
Glucose >10mmol/L
pO2 <8kPa
Albumin <32g/L
Calcium <2mmol/L
LDH >600 IU/L
AST/ALT >200 IU/L

Table 3: Glasgow criteria for prediction of severe acute pancreatitis. An overall score of >3 predicts severe acute pancreatitis.

The APACHE scoring systems were developed initially to predict mortality by looking at a range of physiological variables in the ICU setting[13]. The APACHE II system is a modified version looking at twelve variables including age and five organ system-based variables, which has the advantage of calculation of disease severity on admission and everyday thereafter thus monitoring the progression of or recovery from disease[14].

An ideal scoring system would incorporate both pancreas specific variables and markers of local and systemic organ dysfunction. The Atlanta criteria do this by incorporating scores from the Ranson and APACHE II severity scoring systems, markers of systemic complications or organ dysfunction and local complications (see Table 4).



Severity Criteria	
SEVERITY SCORING SYSTEMS	
RANSON SCORE	>3
APACHE II score	>8
SYSTEMIC COMPLICATIONS OR ORGAN DYSFUNCTION	
RESPIRATORY	PaO2 <8kPa
Renal	Serum creatinine >177mmol/L after resuscitation
Cardiovascular	Systolic blood pressure <90mmHg after resuscitation
Coagulation system	Platelet count <100x109/L or fibrinogen level <1g/L
Gastrointestinal haemorrhage	>500ml per 24 h
Metabolic disturbance	Corrected serum calcium <1.85mmol/L
	Serum lactate levels >5mmol/L
LOCAL COMPLICATIONS	
ACUTE FLUID COLLECTIONS	Occur early in the natural history of acute pancreatitis and lack a fibrous capsule
Pseudocyst	Occurs at least 4 weeks after the onset of symptoms and has a fibrous capsule
Pancreatic abscess	A localised collection of pus containing little or no necrotic pancreatic material
PANCREATIC NECROSIS	Pathological features: diffuse or focal area of non-viable pancreas that may be associated with peripancreatic necrosis CT features: an area of non-enhancing pancreas measuring >3cm in diameter or >30% of pancreatic tissue

Table 4: Atlanta scoring system for diagnosis of severe acute pancreatitis. Each item on its own constitutes severe acute pancreatitis. For organ dysfunction to be a marker of a severe attack it must last for longer than 48 hours.

The UK Working Party on Acute Pancreatitis guidelines (2005) state that Atlanta criteria be used for assessment of severity, however, if organ failure is present in the first week but resolves within 48 hours, this should not be considered an indicator of a severe attack[4]. This recommendation is based on a study by Buter et al. where it was found that patients with organ failure that had persisted for >48 hours had a mortality rate in excess of 50%, whereas patients with organ failure that resolved within 48 hours had a mortality rate of zero[15].

MANAGEMENT OF ACUTE PANCREATITIS

Heather M Sowden and Sonia Littlewood

Other prognostic indicators for a severe attack include obesity[16], CRP >150 mg/L, Glasgow score ≥3, APACHE II >8 in the first 24 hours or persisting organ failure after 48 hours in hospital[4].

Imaging specific to the diagnosis of acute pancreatitis

The abdominal radiograph contributes little to the diagnosis of AP. Ultrasound may only visualise the pancreas in 25–50% of patients[4], however, it is able to detect the presence of gallstones and dilatation of the common bile duct. Contrast enhanced CT has a use in the diagnosis of AP when clinical and biochemical findings are inconclusive and may also be able to rule out other causes such as infarction of the bowel. CT may be used later in the course of the disease to rule out complications of pancreatic necrosis or pseudocyst formation.

Patient scenario

In the following 6 hours Mr AB was given 8 litres of fluid and only produced 85mls of urine.

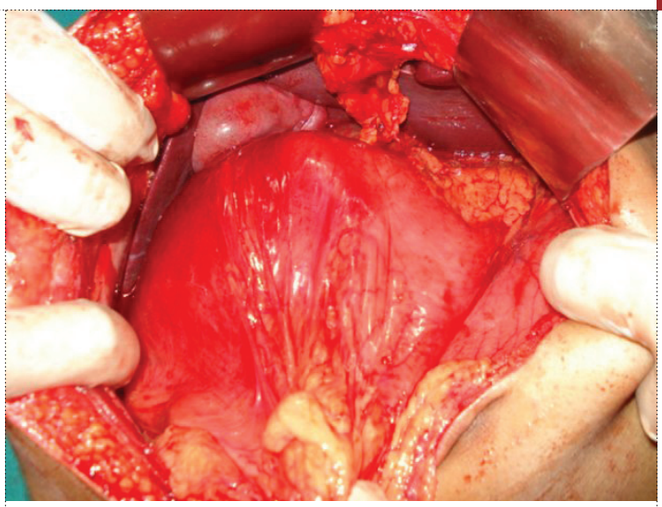
Mr AB was given a patient controlled analgesia system, a central venous pressure line to monitor fluid resuscitation and a combination of chlordiazepoxide, vitamin B complex and thiamine for alcohol withdrawal. Mr AB had had an USS 5-months previous to this, which showed no gallstones and a patent portal vein. Although Mr AB was taking a statin, the AP was deemed to be due to alcohol excess. Six hours later, Mr AB developed shortness of breath and loin pain. A chest radiograph was taken demonstrating bilateral pleural effusions which were drained. His recent bloods showed a raised creatinine of 502 (70–130mmol/L) and urea of 42.1 (2.5–6.7mmol/L) and was only producing 22mls of urine per hour. His CRP had risen to 229 (<5) and his temperature was 38.5°. In view of the raised CRP and pyrexia a CT scan looking for pancreatic necrosis would have been indicated but due to impaired renal function was unable to take place. Mr AB required cardiovascular system support in the form of inotropes.

Initial management

Initially the key measures are prompt fluid resuscitation, both to increase the blood volume, replace the sequestered fluid and to prevent organ damage and subsequent failure due to hypovolaemia. This should occur aggressively in all patients as it cannot always be predicted on admission who is likely to have mild spontaneously resolving pancreatitis and those who are likely to develop complications. Oxygen should also be given, based on evidence that oxygen supplementation together with fluid resuscitation can prevent or improve the clinical picture of organ failure and thus reduce mortality. Continuous monitoring of oxygen saturations, urine output (aiming for >0.5ml/kg/hr) together with CVP monitoring in patients at risk of fluid overload should occur. Analgesia should be provided, using a PCAS if necessary together with anti-emetics. Enteral diet should be stopped to give the pancreas time to rest and recover.

Tests should be done to predict the severity of the attack and look for signs of organ dysfunction through continuous monitoring in those at high risk.

Those with either actual or predicted severe AP should be managed in a high dependency or intensive care unit with full monitoring and organ systems support.



Multi-organ failure and complications

Approximately 20% of those with AP go on to have a severe attack characterised by the systemic inflammatory response syndrome (SIRS) and multi-organ dysfunction (MOD)[17]. These individuals should be managed ideally by multidisciplinary teams with gastroenterologists, interventional radiologists, intensivists and surgeons. Early fluid losses, hypovolaemic shock and symptoms suggestive of organ dysfunction should be proactively sought out and monitored. Hypovolaemia results from a combination of factors including an increased capillary permeability which leads to fluid accumulating within the interstitium and can lead to the development of ascites, an ileus or pleural effusions.

Renal dysfunction can occur as a result of hypovolaemia (pre-renal), septic complications or acute tubular necrosis due to hypoxia.

Pulmonary complications of varying severity occur frequently (some studies >50%) in severe AP with a bimodal peak of distribution[18]. The initial peak occurs on admission with 15% demonstrating radiological abnormalities. By day 5 radiological abnormalities are seen in 71% of patients. Acute lung injuries may also occur as a result of septic shock leading to signs and symptoms of acute respiratory distress syndrome (ARDS).

Patient scenario

By day 3 Mr AB no longer required inotropic support and his renal function was improving producing 30mls of urine an hour, however, his urea and creatinine remained markedly raised. His arterial blood gas results still demonstrated a metabolic acidosis and he was continued on oxygen therapy. A Hickman line was inserted and Mr AB started on total parenteral nutrition.

MANAGEMENT OF ACUTE PANCREATITIS

Heather M Sowden and Sonia Littlewood

Further Management

Nutritional support

Nutritional support is essential in the management of AP. It is debated whether parenteral or enteral feeding is the best approach. Advocates of parenteral nutrition suggest that this method of feeding completely rests the exocrine pancreas[19]. However, evidence has shown that bowel rest is associated with intestinal mucosal atrophy and increased infectious complications due to translocation of bacteria from the gut[6]. TPN is also associated with an increased pro-inflammatory response[6]. Mortality rates are not substantially different in those treated with TPN versus enteral feeding[6], TPN is associated with increased infections, surgical interventions and non-infectious complications[20].

Enteral feeding is usually provided via a fine bore naso-gastric or naso-jejunal tube. In theory naso-jejunal feeding would be more beneficial than naso-gastric feeding as the nutrition is delivered distal to the pancreas, however, studies have demonstrated no significant clinical differences between the two methods[21].

Treatment of causes

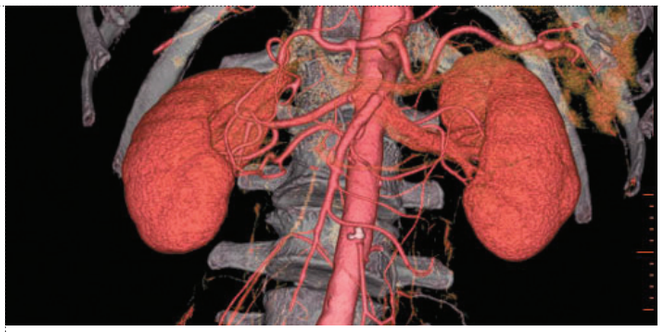
For those with AP of gallstone aetiology and who satisfy the criteria for a predicted or actual severe attack should undergo therapeutic endoscopic retrograde cholangiopancreatography (ERCP) within the first 72 hours after onset of pain. The same applies in the presence of cholangitis, jaundice or a dilated common bile duct. Even if no gallstones are found at ERCP, patients should undergo sphincterotomy. For those with cholangitis endoscopic sphincterotomy or duct drainage by stenting should be performed to ensure relief of obstruction. Definitive management of gallstones by cholecystectomy should ideally occur in the same admission, however, may have to be delayed if the ERCP has worsened the AP. *The UK Working Party on Acute Pancreatitis guidelines* (2005) suggest definitive treatment should occur within 2 weeks of presentation, however, in practice this may be anything up to and including 6 weeks.

When AP occurs secondary to alcohol abuse, patients should be advised to abstain from alcohol permanently in order to prevent further episodes of AP and avoid the development of chronic pancreatitis.

Where metabolic factors, such as hyperlipidaemia and hypercalcaemia, are responsible for AP treatment should be aimed to "normalise" these blood components or in the case of hypercalcaemia rule out underlying hyperparathyroidism or malignancy.

Causative medications should be reviewed and where possible alternatives prescribed.

Viral antibody titres, autoimmune markers, bile samples for bile and pancreatic cytology and sphincter of Oddi manometry may be useful tests to perform during the recovery period if no other cause for AP can be found.



Patient scenario

By day 10 Mr AB's renal function had improved as had his blood gases, he no longer had a metabolic acidosis. He had a CT scan which demonstrated no pancreatic necrosis or pseudocyst formation. He was transferred to the ward and discharged 5 days later and strongly advised to abstain from alcohol and advised to ask his GP for referral to specialist alcohol treatment centres.

Complications

In addition to MOD and sepsis, the mortality associated with AP is closely related to the extent of pancreatic necrosis. CT scanning is used to identify both the presence of and extent of pancreatic necrosis, however, necrosis may not be evident until at least 4 days after the onset of AP. Also the use of CT at this stage may be limited if the use of contrast is contraindicated due to renal impairment. CT imaging may not affect the management of AP in the first week unless prophylactic antibiotics are being considered for management of infected necrosis. Prognosis is worse when pancreatic necrosis affects the head of the pancreas and in the presence of free intraperitoneal fluid and extensive peripancreatic fat stranding[22].

Infection of necrosis is associated with 40% mortality and is the most serious local complication of AP[4]. Antibiotics are unlikely to affect outcomes in patients without extensive necrosis. The risk of infected necrosis and infection in peripancreatic tissue is very small when there is less than 30% necrosis[4].

The UK Working Party on Acute Pancreatitis guidelines (2005) suggest all patients with persistent symptoms and greater than 30% pancreatic necrosis or those with smaller areas of necrosis with clinical suspicion of sepsis should undergo image guided fine needle aspiration to obtain material for culture 7–14 days after the onset of AP.

There is no real consensus on when antibiotic prophylaxis should be used, on whom, which antibiotics and for what period of time. If prophylaxis is to be used it should continue for no longer than 14 days unless guided by bacterial growth on culture when the sample is from fine needle aspirates. A double-blind placebo controlled study by Isenmann and colleagues comparing ciprofloxacin/metronidazole and placebo found no differences between the groups in terms of infected necrosis, systemic complications or mortality rates. However, infectious complications, multi-organ failure, sepsis and SIRS occurred in only 28% of those who received antibiotics compared with 46% of the placebo group.

MANAGEMENT OF ACUTE PANCREATITIS

Heather M Sowden and Sonia Littlewood

Surgical intervention in pancreatic necrosis is guided by the clinical picture. Infected necrosis may be diagnosed by the presence of gas within a pancreatic collection on CT or by fine needle aspiration. All individuals with infected necrosis require either surgical or radiological drainage.

Patient scenario

It took the efforts of a multidisciplinary team including anaesthetists, gastroenterologists, general surgeons and radiologists (to name but a few) to care for Mr AB during his bout of AP. Although he required organ system support for his lungs, kidneys and cardiovascular system he did not go on to develop pancreatic necrosis and the subsequent sepsis that may occur if the necrosis becomes infected. Incidentally Mr AB was readmitted to the hospital 14 days later with another attack of AP secondary to alcohol use.

References

1. Browse NL, Black J, Burnand KG, Thomas WEG (eds) (2005) *Acute Pancreatitis, Browse's introduction to symptoms and signs of surgical disease,* 4th edn. New York: Oxford University Press, pp. 399–400.
2. McKay CJ, Evans S, Sinclair M, et al. (1999) High early mortality rate from acute pancreatitis in Scotland, 1984–1995. *Br J Surg* 86:1302–1305.
3. Toh SK, Phillips S, Johnson CD (2000) A prospective audit against national standards of the presentation and management of acute pancreatitis in the South of England. *Gut* 46:239–243.
4. UK guidelines for the management of acute pancreatitis. UK working party on acute pancreatitis. *Gut* 2005, 54:1–9.
5. Matull WR, Pereira SP, O'Donohue JW (2006) Biochemical markers of acute pancreatitis. *J Clin Pathol* 59:340–344.
6. Frossard JL, Steer ML, Pastor CM (2008) Acute pancreatitis. *Lancet* 371:143–152.
7. Sternby B, O'Brien JF, Zinsmeister AR, DiMagno EP (1996) What is the best biochemical test to diagnose acute pancreatitis? A prospective clinical study. *Mayo Clinic Proc* 71:1138–1144.
8. Wilson C, Heath DI, Imrie CW (1990) Prediction of outcome in acute pancreatitis: a comparative study of APACHE II, clinical assessment and multiple factor scoring systems. *Br J Surg* 77:1260–1264.
9. Lund H, Tonnensen H, Tonnensen MH, et al. (2006) Long-term recurrence and death rates after acute pancreatitis. *Scand J Gastroenterol* 41:234–238.
10. Berry AR, Taylor TV, Davies GC (1981) Pulmonary functions and fibrinogen metabolism in acute pancreatitis. *Br J Surg* 68:870—873.
11. McMahon MJ, Playforth MJ, Pickford IR (1980) A comparative study of methods for the prediction of severity of attacks of acute pancreatitis. *Br J Surg* 67:22–25.
12. Ranson JH, Rifkind KM, Roses DF, et al. (1974) Prognostic signs and the role of operative management in acute pancreatitis. *Surg Gynecol Obstet* 139:69–81.
13. Imrie CW (1997) Classification of acute pancreatitis and the role of prognostic factors in assessing severity of disease. *Schweiz Med Wochenschr* 127:798—804.
14. Imrie CW (2003) Prognostic indicators in acute pancreatitis. *Can J Gastroenterol* 17:325–328.
15. Knaus WA (2002) APACHE 1978–2001: the development of a quality assurance system based on prognosis: milestones and personal reflections. *Arch Surg* 137:37–41.
16. Knaus WA, Draper EA, Wagner DP, et al. (1985) APACHE II: a severity of disease classification system. *Crit Care Med* 13:818—829.
17. Buter A, Imrie CW, Carter CR, et al. (2002) Dynamic nature of early organ dysfunction determines outcome in acute pancreatitis. *Br J Surg* 89:298–302.
18. Martinez J, Johnson CD, Sanchez-Paya J, et al. (2006) Obesity is a definitive risk factor of severity and mortality in acute pancreatitis: an updated meta-analysis. *Pancreatology* 6:206-–209.
19. Windsor AC, Kanwar S, Li AG, et al. (1998) Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity in acute pancreatitis. *Gut* 42:431–435.
20. Marik PE, Zaloga GP (2004) Meta-analysis of parenteral nutrition versus enteral nutrition in patients with acute pancreatitis. *BMJ* 328:1407–1412.
21. Eatock FC, Chong P, Menezes N, et al. (2005) A randomised study of early nasogastric versus nasojejunal feeding in acute severe pancreatitis. *Am J Gastroenterol* 100:432–439.
22. Eatock FC, Brombacher GD, Steven A, et al. (2000) Nasogastric feeding in severe acute pancreatitis may be practical and safe. *Int J Pancreatol* 28:23–29.
23. Isenmann R, Runzi M, Kron M, et al. (2004) Prophylactic antibiotic treatment in patients with predicted severe acute pancreatitis: a placebo-controlled, double-blind trial. *Gastroenterology* 126:997–1004.

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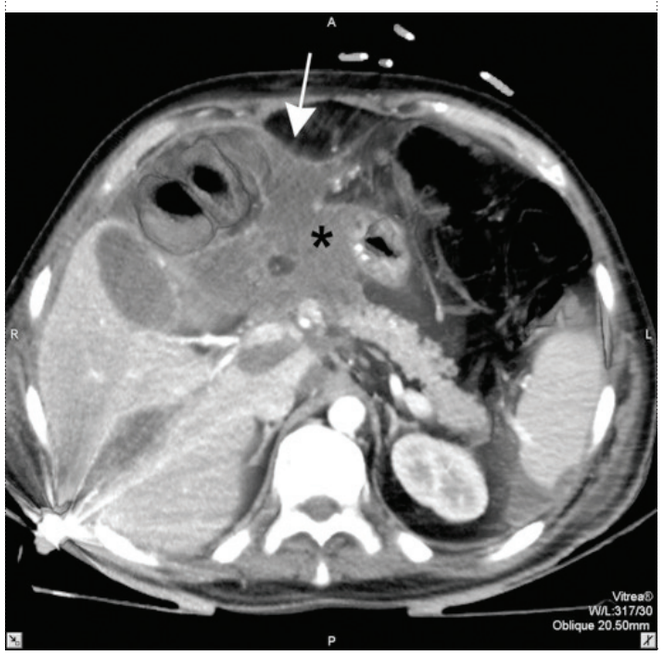

ARTERIAL BLOOD GASES IN SURGICAL PATIENTS

Charlotte Richardson, Hiten Patel, Sonia Bhangu and Aneel Bhangu

A great time to test your knowledge.
Test Yourself.

Introduction

This article aims to give you more experience at interpreting arterial blood gases (ABG), with focus on situations you may find in surgical patients. You should, by now, know the methodology of how to interpret an ABG and so these questions will ask you to identify the type of acid-base disturbance; the likely diagnosis; and then the first steps of management that you should take. When reading the situation, begin to predict what type of acid-base disturbances the patient may be suffering from. The clues are there and you should be doing this as you assess real patients too.

Normal ranges

pH	7.35–7.45
pO_2	12–15KPa
pCO_2	4.5–5.6KPa
HCO_3	22–26mmol/L
Base Excess	± 2
K+	3.5–5.5mmol/L

Case 1

A 78-year-old woman presents with a 1-day history of sudden onset central abdominal pain. The pain has not settled and she has passed a small amount of bloody mucus PR. She is not hungry and has not vomited. On examination she is dehydrated and her abdomen is very tender centrally, but is soft with no masses. She has a pulse of 150 beats per minute, irregularly irregular. She is on 30% oxygen via a facemask. You perform an ABG which shows:

pH	7.2
pO_2	17.5
pCO_2	4.5
HCO_3	17
BXS	-10

1. What is the abnormality on this blood gas?
2. What is the most likely diagnosis?
3. What initial management steps will you perform?
4. Where would this patient be best managed?

Case 2

A 68-year-old woman presents with vomiting and colicky abdominal pain. She has been vomiting several times a day at home for the last 2 weeks. She has not opened her bowels in the last 10 days and has not passed any wind in the last week. She is unable to keep any food down and can only manage a few sips of water. She has previously had two caesarean sections and an appendectomy, all of which were over 20 years ago. On examination she is dehydrated and hypotensive. She has a distended abdomen which is diffusely tender and bowel sounds are absent. PR examination reveals a collapsed rectum (the normal volume of the rectum is lost, suggesting a proximal obstruction). You perform an ABG on air which reveals:

pH	7.55
pO_2	10
pCO_2	6.5
HCO_3	35
BXS	+15
K	+ 2.9

1. What is the abnormality on this blood gas?
2. What is the likely underlying diagnosis?
3. Why has this metabolic abnormality developed?
4. How will you initially manage this patient?

Case 3

A fit and well 35-year-old man returns from the ward following an elective inguinal hernia repair. He is drowsy, has a GCS of 12 (E3V4M5) and oxygen saturations of 88%. His airway is patent, his respiratory rate is 6 breaths per minute and his chest is clear to auscultation. You perform an arterial blood gas which shows:

pH	7.2
pO_2	8.0
pCO_2	7.5
HCO_3	24
BXS	1

1. What is the abnormality on this blood gas?
2. What is the most likely cause?
3. What are the key initial steps you will carry out?

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Case 4

You are asked to see a 75-year-old woman with gallstone pancreatitis on the ward. She has peripheral saturations of 89% (normal the previous day) and a shallow respiratory rate of 22 breaths minute. Her chest is clear to auscultation. She has a distended, painful and guarded abdomen. She is a lifelong non-smoker and was previously fit and well. She has been receiving clexane and paracetamol on her drug chart. The nurse has started oxygen at a rate of 4 litres via a Hudson facemask. An ECG reveals a sinus heart rate of 80 beats per minute. You perform an arterial blood gas:

pH	7.4
pO_2	7.5
pCO_2	4.5
HCO_3	24
BXS	1.5
SO_2	88%

1. What is the abnormality on this blood gas?
2. What is the most likely cause?
3. How can you improve the situation?

Answers

Case 1

1. What is the abnormality on this blood gas?
There is a marked metabolic acidosis. There is little attempt at respiratory compensation, suggesting an acute event.
2. What is the most likely diagnosis?
An elderly female patient in atrial fibrillation with sudden onset abdominal pain is mesenteric ischaemia until proved otherwise. The cause can be arterial embolus or venous thrombosis; in this case, an arterial embolus from the left atrium is the most likely cause.
Learning point: in Mesenteric ischaemia there is a classic triad: (a) a very tender but soft abdomen; (b) tachycardia despite fluid resuscitation, and (c) a metabolic acidosis. Bloody diarrhoea is also common.
3. What initial management steps will you perform?
- Assess with an ABC approach.
- Oxygen.
- Intravenous access (take bloods for FBC, U&E, clotting and group & save). If the patient is on warfarin (especially with AF), find out why and discuss with a haematologist as early as possible for advice on reversal and cover with heparin.
- Fluid resuscitation – commonly with Hartmann's solution (physiologically balanced). The rate is tailored to the clinical findings.
- Analgesia.
- Urinary catheter.
- ECG.

At this stage you need to inform senior doctors that a sick patient needs further management. It is initially a clinical diagnosis, although a CT scan can help prove the diagnosis.

4. Where would this patient be best managed?
Depending on the patient's co-morbid status and wishes, surgery is needed to remove any dead or dying bowel. Until then, resuscitation should continue, which is often best performed in critical care settings with the help of anaesthetic teams.

Case 2

1. What is the abnormality on this blood gas?
There is a metabolic alkalosis with some attempts at respiratory compensation.
2. What is the likely underlying diagnosis?
Adhesion bowel obstruction with secondary vomiting. Previous abdominal surgery suggests that adhesions are the most likely cause.
3. Why has this metabolic abnormality developed?
The patient has been at home and vomiting for some time due to the obstruction. This means she has been vomiting gastric acid, which is rich in hydrogen and potassium. The loss of these without replacement leads to a metabolic alkalosis and hypokalaemia.
4. How will you initially manage this patient?
Initial management is with ABC. The patient needs oxygen, fluid resuscitation (with potassium supplementation), blood tests, an NG tube and a catheter. The surgical registrar needs to review the patient to ensure adequate resuscitation. A CT scan can help delineate intra-abdominal events.

Surgery may or may not be needed but resuscitation, especially with intravenous fluids, is the first priority.

Further fluids are tailored to balance NG and urine output, while monitoring and replacing electrolytes.

Case 3

1. What is the abnormality on this blood gas?
There is a marked respiratory acidosis and hypoxia.
2. What is the most likely cause?
This is due to hypoventilation. The most likely cause in this case is opiate induced hypoventilation secondary to general anaesthesia.
3. What are the key initial steps you will carry out?
High flow oxygen (give 15L via a non-rebreathing facemask with the reservoir bag inflated) and give naloxone in 40 micrograms increments intravenously, titrated to response (draw up a 400 microgram ampoule into a 10ml syringe with saline). Repeat doses may be needed dependent on response.

The anaesthetic team should be made aware. If recovery is fast, the patient can be monitored on the ward with further naloxone, if required, and input from the pain team. If recovery is poor, the patient may need to return to theatre recovery or critical care settings for monitoring and management until he has improved. An ECG and chest X-ray should be obtained to exclude other causes, especially if response is not rapid.

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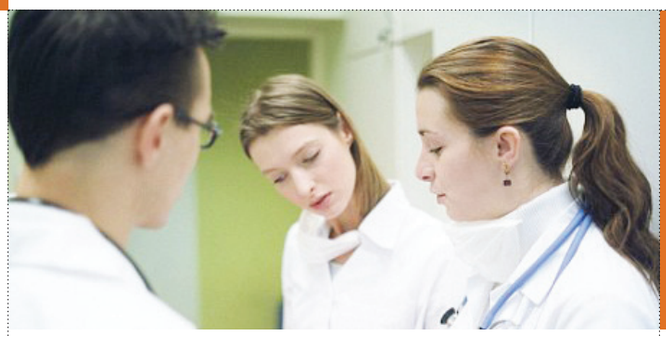

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Case 4

1. What is the abnormality on this blood gas?
Hypoxia. As the pCO_2 is normal this is a type 1 respiratory failure.

2. What is the most likely cause?
It is important to consider primary respiratory problems. A clear chest to examination means that pleural effusions, pulmonary oedema, infections and ARDS are possible but unlikely. An urgent portable chest X-ray will help identify these. She has been receiving clexane and is not tachycardic, so a pulmonary embolism is unlikely. The patient is only on regular paracetamol and is in pain from her abdomen. The pain is causing shallow breathing, leading to hypoventilation with some possible basal collapse (atelectasis). Acute pancreatitis can be extremely painful, and adequate analgesia is a one of the key aspects of treatment.

3. What are the key initial steps you will carry out?
High flow oxygen (give 15L via a non-rebreathing facemask with the reservoir bag inflated) to improve hypoxia. Adequate analgesia is needed; to resolve the initial problem, intravenous morphine can be titrated to pain. However, regular analgesia should be prescribed; a PCA (patient controlled analgesia) may be useful. The patient should be sitting up and urgent chest physiotherapy will help to improve ventilation. Obtain a chest X-ray urgently to exclude other causes.

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